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OF

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A MANUAL

OF

DISEASES OF THE NERVOUS SYSTEM

BY

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UNIVERSITY COLLEGE HOSPITAL AND TO THE NATIONAL HOSPITAL FOR
THE PARALYZED AND EPILEPTIC.

AMERICAN EDITION,

ISSUED UNDER THE SUPERVISION OF THE AUTHOR, AND CONTAINING ALL THE
MATERIAL OF THE TWO-VOLUME ENGLISH EDITION, WITH
SOME ADDITIONS AND REVISIONS.

WITH

THREE HUNDRED AND FORTY-ONE ILLUSTRATIONS.

PHILADELPHIA:
P. BLAKISTON, SON & CO.,
1012 WALNUT STREET.
1888.

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948

Med. 83

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Press of Wm. F. Fell & Co.,
1220-24 Sansom St.,
Philadelphia.

PREFACE TO THE AMERICAN EDITION.

The object of the following work is to afford the student the means of gaining an adequate conception of our present knowledge of the Diseases of the Nervous System, and to supply the practitioner with the information he needs for dealing with those diseases in his daily work. I have endeavored, therefore, to present to the reader the lessons taught by observation rather than the details of the cases observed—to subordinate, as far as possible, theory to fact, the speculative to the practical, and to employ hypothesis only where it is indispensable for the proper grasp of facts.

The first two Parts, dealing with the Diseases of the Spinal Cord and Nerves, have been for some months before the English public, but have been revised for this edition, and some verbal errors have been corrected. The rest of the work is presented simultaneously to American and English readers.

W. R. GOWERS.

QUEEN ANNE ST., LONDON,
February, 1888.

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DISEASES

OF

THE NERVOUS SYSTEM.

INTRODUCTION.

CLASSIFICATION OF DISEASES OF THE NERVOUS SYSTEM.

A CLASSIFICATION of Diseases of the Nervous System, at once scientific, exact, and convenient for systematic description, is not at present possible. If we attempt to classify the diseases according to either seat or nature, we are met at once by difficulties which prevent any complete arrangement. These difficulties arise from the facts that many diseases affect more than one part of the nervous system, that the precise seat of other maladies is unknown, and that we are still ignorant of the nature of a considerable number of diseases. A rough division is often made into two broad classes of "organic" and "functional" disease. The first class of "organic diseases" comprehends those in which there is always a visible lesion, manifesting the morbid process which constitutes the disease. The second class of "functional diseases" is less definite, comprehending (1) those diseases that consist only in a disturbance of function, and are therefore properly so designated, and (2) many diseases which have this in common with true functional disease that they are transient and not permanent, and that they are not known to depend on organic changes. But there is also a large class of diseases in which no constant lesion has been discovered, but which are not transient, and cannot well be placed in either of these classes. This simple classification is therefore inadequate. It may be well to consider in what respect it needs alteration to meet the requirements of our present knowledge.

The term organic disease, as commonly used, means visible disease. But visibility is partly relative; it depends on the means of vision. The application of the microscope to pathology immediately increased the range of organic disease, and a similar increase has resulted, in our own day, from the use of staining agents, which render visible changes that could not before be seen. Still, it is the degree of alteration that determines visibility. Molecular changes in nutrition, considered as such, must be colossal to be detected. Such alterations, not sufficient to be seen, but still considerable, probably constitute the morbid process in many diseases that are commonly classed as functional. Hence we may distinguish a class of "nutritional diseases" from those that consist in a mere derangement of function. While distinguishing these two classes, we cannot entirely separate them, because nutrition and function are mutually dependent, and prolonged functional disturbance may determine nutritional change.

Visible disease varies much in nature and degree. Some forms can always be seen with the unassisted eye; they change the shape, or color, or consistence of the part. Other forms can be detected only with the aid of the microscope, or may be seen with the naked eye when considerable in degree, and only through the microscope when slight in degree. It is convenient to term these "structural diseases." Thus we may distinguish four pathological classes of disease of the nervous system:—

1. *Organic disease* (or *coarse organic disease*); such as tumor, hemorrhage, softening.

2. *Structural disease;* such as most forms of sclerosis.

3. *Nutritional disease;* such as general paralysis of the insane, paralysis agitans, chorea.

4. *Functional disease;* such as reflex convulsions, and many forms of hysteria.

In the first class the morbid process always begins outside the nerve elements themselves. In the second class it may begin within or outside them. In the third and fourth classes these elements are probably always primarily affected.

Such a classification helps us to obtain clearer views of the primary relations of disease, but is not convenient for systematic description. For this a hybrid system is necessary, in which organic and structural diseases are classified according to their seat,—in the nerves, spinal cord, or brain. With each group may be placed some nutritional or functional maladies of local incidence. The bulk of such diseases are best considered after the organic diseases have been described, since many of them are of wide distribution or uncertain seat.

PART I.

GENERAL SYMPTOMATOLOGY.

SYMPTOMS AND THEIR INVESTIGATION.

THE nervous system is almost entirely inaccessible to direct examination. The exceptions to this are trifling. The termination of one nerve, the optic, can be seen within the eye. Some of the nerve trunks in the limbs can be felt; a few, as the ulnar, in the normal state; others only when enlarged by disease. As a rule, morbid states of the nervous system can be recognized only by the derangement of function that they cause. The functions of the nervous system are often roughly divided into mind, motion, and sensation, but they are far more extensive than those words denote. The functions of all the organs of the body, all states of vessel and of circulation, and all processes of nutrition, are under the control of the nervous system. The disturbance of nerve function produced by disease is conterminous in extent with the organism itself. Our knowledge of these symptoms is constantly increasing in range and in degree. They differ, however, very much in relative importance. Some are more frequent in occurrence than others, and are more direct in significance. Many have a special relation to certain parts of the nervous system, and may be considered most conveniently when we deal with the diseases of that part. Others occur in diseases of all parts. It may be well, at the outset, briefly to describe the general characters of some of these most frequent symptoms.

Motor Symptoms.—Disturbance of motion is one of the most common of the effects of disease. Movement is produced by muscles, but the muscles are excited to contraction by the nervous system. Most alterations of motility, both defect and excess, are due to nerve derangement. The conditions are very few in which motor changes originate in the muscles themselves. Defect of movement is of muscular origin only in some cases of primary alteration in the nutrition of the muscles. When there is a general impairment of nutrition, the muscles participate in this; they are ill-nourished, and therefore feeble. Such a condition is met with in chronic wasting diseases, in anæmia, and as an acute affection in all acute febrile diseases. This is the chief cause of the muscular weakness that so often succeeds fevers. In some chronic diseases the muscles undergo a primary degeneration. One is an extremely rare form of muscular atrophy. Another is the disease known as "pseudo-hypertrophic paralysis." A third is ossification of the muscles. In almost all other conditions, loss of power in the muscles is dependent on the state of the nervous system.

So also with increased and involuntary muscular action—spasm. We know even less of what has been termed "idio-muscular contraction," that is, contraction originating in the muscle itself, than we do of "idio-muscular paralysis." Whenever the whole of a muscle contracts, the contraction is probably always due to the influence of the nervous system, even though, as in ordinary cramp, it seems to us to be purely local. We can scarcely conceive that all the fibres of a muscle can contract simultaneously except under central influence. Even muscular "tone" is dependent on the connection of the muscle with the nerve centre. The partial contractions that are termed "fibrillary" are perhaps local, since they are readily produced by mechanical stimulation, such as a tap on the muscle. But it is probable that fibrillary contractions, although sometimes local, are often of central origin. This is true also of the flickering contractions that are so common in the orbicularis palpebrarum and other muscles, in persons whose nervous system is enfeebled.

Defect of movement may present every degree, from slight weakness to absolute loss. There may be an inability even to make the muscle contract, or the voluntary contraction may be too feeble to move the parts to which the muscle is attached. This difference is to some extent relative, since it varies according to the mobility of the part to be moved. A contraction that would move a finger may be insufficient to move a leg. The difference may vary in the same part according to position. A degree of power in the flexors of the hip that will move the leg when the patient is lying on his side may be insufficient to do so if he is lying on the back. Or the part may be moved, but with less force than normal. The term "paralysis" has long been applied to all degrees of defect of power, a distinction being made between complete and incomplete paralysis. Partial loss of power is also sometimes termed "paresis."

Our means of measuring defects of muscular power are very imperfect. The force of some movements can be measured by means of an instrument called a "dynamometer." Several forms of dynamometer have been devised, but that in general use consists of an oval steel ring, which can be narrowed either by compression applied to its smaller diameter, or by traction applied to its longer diameter. The change in the shape of the dynamometer, and therefore the force exerted, is shown by the movement of an index on a scale that has a double graduation, an inner for compression, and an outer for traction. This form of dynamometer is known as "Charrière's," from the name of its first maker. An older form of dynamometer is that designed by Duchenne. It is less convenient for testing the grasp, although better adapted for indicating the force of traction. Practically, however, the use of these instruments is confined to the measurement of the grasp. The estimation of the strength of other movements is difficult, and liable to much error. In examining these it is best for the observer to oppose the movement and note the resistance that the patient can overcome. The wide variations in strength among different persons renders absolute estimation possible only when the defect is considerable. Slight changes are recognized by comparing the power on the two sides. It should be remembered that the limbs on the right side are normally stronger than those on the left, the relation being about as five to four. Care must be taken to secure uniformity of the conditions under which the observations are made.

Incoördination of Movement.—A muscular movement involves an exactly proportioned contraction in the muscles that effect the movement, often numerous, and coöperating in various degrees. It involves also a contraction in the opponents of these muscles, less in degree, but with equal delicacy of proportioned strength. If, instead of this balanced adjustment, the contraction varies in degree in the same or in different muscles, the movement is not that which is intended—is irregular. An excess or deficiency of contraction leads to a corresponding change in the contracting opponents. Attempts to correct the error may themselves be wrong in degree and increase the disturbance. This condition is termed incoördination, or "ataxy." It may be simple unsteadiness of movement, or the excessive contractions may be so sudden and considerable as to cause "jerky incoördination." It is readily recognized by simple observation, or may be recorded by an attempt to draw a straight line on a sheet of paper, or to hold a point in contact with a moving surface (*e. g.*, of blackened paper) on which the irregularities of movement are shown.

Another method of obtaining evidence of it has been suggested by Blix. It consists in making the patient suddenly try to touch with the point of a pencil a spot on a sheet of paper suspended before him, his eyes being closed at the moment of the attempt. After a certain number of attempts the average distance from the spot of the dots he has made is ascertained, and, compared with the result given by a normal individual, affords a numerical indication of the degree of incoördination. The method is effective, although the result is scarcely worth the trouble.*

Whenever there is much incoördination, spontaneous movements occur when the patient tries to hold the limb still in a certain posture. The irregularity obtains in the fixed as well as in the mobile condition. In the latter it causes ataxy, in the former spontaneous movements.

Sensory Symptoms.—Deranged sensation is another very common symptom. It has often to be searched for, as a significant defect may be unknown to the patient. Each form of sensation, of touch, temperature, and pain, must be separately tested, since one may be affected and not another.

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* Blix, *Neur. Centralb.*, *1884*, p. 83.

Tactile Sensibility.—In testing tactile sensibility care must be taken that the instrument employed does not give an impression of heat or cold, lest the patient perceive by the sense of temperature that which he cannot discern by the sense of touch. The observer's finger may be used, if it is not cold, or the skin may be touched with the feather end of a quill pen. The eyes of the patient should be closed during the examination, and, since he is apt to fancy that he feels a light touch when he does not, he should occasionally be asked if he can feel, when no contact is made. If there is no absolute loss, the character of the sensation may be changed; throughout one side a touch may cause a sensation distinctly different from that which is produced when the other side is touched. Loss of sensibility to touch is termed "anæsthesia," but this word is often applied loosely to all forms of impaired sensibility.

In examining the tactile sensibility, it is important to ascertain, not only whether the patient can feel, but whether he is able to recognize the place touched,—whether he can correctly "localize" the sensation. For this he must be asked, not only whether he feels the touch, but to say or point out where he feels it. The part touched should be frequently varied, and the eyes, of course, kept closed. Another test for tactile sensibility depends on the fact, ascertained by E. H. Weber, that the distance apart at which two points are discriminated is nearly the same in different individuals in the same part of the body, although it varies much in different parts of the body. The normal distance being known, the increased distance at which the points have to be placed for their discrimination is a measure of the degree of defect. The points should not be so sharp as to occasion pain. A pair of blunt-pointed compasses may be employed, but the most convenient instrument is one contrived by Dr. Sieveking, and called the "æsthesiometer." It consists of a graduated bar, and two points, one of them being movable and the other fixed. The greater the degree of tactile sensibility, the nearer together can the points be, and still be discriminated. The distance at which they are normally distinguished must be known before any inference can be drawn. The most important average distances, ascertained by Weber, are as follows: The distance is smallest, *i. e.*, the sensitiveness is greatest, at the tip of the tongue, where the points are discriminated when only 1.5 mm. apart. Then come, in order of sensitiveness, the finger tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the tip of the nose, 6 mm.; the cheeks and the backs of the fingers, about 12 mm.; the forehead, 22 mm.; the neck, 34 mm.; the forearm, lower leg, and back of foot, 40 mm.; the chest, 45 mm.; the back, 60 mm.; the upper arm and thigh, 75 mm. Slight variations exist in different individuals, and a deviation from the normal that is uniform in degree throughout the body is probably physiological. In using this test it is necessary to touch the skin with the two points at the same moment, and with equal pressure. The examination requires time and patience, and the results are considerably modified by the intelligence of the patient. Moreover, the power of discrimination is increased by practice. It is rare to obtain a conclusive result unless there is a degree of defect that causes a slight absolute loss, such as may be detected with a feather. For these reasons, the practical value of the test is less than was at first anticipated. It is more useful for the estimation of changes of sensibility in the same person and the same part than for actual diagnosis.

Another test that has been proposed is to move a pointed object along the skin, and note the minimum movement that can be recognized as such (Leube). This test has not come into general use.

Curious modifications of tactile sensibility are sometimes observed. In one of these, a single touch is felt as if it were two or even three, a condition that has been termed "polyæsthesia" (Fischer). In another, an impression on one part is referred to some other part, usually in the same limb, rarely in another limb. In another variety, an impression on one part has been referred to the corresponding place on the opposite side of the body; this has been termed "allocheiria" (Obersteiner).

Perception of the degree of pressure on the skin is probably subserved by the nerves for tactile sensibility. It may be estimated by simple pressure applied through any instrument, or by small weights, or by an instrument contrived for measuring the pressure applied to an artery to ascertain the tension of the pulse. In this, a spring indicates the degree of pressure applied. The part tested must be supported, so that the muscles are not brought into action. The point to be ascertained is the minimum variation that can be recognized. In health, this is about one-twentieth of the total pressure, whatever the latter may be.

Sensibility to pain is subserved by what are called nerves of "common sensibility." It may be tested by a prick or a pinch. For a prick, too fine a point must not be used, not only because a sharp point may inflict a needless wound, but because, in the less sensitive parts of the skin, where the terminal nerve plexus is wide, a fine point may here and there be unfelt, although it penetrates the skin. Hence a somewhat blunt point should be employed. Nothing answers better than the point of a quill pen. The faradaic current may be employed to test the sensibility to pain, wire terminals being most suitable for the purpose. Its advantage is the delicacy with which it can be graduated, but it does not furnish any absolute standard, and is chiefly useful for comparing the sensitiveness of corresponding regions on the two sides.

Sensibility to pain may be changed with or without sensibility to touch. Its loss is termed "analgesia," but is often included in the general term "anæsthesia." A painful sensation is often felt more acutely than normal; this is called "hyperæsthesia," or, more correctly, "hyperalgesia." Occasionally a touch on the skin gives rise to pain, but it is probable that this is due to the stimulation of the over-sensitive nerves of common sensibility, and is not an intensification of a tactile sensation. Both tactile and painful impressions may produce sensations that are abnormal in character, described as "thrilling," "tingling," etc. This perverted sensation has been termed "paræsthesia," or "dysæsthesia," words that have also been applied to purely subjective sensations.

Sensibility to temperature is usually affected with sensibility to pain, but often not in the same degree, and one may be impaired without the other. There may be an absolute inability to recognize either heat or cold as such, or slight degrees of each may be unperceived, while considerable degrees are recognized correctly. In the latter case there is impairment of the power of perceiving differences in temperature, analogous to the defect in the perception of differences in pressure. There may be a perverse sensation, whereby hot objects feel cold, and cold objects hot. The pain that extreme degrees of temperature normally produce may be felt with undue readiness or less readily than normal.* When perception of pain is delayed, that of temperature may be delayed also. It must be remembered that, in health, this sensation is less rapid than that of pain, because time is required to raise the temperature of the skin sufficiently to stimulate its nerves. For coarse examination, hot and cold spoons may be employed; for ascertaining the power of differential discrimination, test tubes, containing water at different temperatures, are necessary. For practical purposes, the result thus obtained is hardly commensurate with the time required for the examination.

It is necessary always to test separately the sensibility to heat and to cold, as there is strong reason to believe that these are subserved by different nerves. If very minute points of the skin are examined, it is found that at some points only heat, at others only cold, is perceived. The stimulation of points of the skin by electricity shows also that at some points the peculiar electrical sensation is produced, at others, sensation of cold, at others, sensation of heat. Further, the points identified as "cold points" and "warm points" by the one method, correspond with the points ascertained by the other method.† The conclusion from these observations is confirmed by the fact that in disease the sensibility to heat and to cold may be affected in different degrees.

The temperature sense varies much in different parts. Sensibility to cold is least at the epigastrium, that to heat least on the back. In the leg, sensitiveness to both heat and cold is greatest at the knee, and lessens upward and downward.

Muscular Sensibility.—The term "muscular sense," as often used, includes more than one form of sensation. There is, first, a true muscular sensibility. The muscles are abundantly supplied with sensory nerves, which are stimulated by pressure and by extension. They are said to end in the interstitial tissue between the muscular fibres (Tschirjew). A painful sensation is readily produced by compression of a muscle, and the pain of cramp, distinctly felt in the substance of the muscle, is probably due to the compression of these nerves by the extreme shortening (and therefore widening) of the muscular fibres in their contraction. After cramp, the nerves are left in a state of exalted excitability, so that extension of the muscle gives rise to pain that is not produced in the normal state. The pain caused by a strong electrical stimulation is probably due to the excitation of these nerves, either direct or by their compression. They appear to subserve the consciousness of contraction which is involved in the recognition of resistance (such as the estimation of a weight), and also, in part, the recognition of posture, even on passive movement. The muscles adapt themselves to all passive movements, and the impression of the elongation or contraction of the muscle probably enters, with the impressions from the skin, etc., into the perception of posture. When the limb is at rest, sensory impressions from the skin and joints probably constitute the chief source of information.

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* See on this point Donath, *Arch. f. Psych.*, Bd. xv.

† Blix, *Zeitschr. f. Biologie*, Bd. xx, p. 141; Eulenberg, *Zeitschr. f. klin. Med.*, Bd. ix, Heft ii. The observations have also been corroborated by Goldscheider and Herzen.

In some morbid conditions the muscular sense is lost, the patient cannot appreciate the difference between light and heavy objects. A poker and a feather seem to be of the same weight. He is also unable to recognize the posture into which his limbs are put by another person. This loss may occur when tactile sensibility is unimpaired. Conversely, the sensibility of the skin may be lost, while that of the muscles is unaffected. This separate affection of cutaneous and muscular sensibility is often seen produced by unilateral lesions of the spinal cord. This question is further considered in the account of the conducting paths of the cord.

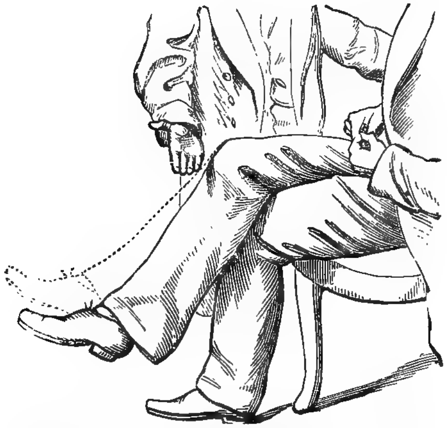
The chief tests for the muscular sensibility are the following: (1) The power of appreciating differences of weight, *i. e.*, variations in the resistance to contraction. In this examination the patient's eyes should be closed, and the objects used should be of uniform size. Leather balls, like cricket balls, containing various weights, are in use at the Queen's Square Hospital for this purpose. It is desirable to reduce the stimulation of the cutaneous nerves to a minimum, by placing the weights in a bag suspended by a string to the part to be tested so that only a small area of the skin shall be pressed upon. The form of the weight is then of little importance. The point to be tested is the power of discriminating differences in weight. This is greater than in the case of the nerves of the skin, since a difference of one-fortieth of the total weight can be recognized under normal conditions. (2) The power of recognizing the posture into which the limb is put; if cutaneous sensibility is normal the parts handled must be grasped firmly, and pressed on both sides, so that the direction of pressure may not suggest the posture. Several observations should be made to eliminate the chance of accidental correctness. (3) The sensitiveness of the muscles to deep pressure. (4) Their sensitiveness to electrical stimulation. It is difficult to test accurately the electro sensibility unless the sensitiveness of the skin is removed, as by the injection of cocain.

Another source of information regarding motor processes is afforded by the degree of central innervation, *i. e.*, the activity of the motor centres. This was pointed out by Sir Charles Bell. It is the innervation, and not the actual muscular contraction, that determines the strength of associated movements, and influences the centre concerned in maintaining the equilibrium of the body. We see this clearly in the case of the muscles of the eyeball. If an ocular muscle is weak, and an increased effort is necessary to direct the eye toward a given object, the object is referred to the place, not toward which the eye is directed, but toward which it would be directed by the increased innervation if the muscle were normally strong. If the patient tries to touch the object, the hand is moved in accordance with the innervation, and goes too far, beyond the object. Hence it is clear that the guide to other motor centres is the central innervation, and not the accomplished movement,—not, therefore, the peripheral stimulation of the muscle nerves. It is equally clear that it is the peripheral stimulation of the muscle nerves that is concerned in the sense of resistance, because the appreciation of weights is not interfered with by simple weakness. It is doubtful whether the impression of motor innervation affects consciousness so as to constitute an actual sensation, comparable to that due to the other sensory nerves, or even to the muscle nerves.

Reflex Action.—Many symptoms of disease of the nervous system are due to derangement of the various reflex actions. These are numerous, and we can now only consider their general characters. Each action is effected through an afferent sensory nerve, an efferent motor nerve, and a system of nerve cells and fibres connecting the two in the spinal cord, the "reflex centre." The centre is usually a complex and often extensive structure, and in it are paths of different "resistance," determining the form of reflex action and its extent, according to the source and intensity of the sensory impression. The reflex centre is between the roots of the sensory and motor nerves concerned, and with them constitutes what may be termed a "reflex arc." The sensory impulse may not only excite a motor process, it may also pass up to the brain, and affect consciousness as a sensation. Further, the reflex centre is to some extent under the control of the cerebral centres.

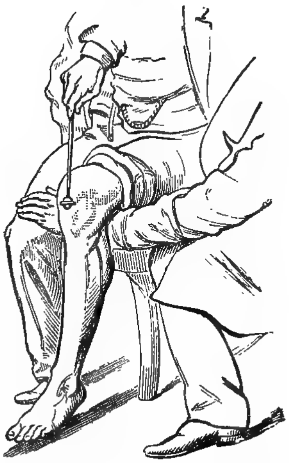
Cutaneous Reflex Action.—Two chief forms of reflex action must be distinguished. The first is that excited by stimulation of the skin, more readily by a gentle stimulation, as a touch, than by a strong, painful impression. The cutaneous reflex actions may be excited at almost any part of the skin, but at some parts they are very definite in character, and are distinguished by special names. The most important are the "plantar reflex," from the sole; the "gluteal reflex," a contraction in the gluteus when the skin over the muscle is stimulated; the "cremaster reflex," a retraction of the testicle on stimulation of the skin on the inner part of the thigh; the "abdominal reflex," in the muscles of the abdominal wall when the skin over the side of the abdomen is stroked; the upper part of this reflex is a very definite contraction at the epigastrium, and has been termed the "epigastric reflex." A series of reflex actions may be obtained in the muscles of the back, the highest being in the muscles of the scapula. These spinal reflex actions differ in their excitability in different individuals, and are always more readily produced in the young than in the old. In the region of the cranial nerves, the most important reflexes are those of the eye; the conjunctival reflex, the contraction of the iris on exposure of the eye to light, and its dilatation on stimulation of the skin of the neck.

FIG. 1.



The Knee Jerk.—The dotted line indicates the movement which follows the blow on the patellar tendon.

FIG. 2.



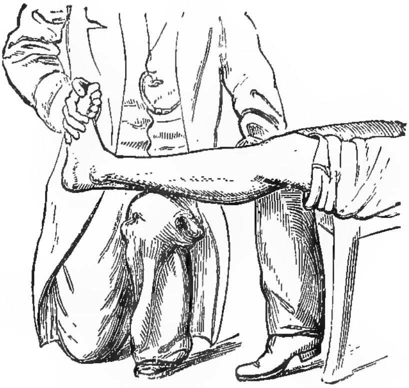
The Knee Jerk.—Method of obtaining it when it is not readily produced in the ordinary way.

Muscle Reflex Action; "Tendon Reflexes."—The second group of phenomena which depend on reflex action are those which have been termed "tendon reflexes." They were first systematically studied by Erb and Westphal, but previously partially recognized and employed in diagnosis by Charcot.

The first of these is the jerk of the leg which occurs when the patellar tendon is tapped. It has been called the "knee phenomenon" by Westphal, the "patellar tendon reflex" by Erb, the "knee jerk" by myself. To obtain the jerk, the knee must be flexed so that the quadriceps femoris is gently extended, and the leg must be free to move. If then the patellar tendon is struck, the quadriceps contracts and jerks the leg forward. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle. The posture commonly employed is with the leg to be tested across the other, the knee of the supporting leg being at a right angle (Fig. 1). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the observer may place his arm beneath the patient's thigh, just above the knee, and rest his hand on the patient's other knee (Fig. 2). Children may sit on the edge of a chair, adults on the edge of a table. If the legs are vertical, the effect of the blow and of the muscular contraction must be carefully distinguished. If the bent fingers of each hand are interlocked, and the hands pulled strongly, the knee jerk occurs more readily (Jendrassik). It is essential that the muscles should be relaxed from voluntary contraction. Any contraction of the flexors may be ascertained by feeling the hamstring tendons, and pressure on these by the fingers often helps to secure relaxation. The blow may be given by the side of the hand, a percussion hammer (Fig. 2), or a stethoscope with an india-rubber edge to the ear piece. If its existence is doubtful, the skin should be bared. In many cases the movement may be obtained by a downward blow upon the patella, by a blow on the quadriceps tendon above the patella, or by a blow on the substance of the muscle, almost as readily and strongly as by a blow on the patellar tendon. In cases in which it is in great pathological excess, it may even be excited by a blow on the tibia. When it is in excess, it may be conveniently brought out in a somewhat different way. As the patient lies in bed, the finger of one hand is placed across the quadriceps tendon just *above* the patella, and the patella pushed down, so as to make the quadriceps tense. The finger is then percussed in the direction in which the patella is being pushed, so as suddenly to increase the tension in the muscle. The blow is instantly followed by a contraction, jerking the patella and finger upward. Very often this single contraction is immediately succeeded by a second, and this by a third, and so on—a series of quick clonic contractions, "clonus," recurring as frequently as eight per second. By grasping the patella firmly, and suddenly pushing it downward, so as to make the muscle tense, this clonus may also be set up, as Erb has shown. It may continue as long as tension is kept up, but instantly ceases when the muscle is relaxed.

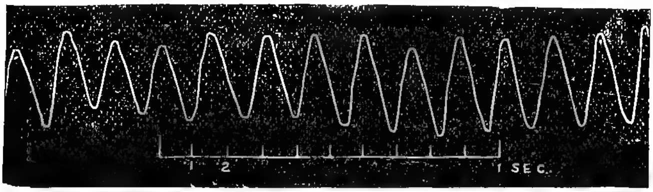
The next important phenomenon belonging to this group occurs at the ankle joint. If the calf muscles, which are connected with the Achilles tendon, are made tense, and this tendon is tapped, the muscles contract, causing a slight extension movement of the foot; just as the muscles of the thigh contract when the patellar tendon is struck. In cases in which these phenomena are excessive,—just as sudden tension in the thigh muscles will cause a contraction, followed by others in a continuous series—so, in such cases, if the calf muscles, which extend the ankle joint, are suddenly put on the stretch by pressing the hand against the sole of the foot (Fig. 3), a quick contraction occurs, instantly ceasing, but if the pressure is kept up, instantly renewed, and recurring as long as the tension is maintained, as a clonic series of spasmodic contractions—the "ankle clonus" or "foot clonus" (or "foot phenomenon"—Westphal). It can often be obtained best when the knee is not completely extended. The movement is very uniform, from six to nine contractions occurring per second. By attaching a writing point to the foot, and making it trace a line on a revolving cylinder covered with blackened paper, tracings may be obtained (Fig. 4), which are almost as regular as the tracings of a tuning fork. The foot clonus can be more frequently obtained than the clonus in the extensors of the knee, but the two have the same time and are evidently of the same nature. A clonus quite similar may sometimes be obtained in other muscles—peronei, flexor brevis pollicis, flexors of the fingers, etc.

FIG. 3.



Method of Eliciting the Foot Clonus.

FIG. 4.



Tracing of the Foot Clonus in Paraplegia.
(The tracing reads from right to left.)

When a tendon is tapped, and its muscle contracts, the occurrence has somewhat the aspect of a reflex action. It was assumed by Erb that the contraction is a true reflex action, the stimulus being the excitation of nerves in the tendon. Hence it has been termed "tendon reflex." This view has received apparent confirmation by the discovery of certain facts: (1) That there are nerves in tendon. (2) That these phenomena depend for their occurrence on the integrity of the reflex path to, through, and from the spinal cord, and are arrested by a lesion in this path. By experiments on animals (in whom similar contractions may be obtained) it has been found that they are prevented by division of the nerves to the muscles, by division of either the anterior or posterior roots of the spinal nerves, or by destruction of the spinal cord.* The knee jerk cannot be obtained in locomotor ataxy (damage to the posterior nerve roots), or in infantile paralysis (damage to the gray matter, the reflex centre). (3) That these phenomena are in excess in some cases, in which the reflex action from the skin is in excess.

These facts certainly prove that some reflex influence is concerned in the production of the phenomena. But (as Westphal has always maintained) they do not necessarily prove that the contractions depend on a simple reflex action from the tendons. Many of the facts cannot be explained by the "tendon reflex" theory. There is the great fact that passive tension is necessary for the tap on the tendon to be effective, and that when the phenomena are in excess, sudden tension alone will suffice to develop the contraction. But tension acts upon the muscle as well as upon the tendon, and frequently the contraction may be distinctly excited by stimuli which act on the muscle and have no action on the tendon. For instance, in cases in which these phenomena are in excess, if the foot be gently pressed up so as to make the calf muscles tense, and the muscles on the front of the leg be tapped, the calf muscles contract (just as they do when, under the same circumstances, the tendon is tapped), and cause a brief extension movement of the foot. I have termed this the "front-tap contraction." It is a very delicate test of increased irritability, and it is also of considerable theoretical interest, since we have in it a contraction developed by a stimulus which does not in any way affect the tendon. It can affect the gastrocnemius directly, for by placing the hand on the calf, a vibration may be felt through the leg. If the tibia, instead of the muscle, is tapped, the contraction is much slighter, or does not occur. Moreover, a tap on the tendon itself only excites the contraction when it increases the tension of the tendon; *i. e.*, when it acts upon the muscle also. In the case of the Achilles tendon, this may be easily demonstrated with a little care. A gentle tap on the side of the tendon will excite the contraction as readily as a tap on the back of the tendon, but if the other edge of the tendon is supported (as by the fingers of an assistant), the same tap will no longer be effective, because it no longer increases the tension. The strongest proof, however, of the independence of the phenomena on any stimulation of the tendon is afforded by the experiments of Tschirjew, who divided carefully all the nerves to the patellar tendon, and still found that the tap upon it made the tense muscle contract.

Thus the evidence seems conclusive that the contractions are not excited by stimulation of the nerves of the tendon, but that the stimulus originates in the muscle, the tendon being only, so to speak, an instrument by which that stimulation is produced. But if the muscle is stimulated and then contracts, is not the contraction excited locally, as Westphal has, from the first, urged? A reflex action takes a certain time, which is needed for the stimulus to travel to and from the cord, and for the reflex process to occur in the centre. According to received physiological data, an interval of at least one-fifteenth of a second would be needed for the knee jerk, if it were a reflex process, and rather more for the movement at the ankle. I have found that when the Achilles tendon, or the front of the leg is tapped, the resulting contraction occurs in about one-thirtieth of a second.† The interval for the knee jerk has been found to be about one twenty-fifth or one-thirtieth of a second.‡ If the patella is pressed down and tapped, in the way I have just described,

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* See Tschirjew, *Archiv für Psychiatrie*, Bd. viii, Heft 3.

† *Med.-Chir. Trans.*, *1879*, p. 292. The measurement has been since confirmed by Waller, *Brain*, July, 1880.

‡ .039 sec. Burckhardt; .032–.034 sec. Tschirjew; .04 sec. Brissaud; .03–.04 sec. Waller; .03 sec. Eulenberg. Some measurements which I made of the interval (*Med.-Chir. Trans.*, *1879*, p. 275) gave a longer interval, probably in consequence of the movement of the foot being taken as the indication of the commencing contraction. "Load" will increase greatly the period of latent stimulation, probably by causing the

I have found that the interval between the tap and the resulting contraction is often not more than one-fortieth of a second. The shortness of the interval makes it difficult to believe that these contractions can be reflex, and supports the theory that they are excited locally.

But to this view, that the contractions are excited locally, is apparently opposed the fact that they are prevented by whatever lesion arrests reflex action. Some have endeavored to explain the discrepancy by the suggestion that reflex action may occur in a much shorter time than is commonly supposed. This suggestion is not at present justified by any known facts. Another and, I think, much more probable explanation is this. If we regard the contractions as local, we have still to account for the irritability which permits the local stimulus to cause a contraction. This irritability is developed by passive tension. If the muscle is relaxed, the fibres may contract if they are struck directly, just as do the fibres of a separated frog's muscle, but no contraction can be produced by striking the tendon. Hence I have suggested that the tension excites, by a reflex influence, a state of extreme irritability to local stimulation,—such as that of a tap on the tendon, or such as the vibration from a tap near the muscle, or from a tap on the bone to which the tendon is attached,—which thus excites a visible contraction.*

The explanation receives some confirmation from the very interesting observations of Tschirjew.† He has found that if the nerve to a separate muscle be divided, the muscle remains of just the same length. If, however, a weight be first attached to the muscle, when the nerve is divided the muscle lengthens. This shows that the tension does excite a slight contraction which is dependent on a central influence. It is in this condition only that the local stimulation is effective. If the tension put on a muscle is gentle and gradual, it may only develop the irritability, and an additional local stimulation is necessary to produce a visible contraction. If, however, the tension is sudden and forcible, it not only develops the irritability, but produces visible contraction in the muscle thus rendered irritable—as in setting up the foot clonus. I have shown that the relaxation of the muscle, between the successive contractions, is not complete: there is a persistent residual contraction, *i. e.*, a tonic contraction on which the clonic contractions occur. When one clonic contraction is over, the tension continuing, a second is instantly developed.

The sensory nerves of muscles have been shown by Tschirjew to commence, not within the muscular fibres, but in the interstitial connective tissue. The afferent impulse produced by tension is apparently due to the tension acting on these nerves: the visible contraction is excited by tension or vibration affecting the muscular fibres themselves. The latter is ineffective unless the muscles are brought into a state of special excitability through the cord. The reality of an afferent impulse from the muscle, produced by tension, is demonstrable to any one who will permit his ankle to be suddenly flexed. A distinct pain is felt in the muscle (none, be it observed, in the tendon). It is not surprising, therefore, that this afferent impulse should, very often, not merely develop the reflex excitability, or tonic contraction, but also cause a more widely-spread reflex action. The attempt to get the foot clonus, for instance, will cause a flexion of the hip joint; the attempt to obtain the knee jerk may cause a movement in the opposite leg or a start back of the body. But these reflex contractions, if carefully observed, confirm the theory which has been put forward in the preceding pages, for they distinctly succeed, at an appreciable

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initial contraction to expend itself on the elasticity of the muscle. The measurements given above were obtained by recording the commencing contraction of the muscle.

* This explanation was originally given for the "foot phenomenon" alone (*Med.-Chir. Trans., 1879*, p. 295). I now think that it is equally applicable to the knee jerk. The evidence of the identity in nature of the two is very forcibly stated by Waller (*loc. cit.*).

† *Reichert und Du Bois-Reymond's Archiv, 1879.*

interval, the local contraction. Burckhardt has found that the latent interval for a skin reflex is three times as great as for the knee jerk. I think that this theory of reflex irritability and local stimulation affords a full explanation of all the relations of these phenomena to the central nervous system, and to the other phenomena of disease, and it is the only theory which adequately explains them.

It seems, therefore, that the term "tendon reflex" is altogether inaccurate. The phenomena are, according to the explanation above given, dependent on a "muscle reflex" irritability, which has nothing to do with the tendons. If we wish to describe them by a general term, it is best to employ one which does not involve any special theory of their nature. They may be termed "tendon muscular phenomena," but the intervention of tendons is not necessary for their production; the one condition which all have in common is that passive tension is essential for their occurrence, and I have suggested* that they be termed *myotatic* contractions (*τατικος*, extended).† The irritability, on which they depend, is due to and demonstrative of a muscle reflex action which depends on the spinal cord. It is highly probable, as Tschirjew suggested, that the condition on which the myotatic irritability depends, is identical with muscular "tone." Since the experiments of Heidenhain, it has been generally admitted that "tone" depends on tension, and is a reflex process.‡ A true "tendon reflex" may be excited by pinching the tendon, which is a sensitive structure; § but the movement produced is a start of the whole limb, precisely such as results from a pinch of the skin.

The knee jerk is sometimes absent in diabetes. We do not know by what mechanism it is abolished in this disease.

Paradoxical Muscular Contraction.—This name has been given by Westphal‖ to a slow tonic contraction occurring in a muscle when suddenly relaxed, or rather when its course is suddenly shortened. It is best seen in the tibialis anticus. If the foot is grasped, and passively flexed on the leg, the tibialis anticus contracts, sometimes after an appreciable interval; its tendon stands out, and the contraction keeps the foot flexed for some minutes (in one case for twenty-seven), and then slowly relaxes. Repetition lessens the contraction. It is not voluntary (although a voluntary contraction may simulate it), for it may be unilateral. The contraction produced by faradaism or by the will may be similarly lasting. The same phenomenon may be observed in the extensors of the toes, sometimes in the flexors of the knee, rarely in the arm muscles. Its mechanism is uncertain,—whether reflex or not, whether produced through the muscle itself, or through the tension on the antagonists, as Erlenmeyer has maintained. The former is perhaps the more probable. It is interesting as an excess of the adaptation which muscles undergo on their passive elongation or shortening. This is probably one element in the appreciation of posture. In the cases that have presented this phenomenon there has been no muscular rigidity, no great excess (even sometimes a loss) of the myotatic irritability. It occurs sometimes in the early stage of tabes, and in some other affections, but its significance is not known. A phenomenon somewhat similar occurs in hysteria (Féré); the facts of the hysterical cases, and the effects of chloroform observed by Westphal, suggest a relation between the condition and cataleptoid states.

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* "Diagnosis of Diseases of the Spinal Cord," 2d edit., 1881, p. 29.

† If it should ultimately be proved (which is very improbable) that so short an interval as one-fortieth of a second is sufficient for a reflex action, and that each contraction is reflex, the term "myotatic" will still be accurate, since it will remain true that tension is essential for the production of these contractions.

‡ By some interesting researches recently published, Mommsen has reached the same conclusion, that muscular tone is dependent on a muscle reflex action excited by tension acting on the sensory muscle nerves. (*Virchow's Archiv*, Bd. 110, p. 22.)

§ The sensitiveness of tendons was noted by Cowper at the beginning of the last century, in a case in which he sutured the Achilles tendon. (*Phil. Trans.*, No. 252, p. 153.)

‖ *Arch. f. Psych.*, Bd. x, p. 243.

Changes in Nutrition.—The nutrition of the tissue elements is largely under the influence of the nervous system. Whether this influence is exerted through special "trophic" nerves, or merely through the motor, sensory, or especially through the vasomotor nerves, is a question that has been much discussed, and is still undecided. Many disturbances of nutrition can be explained without the hypothesis of special trophic nerves. The fact regarding these changes that is best established and most important, is that the most acute disturbance of nutrition is the result of irritation of the nerve trunks and centres, and is in proportion to the intensity of that irritation. If a thread is passed through each sciatic nerve, and one is also irritated by the application from time to time of irritant liquids, trophic changes occur in that limb with greater rapidity, and far greater intensity, than in the other. (Lewaschew.)

Nutritive changes in the skin are easily recognized, but they differ much in their character according to their acuteness. When they are rapid, and due to very acute irritation of the nerves or the spinal cord, such as attends acute myelitis, the temperature of the limb is raised, the vessels readily dilate, and remain dilated for a long time, bullæ form, containing a dark-colored liquid, and slight pressure occasions a slough. These changes sometimes occur spontaneously, but are more often excited by some cutaneous irritation, and very slight irritation of the skin will suffice to produce them. Trifling pressure will set up a slough, and extensive vesication may result from the application of a hot-water bottle that is not more than pleasantly warm to a healthy hand. When the nerve irritation is intense, effusion may occur into the joints.

In the case of the fifth nerve, acute trophic changes occur in the eyeball chiefly when the disease involves the Gasserian ganglion, or the nerve in front of it. To produce the same effect, a lesion behind the ganglion has to be more irritating than one in front of it. We do not know whether a similar relation obtains in the case of the spinal nerves and the spinal ganglia. Very intense changes in nutrition are common in acute inflammation of the spinal cord.

The alterations in nutrition in chronic lesions (which have been carefully studied by Paget and Weir Mitchell) differ considerably from those that result from acute irritation. There is a slow change in the nutrition of the skin, which becomes red, thin, and shiny—the "glossy skin" of Paget. The subcutaneous tissue also wastes, so that the finger tips become pointed. The growth of the hair and nails is retarded, and the latter become brittle. The bones may suffer in their nutrition, and may break more easily than in health. If the lesion occurs during the period of growth, this is retarded, and the affected limb becomes shorter than the corresponding limb on the other side. We do not yet know by what nerve fibres this influence on growth is exerted.

The *muscles* suffer in their nutrition from lesions of the motor nerves. The bulk of a limb depends chiefly on the muscles, and therefore the interference with their nutrition is quickly shown by a diminution in the size of the part. At first there may be merely flabbiness, and an appearance of wasting that is not confirmed by measurement, but there is soon an actual diminution in the circumference of the limb. In extreme cases all the muscular tissue disappears, and the contour of the limb is considerably changed. In comparing the size of the limbs on the two sides, it should be remembered that the limbs on the right side are normally somewhat larger than those on the left side, and that the difference varies according to the occupation of the individual, and the extent to which this involves an excessive use of the limbs on one side. In measuring, great care is necessary to secure, as nearly as possible, the same conditions on each side, both as regards the state of the muscles and the place of measurement. It is best, wherever possible, to take the maximum measurement in each part, rather than to attempt to make the measurement in the same place. In the calf, the maximum circumference should always be taken. In the thigh, accurate measurement is extremely difficult, because a maximum cannot be taken. We may endeavor to measure at the same point in the thigh, at the same distance from the condyle, but it is very difficult to be exact, and a more accurate comparison can often be made by taking the minimum circumference above the knee, in spite of the fact that the muscular tissue there is small, and the difference less than it is elsewhere. In the forearm the best result is obtained by taking the maximum measurement around the muscular prominence below the elbow, over the supinator longus. In the upper arm, the circumference is nearly the same in the middle third, and the middle of the humerus may be measured, with little risk of error. In all cases care must be taken to draw the tape equally tight at each place. It is easier to do this with a flexible steel measure than with an ordinary tape measure. Exactness in this respect is best secured by having a spring at one extremity of the tape, with an index that will show the tension, which can thus be kept the same at each place.

Electrical Irritability.—The nerves and muscles are excitable by electricity, and the excitability is changed by disease, of which the change is often an important symptom. It indicates the state of nutrition of the nerve fibres and muscles, and from this we can often draw important inferences regarding the condition of the centres. An outline of the most important facts regarding electricity in its application to medicine, is given in the Appendix, but it may be well to mention here the chief facts of diagnostic importance.

In the normal state, nerve fibres are stimulated by either the induced or the voltaic current, the stimulation of the motor nerves being shown by contraction in the muscles supplied by them, that of the sensory nerves by the sensation that is caused. The contraction of the muscles is continuous when the faradaic current is applied, but if the isolated shocks of which the current consists are separately passed, each causes a brief, momentary contraction. When the voltaic current is applied, contraction occurs, with a current of moderate strength, only when the strength of the current is changed, and chiefly when the current commences or ceases to pass, *i. e.*, when the circuit is "made" or "broken." The stimulation of the sensory nerves is greatest at those times, but occurs also, in a much slighter degree, during the whole time that the voltaic current is passing. Hence this is probably the case also in the motor nerves, although the stimulation is too feeble to produce a contraction in health with a strength of current that can be borne. In proportion as the nutrition of the nerve fibres is impaired, their excitability is lowered and a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i. e.*, when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple, because in them there are two excitable structures—the terminations of the nerves, and the muscular fibres themselves. Of these the nerve fibres are the more sensitive to faradaism, and the faradaic stimulation of a muscle under normal circumstances is by means of these motor-nerve endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradaism than the nerve, apparently because they are incapable of ready response to a stimulus so very short in duration as are the shocks of which the faradaic current consists. (The proof of this consists in the fact that under the influence of curara, which removes the excitability of the terminations of the motor nerve, the muscle requires a stronger faradaic current to stimulate it than in the normal state.) But when the nerve is degenerated the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state; a contraction occurs when the circuit is completed or broken—distinctly slower than that which occurs when the nerve fibres are intact, and due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that produced by the faradaic shock, is ground for believing that, in health, the voltaic, as well as the faradaic current, causes the muscle to contract chiefly by exciting the motor-nerve endings. When the motor nerve is degenerated, and will not respond to faradaic or voltaic stimulation, the muscle also loses all its power of response to the former. Apparently the nerve degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradaism, which it possessed in the normal state, is lost. But the response to the voltaic current remains, and becomes quickly more ready than in health, doubtless in consequence of nutritive changes which develop what the older pathologists called, truly enough, "irritable weakness." Moreover, there may commonly be observed a change in the readiness of response to a certain mode of stimulation with voltaism—a "qualitative" change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before closure contraction occurs at the positive pole. But, in the morbid state we are discussing, closure contraction may occur at the positive pole as readily as at the negative, or even more readily—and contractions, when the circuit is broken, occur far more readily than in the normal state. This condition, then—faradaic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve fibres are degenerated; and if we test *them* we shall find no response to any stimulus, voltaic or faradaic. It occurs when the nerves are separated partially or completely from their motor-nerve cells, and if no such separation exists, it indicates an acute degenerative change in those nerve cells.

But the motor-nerve cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the irritability of the fibres is lessened gradually and slowly. The irritability of the intramuscular nerve endings is lessened in the same degree as that of the nerve trunks, and we have a diminution to both faradaism and voltaism. The nutrition of the muscular fibres is slowly, gradually, impaired; and when the nerve fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre irritability is lost and the muscle-fibre irritability retained; hence there is no condition of lost faradaic and increased voltaic irritability such as characterizes the degenerative reaction just described. Irritability is changed to the one form of stimulus just as to the other.

Between these two forms there are intermediate conditions. For instance, the nerves may present normal irritability, and the muscle the increased voltaic irritability and changed order of contraction met with in degeneration. Probably, in these cases, some nerve fibres are degenerated, and lead to the increased irritability of some muscular fibres. In both nerve and muscle the character of the reaction is determined by the more irritable structures; hence it is normal in the nerve and altered in the muscle. This has been termed by Erb the "middle form" of degenerative reaction. It would be more accurate to call it the "mixed form."

The various changes in irritability have been thought to indicate the existence, and various affection, of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor-nerve cells. Remembering that the nerves and muscles contain fibres which suffer in different degrees, the phenomena at present ascertained may all be explained on the simpler principle stated, without the assumption of these special centres, of the existence of which there is, indeed, no evidence.

Frequently the lowered irritability of degeneration in the nerves is preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, "functional" maladies.

THE MUSCLES: THEIR ACTION AND PARALYSIS.

Disease of the motor nervous system is largely manifested by loss of muscular action. Individual muscles, as well as groups of muscles, are often separately affected. Hence it is desirable to consider the symptoms of the paralysis of the more important muscles, before we enter on the study of special diseases.

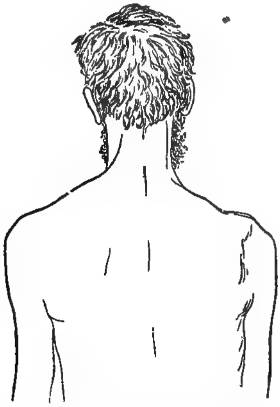
The symptoms of the palsy of any muscle is a loss of its normal action, and a knowledge of this action is essential for the comprehension of those symptoms. The two must therefore be considered together. They are positive and negative aspects of the same facts. It may be well, at the same time, to mention the nerve by which each muscle is supplied.

The action of muscles is threefold. (1) By their tonic contraction they maintain the parts in a certain posture, independently of voluntary effort. By actual contraction they (2) produce certain movements, and also (3) oppose the action of other muscles by a feebler contraction and thus steady the movement that results.

The complex way in which muscles act together, and modify each other's effect, renders the subject a very large one. Here, only an outline can be given of the more important facts concerning the more important muscles. The reader who desires to pursue the subject further can do so in the "Physiologie des Mouvements," of Duchenne, to whose researches our present knowledge is largely due.

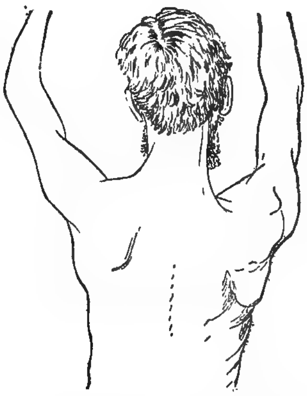
The **Diaphragm** (phrenic nerves from the fourth and fifth cervical), although a double muscle with two nerves, habitually acts as a whole, the two halves contracting simultaneously and diminishing each lateral curve of the arch. The central tendon descends but little. The abdominal viscera are depressed, and the parietes protruded. If the hand is placed beneath the ribs, the descent of the viscera beneath the diaphragm can be felt. When the diaphragm contracts alone, as when the intercostals are paralyzed, or the phrenic nerve is faradized, the ribs to which the muscle is attached are slightly raised during its action, and this elevation causes a slight expansion of the thorax. In ordinary breathing this expansion is lost in the action of the intercostals. In paralysis the inspiratory protrusion of the upper part of the abdomen is lost; it even recedes during inspiration instead of advancing, and a descent of the viscera can no longer be felt by the hand. There often results a remarkable alternation in the respiratory movements of the thorax and abdomen, the retraction of the one corresponding to the protrusion of the other.

FIG. 5.



Paralysis and wasting of trapezius; alteration in contour of shoulder at rest.

FIG. 6.



Ditto, when the arms are raised (the right one being aided by another person).

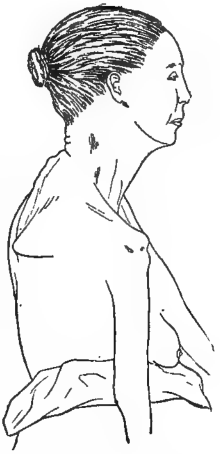
Sterno-mastoid (spinal accessory nerve), passing from the sternum and adjacent part of the clavicle to the mastoid process, inclines the head toward, and rotates the face from, the side on which the muscle contracts. Both muscles together support the head in the vertical position, and, if it is bent back, they bring it forward into, but not beyond, this position. Paralysis of one muscle has no influence on the position of the head, and but little on its movements. Other muscles supplement the loss. There is no such thing as a "paralytic torticollis." In palsy of both muscles, the head can be balanced in the vertical position, but if it falls back, it can be brought forward only with great difficulty. Each sterno-mastoid is associated in action with the muscles of the other side; it is a "contra-lateral muscle." For instance, in using the right arm, the head is turned to the right by the left sterno-mastoid. This association is sometimes reproduced in disease.

MUSCLES MOVING THE UPPER LIMBS.

Muscles Moving the Scapula and Shoulder Joint.—The *Trapezius* (spinal accessory, lower cervical and upper dorsal nerves) consists of three parts. The first, from the occipital bone to the outer end of the clavicle, is rarely used except in breathing (respiratory portion, Duchenne). The second part is that which passes from the lig. nuchæ, lowest cervical, and upper three dorsal spines, downward and outward to the acromion and outer part of the spine of the scapula. The lowest part passes from the dorsal spines below the third, outward and partly upward, to the inner half and base of the spine of the scapula. The second part is the chief elevator of the scapula and shoulder. With the third part it brings the scapula toward the spine, and puts the shoulder back. Both parts tend to rotate the scapula—acromium up, lower angle out. By this rotation the arm is carried above the horizontal level, to which the deltoid raises it. Paralysis of the highest part has little influence on the movement of the scapula, but causes a change in the contour of the neck (Fig. 5) especially conspicuous on deep inspiration. The change in the shape of the neck is very great, when the arms are raised, if the whole trapezius is wasted (Fig. 6). In palsy of the middle part, the elevation of the shoulder is imperfect; in that of the third part, the scapula is farther from the spine than normal. In palsy of all parts, the scapula becomes rotated (acromion down, inferior angle in) by the weight of the arm and the contraction of the opponents (Fig. 7). The rotation may mask the displacement outward, due to the paralysis of the lowest part. If the clavicular part remains, there may be no rotation, but the scapula is lower than normal.

The *Rhomboids* (fifth cervical nerve, by a branch that passes through the scalenus) first rotate the scapula on the outer angle, moving the lower angle inward, and then move the whole scapula upward and inward. In strong elevation, they aid the trapezius, which prevents the rotation of the scapula. The rotatory action aids forcible depression of the raised arm. The muscles also fix the scapula for the action of the teres major. Their tone helps to keep the scapula against the thorax (opposing the pectoralis) and in its vertical position (opposing the serratus), and hence, in paralysis, the edge of the scapula, at rest, stands out a little, leaving a furrow, and the scapula is slightly rotated (lower angle out). Movement is but little interfered with by the paralysis of the rhomboids, the most important effect being that the movement backward of the raised arm by the teres and deltoid is feeble for want of the fixation of the scapula.

FIG. 7.



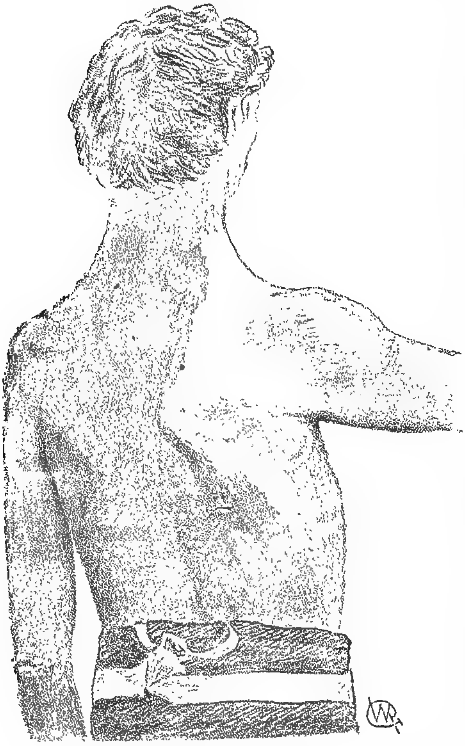
Paralysis and wasting of deltoid and trapezius; rotation of scapula, from weight of arm, in consequence of the paralysis of the trapezius; progressive muscular atrophy.

The *Levator anguli scapulæ* (third cervical nerve) first rotates the scapula on the outer angle and then raises it. The muscle is usually paralyzed with the trapezius, and then the scapula falls, but the special effect of its palsy is lost in that of the trapezius. If the levator is preserved, and the trapezius paralyzed, there is great rotation of the scapula, which is, as it were, suspended by its inner angle (Fig. 7).

Serratus magnus (posterior thoracic nerve, from the fifth and sixth cervical) carries the scapula outward, forward, and slightly upward, when the arm is put forward. It tends to rotate the scapula on the inner angle (acromion up), the lower fibres most powerfully, but this rotation is prevented by the rhomboids and levator anguli. It does not raise the shoulder when the arm is hanging. It helps to fix the scapula when the posterior fibres of the deltoid move the raised arm back. If the scapula is fixed by the rhomboids, the serratus can act on the ribs, and aid forced inspiration. It has most inspiratory effect when the arms are elevated.

In paralysis there may be little change in the position of the scapula at rest, but often there is slight rotation (lower angle in) from the unopposed tone of the rhomboids. When the arm is moved forward by the anterior part of the deltoid, the scapula, no longer held against the thorax and moved forward by the serratus, is rotated on its vertical axis by the action of the anterior part of the deltoid on the humerus, and of the middle part on the scapula. Thus the posterior edge recedes from the thorax, leaving a groove into which the hand can sometimes be placed (Fig. 8). The scapula is, at the same time, rotated; lower angle inward and upward. Elevation of the arm above the level of the shoulder is much weakened, but can be imperfectly effected by the middle part of the trapezius. Loss of the serratus weakens other movements, but does not abolish any. Inspiratory expansion of the thorax, when the arms are raised, is distinctly less on the paralyzed side (Poore).

FIG. 8.



Paralysis of the serratus magnus; eversion and rotation of scapula when the arm is put forward.

The *Deltoid* (circumflex nerve, from the four lowest cervical) abducts the humerus, the anterior and posterior fibres also moving the arm forward and backward respectively. The arm is raised least by the posterior, and most by the anterior fibres, but even the latter only elevate it to a right angle with the trunk. Hence, if raised by the anterior fibres, and then moved back by the posterior, it is at the same time depressed. Elevation above a right angle is by rotation of the scapula (trapezius and serratus). These muscles also fix the scapula for the deltoid, preventing the rotation (acromion down, lower angle in) that the deltoid, acting alone, would cause. In paralysis, abduction of the arm, direct, forward, and backward, is almost lost. All the abduction that remains is a trifling movement by the supraspinatus. An attempt to abduct results in rotation of the scapula and elevation of the shoulder (Fig. 9) from an excessive innervation of the associated trapezius and serratus, which, as we have seen, fix the scapula when the deltoid acts. Paralysis of single parts of the deltoid causes loss of the corresponding movement of the arm, but if the middle part only is paralyzed, there is still a limited power of direct abduction by the conjoined contraction of the anterior and posterior parts, aided by the supraspinatus.

The *Supraspinatus* (suprascapular nerve) abducts the arm, moves it forward, and rotates it in. It thus aids the deltoid. Isolated paralysis of the supraspinatus has little influence on movement or position, but if the deltoid is also paralyzed, the head of the humerus falls away from the acromion far more than when the deltoid is paralyzed alone.

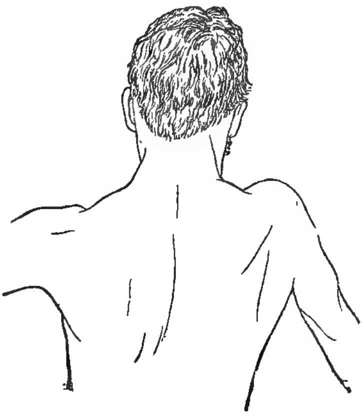
The *Infraspinatus* (suprascapular nerve) rotates the humerus outward, and in paralysis this movement is lost. A difficulty in writing is produced, the movement along the line being by this rotation of the humerus.

The *Teres minor* (circumflex nerve) has a similar action to the infraspinatus, and its palsy has a similar effect.

The *Subscapularis* (short subscapular nerve, from the fifth and sixth cervical) rotates the humerus in, and its paralysis lessens this movement.

The *Latissimus dorsi* (long subscapular nerve, from the brachial plexus—last four cervical nerves) lowers the raised arm, and puts it back; the upper part adducts the scapula, the lower depresses the shoulder by acting on the humerus, which it tends to drag out of the socket. It inclines the trunk a little, and both muscles together extend the trunk. In paralysis, forcible backward depression of the raised arm is lost, and the shoulder cannot be put back without being also raised (by the trapezius).

FIG. 9.



Paralysis of right deltoid; elevation of shoulder by trapezius on an attempt to raise the arm, which is slightly abducted by the supraspinatus.

The *Pectoralis major* (anterior thoracic nerves from the brachial plexus) consists of two muscles, the action of the clavicular and sternal parts being different. The clavicular (which arises also from the highest part of the sternum), if the arm is hanging, brings the shoulder forward and upward, as in shivering; if the arm is raised, it is brought forward and lowered to the horizontal position. The muscle is thus concerned, Duchenne says, in the "cut" of the swordsman and the benediction of the priest. The sternal portion lowers the raised arm from every position, and if the arm is hanging, it draws the shoulder down. Paralysis of the upper part has little effect on the movement of the arm, because the anterior fibres of the deltoid have the same action. It is easily recognized by making the patient put his arms in front of him and press the palms together. In paralysis of the lower part, even with the latissimus, the raised arm can still be lowered accurately, by the weight of the arm and relaxation of the elevators, but it cannot be lowered against even a slight resistance. Thus a blacksmith with this defect could wield a heavy hammer, but could not blow the bellows by pulling downward a cord (Duchenne).

The *Teres major* (short subscapular nerve) approximates the humerus and the outer edge of the scapula, by bringing the former to the side of the trunk, and rotating the latter. The simultaneous contraction of the levator anguli and rhomboids, fixing the inner angle, causes this rotation to raise the prominence of the shoulder. Hence, in a forcible elevation of the shoulder, the arm is pressed against the side, the lower fibres of the latissimus and pectoralis major aiding the adduction. The teres cannot alone put the arm behind the trunk. In paralysis, the elevation of the shoulder, with the arm against the side, is lost.

Muscles Moving the Forearm.—*Triceps* (musculo-spiral nerve).—The long head has an action similar to the teres, but feebler. It contracts when the arm is forcibly lowered, and prevents the displacement downward of the head of the humerus by the actual depressors, the latissimus and pectoralis. All parts extend the elbow, the long head with less force than the others, but its action on the shoulder joint, just mentioned, is important, because forcible depression of the raised arm is often associated with extension of the elbow. In paralysis of the triceps, the elbow can only be extended by the weight of the forearm, and extension against gravitation is impossible. Thus a man with paralysis of the triceps cannot raise his hat in the customary manner. Flexion of the elbow is uncertain, on account of the loss of the antagonistic steadying force.

The *Brachialis anticus* (musculo-cutaneous and musculo-spiral nerves) flexes the elbow simply. Its rare isolated palsy has little effect, since it is supplemented by the biceps and supinator longus.

The *Biceps* (musculo-cutaneous nerve) supinates the forearm if it is pronated, and then flexes the elbow. In paralysis the flexion can still be effected, but the traction on the humerus causes pain at the shoulder, from the loss of the support of the long head of the biceps.

The *Supinator longus* (musculo-spiral nerve) places the forearm midway between pronation and supination, and then flexes the elbow. If it is paralyzed, there is a tendency for supination to accompany flexion (biceps), and if the brachialis anticus is also paralyzed, the elbow can only be flexed when the forearm is supinated.

If the three direct flexors of the elbow are paralyzed, feeble flexion is still possible by the extensors of the wrist, which cross the elbow joint, but only after the forearm has been pronated and the wrist over-extended.

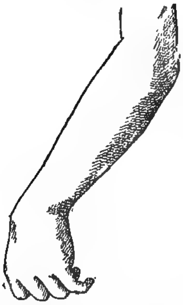
Pronators.—The *p. teres* and *p. quadratus* (median nerve) both pronate strongly, and their palsy causes loss of this movement, but pronation to the mid-position is still possible by the supinator longus.

The *Supinator brevis* (musculo-spiral nerve by posterior interosseal branch) is the only simple supinator. If it is paralyzed, supination can still be effected by the biceps, and also by the supinator longus as far as midway between pronation and supination.

Muscles Moving the Hand.—*Flexors of Wrist.*—The *f. carpi ulnaris* (ulnar nerve) and *f. carpi radialis* (median) flex the wrist. The ulnar tends to turn the supinated hand still more out, but they do not move the wrist joint laterally. Flexion indeed hinders the lateral movements of the wrist, in consequence of the shape of the articular surfaces. The f. c. ulnaris flexes the fifth metacarpal bone on the carpus, as well as the wrist joint. In paralysis, flexion of the wrist can only be effected by the flexors of the fingers when these are extended. Extension of the wrist is unsteady from the loss of the synergic contraction of the flexors.

Extensors of Wrist.—*E. carpi radialis brevis et longus: e. c. ulnaris* (musculo-spiral nerve and its radial branch). The short radial is a direct extensor; the long radial and the ulnar move the hand laterally as well. In paralysis of all three extensors, the wrist can only be extended by the extensors of the fingers when the phalanges are flexed. In loss of the short radial, direct extension is still possible by the long radial and ulnar, and lateral extension by one of these alone. If either is paralyzed, together with the short radial, direct extension is lost, and only lateral extension, in the direction of the remaining muscle, is possible. Paralysis of either lateral extensor, long radial or ulnar, leads to permanent deviation of the wrist in the direction of the remaining muscle (Fig. 10). The loss of the long radial is more serious than that of the ulnar, because the radial lateral movement is of more importance, being needed for the convenient movement of the hand to the mouth. Paralysis of the extensors impairs flexion of the fingers, from the great shortening of the course of the tendons, by the flexion of the wrist that occurs. The ulnar extensor acts also synergically with the extensor of the metacarpal bone of the thumb, as may be noted if the finger is placed on the tendon beneath the styloid process of the ulna. Hence, in paralysis of this extensor, the hand deviates laterally when the thumb is strongly extended.

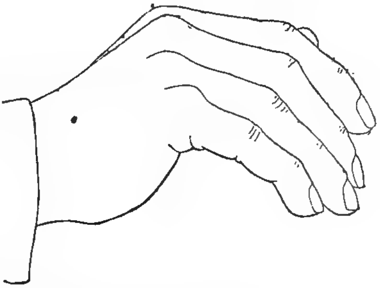
FIG. 10.



Paralysis of the long radial extensor of the wrist in a young child; habitual deviation of the hand toward the ulnar side. (*After Duchenne.*)

Extensors of the fingers; Extensor communis digitorum; E. indicis; E. minimi digiti (musculo-spiral nerve). The common extensor moves the fingers and then the wrist. When the muscle is faradized, the extension begins at the distal phalanges, and these become flexed again, when the hand is extended beyond the plane of the forearm, by the tonic force of the flexors, the course of their tendons being elongated by the extension of the wrist. But physiologically the muscle has little action on the last two phalanges, since they cannot be extended by the long extensor, if the interossei, their proper extensors, are paralyzed. During extension by the communis the fingers are separated from the second. The extensors of the first and last fingers have a similar extensor action, but, in addition, they adduct their respective fingers toward the middle finger. In paralysis the extension of the fingers is impossible, but if the proximal phalanges are passively extended, the middle and distal joints can be extended by the interossei. For the lateral movements of the digits, extension of the proximal phalanges is essential, and hence these movements are lost, but they can be performed if the proximal phalanges are passively extended. The posture of the fingers due to contraction of the palmar fascia resembles that in palsy of the long extensor (see Fig. 11), but an examination of the palm shows the cause of the flexion.

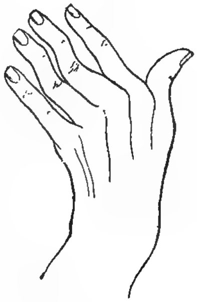
Fig. 11.



Posture of the hand in contraction of the palmar fascia, resembling that in paralysis of the long extensors of the finger. (*From a sketch by Mr. V. Horsley.*)

Flexors of fingers; F. sublimis (median nerve); *F. profundus* (median and ulnar nerves). These muscles flex chiefly the second and third phalanges, the first phalanx being flexed by the interossei. The superficial muscle flexes the second phalanx on the first, the deep flexes both. The action on the first phalanx is confined to extreme flexion of the fingers, and is the less the more the wrist is flexed. But if the flexion of the middle and distal phalanges is prevented, the first is strongly flexed. In extreme shortening of the course of the tendons by flexion of the wrist, the action on the fingers is very feeble, and hence there is a normal synergic action of the extensors of the wrist. When the extensor of the fingers is in strong action, extending the proximal phalanges, the action of the flexors on the second and third joints is very strong (tearing position). In paralysis of these muscles the power of flexing the last two joints is lost, but the interossei still flex the metacarpo-phalangeal joints. Paralysis of the deep flexor alone causes loss of the power of flexing the distal joint, but interferes with many movements, such as playing on the piano. In paralysis of these muscles, the unopposed tone of their opponents, the interossei, which extend these joints, lead in time to over-extension, and, with repeated passive pressure in using the fingers, may even produce a subluxation backward. In palsy of the sublimis this effect is chiefly seen at the middle joint (Fig. 12), in that of the profundus at the distal joint.

Fig. 12.



Paralysis of the fibres of the flexor sublimis which act on the two middle fingers: twelve years' duration. The second phalanges of these fingers are bent backward and subluxated from the contracture of the unopposed interossei, while the last phalanges are kept in position by the unaffected flexor profundus. (*After Duchenne.*)

Interossei and lumbricales (ulnar nerve, except the outer two lumbricales, which are supplied by the median). The interossei abduct and adduct the fingers, but only when these are extended at the metacarpo-phalangeal joints, and some effort is required for adduction, since the tendency of the long extensor is to separate the fingers, and this influence has to be overcome. They also extend the second and third phalanges on the first, and flex the first on the metacarpal bones. The lumbricales aid the flexor-extensor action of the interossei, but do not move the fingers laterally. The opposite action of the forearm muscles and of the interosseal extensors and flexors is very important. Their synergic action steadies movements, and in many actions they contract alternately. Thus in making a down-stroke with a pen or pencil, the long flexors bend the last two joints; while in making an up-stroke, these are extended, and the metacarpophalangeal joint is flexed, by the interossei. In paralysis of these muscles the lateral

FIG. 13.

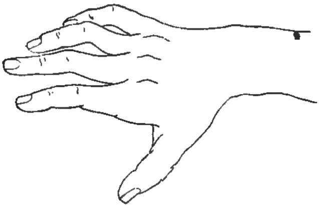


FIG. 14.

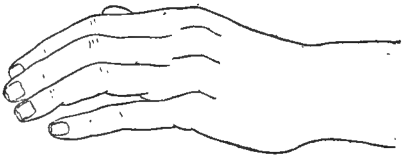


FIG. 13.—Recent incomplete paralysis of the interossei from a punctured wound of the ulnar nerve at the wrist: attempt to extend fingers. The loss of extension of the last two phalanges is chiefly marked in the last two fingers, from the influence of the lumbricales (supplied by the median) on the others. (*After Duchenne.*)

FIG. 14.—Paralysis of the interossei (ulnar nerve) slight in degree: attitude of fingers at rest.

FIG. 15.

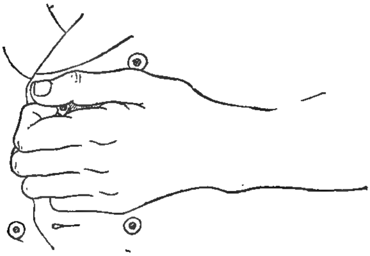


FIG. 16.

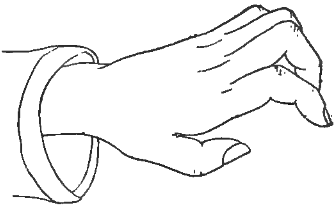


FIG. 15.—Attempt to unbutton waistcoat by the hand shown in the last figure; extreme flexion of the two last phalanges, and extension of the first, on the attempt to use the fingers. (*From nature.*)

FIG. 16.—Old-standing palsy of interossei and thenar muscles, showing the over-extension of the first and flexion of the last two phalanges.

FIG. 17.

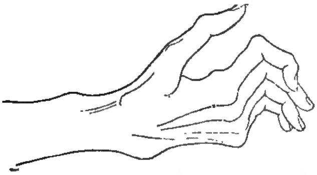


FIG. 18.

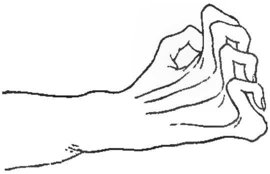


FIG. 17.—Paralysis of all the intrinsic muscles of the hand and of the long flexor of the thumb, in consequence of an injury to the brachial plexus in dislocation of the shoulder. The fingers present the claw-like attitude, the thumb is in extension. (*After Duchenne.*)

FIG. 18.—Paralysis of the ulnar nerve from a wound at the wrist (indicated in the figure). Extreme claw-like hand from the unopposed contraction of the common extensor and long flexors of the fingers and thumb. (*After Duchenne.*)

movements are lost, but a slight abduction and adduction of the index can still be effected by its long extensors. Only the first phalanx can be extended, and flexion is almost confined to the last two phalanges. The first three lumbricales, being supplied by the median nerve, often escape when the other muscles are paralyzed by an injury to the ulnar nerve, and these aid the others when they are merely weak; hence the index and middle fingers seem to recover before the others (Fig. 13). The position of the hand at rest becomes altered. Normally there is slight flexion at all joints by the tone of the muscles, interossei and long flexors. In paralysis the first phalanx is in a line with the metacarpal bones, while the other phalanges are flexed, the middle more than the distal (Fig. 14). In action this flexion is always increased, the metacarpo-phalangeal joints become over-extended, and the other joints strongly flexed (Fig. 15). Gradually the hand assumes this posture even at rest (Fig. 16), and ultimately the posture becomes warped into a deformity by the over-extension of the first phalanges, and extreme flexion of the others, due to the contracture of the long extensor and of the flexors; the tendons of these muscles stand out conspicuously on the back and in the palm, and a claw-like attitude is developed, the "main en griffe" (Figs. 17 and 18). Changes in the articulations may ultimately limit even passive movement.

Muscles of the Thumb.—*Extensor secundi internodii pollicis* (musculo-spiral nerve) extends both phalanges, and moves the whole thumb backward and from the fingers, so as to bring it behind the plane of the metacarpus. It may ultimately extend the wrist joint, but it never supinates. It is not used in extending the thumb when this is opposed to the first finger. In paralysis, the metacarpal bone of the thumb is slightly flexed on the carpus, and is inclined forward. The second phalanx is flexed on the first, and can only be extended (by the abductor and outer part of the short flexor) when the metacarpal bone is adducted, and the first phalanx is flexed. The constant flexion of the second phalanx interferes with the movement of the index finger, unless the patient remembers to move the thumb out of the way by the extensor of the metacarpal bone. Writing is not interfered with because the muscle is not concerned in extension with opposition.

The *Extensor primi internodii pollicis* (musculo-spiral nerve) is the true abductor of the thumb. It moves the metacarpal bone outward, and extends the first phalanx. It would move the whole hand in the same direction as the thumb, were not this tendency counteracted by the synergic contraction of the extensor carpi ulnaris (*q. v.*). It does not pronate or supinate. In paralysis, abduction of the metacarpal bone is less than normal. There is an undue flexion of the first phalanx, and the metacarpal bone is flexed on the carpus, so that the thumb is drawn toward the palm. The loss of this muscle is, however, compensated to a considerable extent by other muscles.

The *Extensor ossis metacarpi pollicis* (musculo-spiral nerve) is really the long abductor of the thumb. It moves the metacarpal bone outward and forward, flexing it on the carpus, and then flexes the wrist with slight pronation. It thus moves the thumb as much forward as outward. In *paralysis* the metacarpal bone is, at rest, less inclined forward than normal, and somewhat abducted, but the first phalanx is in its normal position. Movement of the thumb is but little interfered with.

In combined palsy of the extensors of the first phalanx and of the metacarpal bone (common in lead palsy), the thumb becomes adducted, and is parallel to the radius. The first phalanx is slightly flexed by the thenar muscles.

The *Thenar muscles* constitute two groups: (1) The short *abductor and outer portion of the short flexor* (median nerve) move the metacarpal bone forward and inward (flexing the first phalanx), incline it outward, and rotate it inward, so as to place its palmar aspect opposite the fingers. The second phalanx is ultimately extended. If the metacarpal bone is previously abducted the movement is greater and amounts to circumduction. (2) The *adductor and inner part of short flexor* (ulnar nerve) go to the inner side of the first phalanx. The metacarpal bone is moved toward that of the second finger; if previously flexed, it is extended; if previously opposed to the index, it is moved a little outward. The phalanges follow the movements of the metacarpal bone, but the first is slightly flexed and the second is extended, as the fingers are by the interossei.

The *Opponens pollicis* (median nerve) flexes the metacarpal bone on the carpus, and abducts it, but this movement is insufficient to oppose the thumb to the index; the conjoint action of the abductor is necessary (see above).

The *Flexor longus pollicis* (median nerve) flexes the second phalanx forcibly and the first feebly. It has no action on the metacarpal bone. It is used in writing (making a stroke toward the body) and in picking up a small object, etc. In paralysis this flexion is lost, and with it these actions; if an object is held between the tips of the thumb and forefinger, the last phalanx of the thumb is bent back. Other movements of the thumb are not interfered with.

If all the thenar muscles are paralyzed, the metacarpal bone is in the plane of the index, and drawn toward it by the extensor of the second phalanx, which moves the metacarpal bone inward and backward. The whole thumb corresponds with the metacarpal bone in position, the phalanges being normal. In paralysis of the short abductor and flexor, the second phalanx cannot be extended unless the metacarpal bone is abducted. These short muscles normally prevent the abduction that the extensor of the second phalanx tends to produce. The latter, moreover, prevents undue adduction when the special abductors are paralyzed, but brings the metacarpal bone into the plane of the index. Thus the posture of the hand at rest resembles that of the hand of an ape (Figs. 19 and 20). If the abductor and opponens are paralyzed, the tips of the thumb and fingers can only be brought together by flexing the last phalanges of the digits (Fig. 22). Then the thumb can be brought into contact with the finger by means of the short flexor, which inclines the metacarpal bone sufficiently to effect this, although not enough for the tip of the first finger to touch the thumb when its phalanges are extended.

FIG. 19.

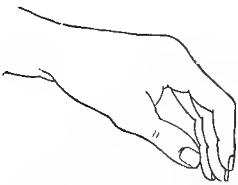


FIG. 20.

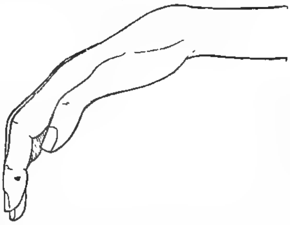


FIG. 21.

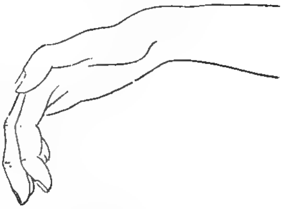
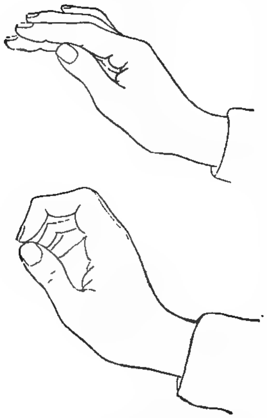


FIG. 19.—Normal position of the thumb (for comparison with the succeeding figures).

FIG. 20.—Position of the hand in long-standing paralysis and wasting of the thenar muscles. Under the influence of the long extensor the metacarpal bone of the thumb has been brought into the same position as the other metacarpal bones, being rotated slightly, so that the back of the thumb is in the plane of the back of the hand, like the hand of the ape. (*After Duchenne.*)

FIG. 21, from another case, shows the same condition, but still greater displacement of the metacarpal bone has taken place, from the greater contraction of the extensor. (*After Duchenne.*)

FIG. 22.



Paralysis of the abductor brevis and opponens pollicis. From the want of these muscles the thumb can only be brought in contact with the tip of the index by strong flexion of the last two phalanges of the fingers, otherwise the tip of the thumb only reaches the middle of the second phalanx. (*After Duchenne.*)

If the short flexor is paralyzed, the thumb can still be opposed to the first two fingers by the abductor, but it cannot be opposed to the two last fingers, on account of the deficient lateral inclination of the thumb, which should be produced by this muscle. Writing is easy by means of the short abductor, whereas, if this is lost, although the thumb can be opposed to each of the fingers, writing is much interfered with.

If all the thenar muscles are paralyzed, a certain amount of opposition of the thumb and fingers is still possible, by means of the flexion of the last phalanges of the thumb and fingers. If all are paralyzed except the adductor, objects can still be held between the thumb and side of the palm.

MUSCLES OF THE LOWER LIMBS.

Muscles Moving the Hip Joint.—The *Gluteus maximus* (small sciatic nerve chiefly) extends the hip joint, and feebly rotates the thigh outward. It is the most powerful extensor of the hip, and it is chiefly used when a forcible extension is required, and the joint has been previously flexed. It is employed, not in standing, or in walking on level ground, but in going upstairs, or uphill, and in rising from a seat. When it is paralyzed these movements are difficult.

The *Gluteus medius* (gluteal nerve) is the chief abductor. All parts of the muscle have this action, but, in addition, the anterior third moves the thigh forward and rotates it inward, while the posterior third moves it backward and rotates outward. The successive action of the several parts causes circumduction. The *Gluteus minimus* (gluteal nerve) has probably the same action. In paralysis, abduction and circumduction are lost; in standing on the other foot the pelvis is inclined from the affected side, and hence, in walking, there is an oscillation of the trunk, which becomes very considerable if the muscles of both sides are affected. Moreover, the unopposed tone of the outward rotators produces a permanent rotation of the leg, so that the toes are directed outward, and, from the altered position of the foot, the propulsion of the body in walking is deficient.

The *Pyriformis, Gemelli, Obturator internus*, and *Quadratus femoris* (special nerves from the sacral plexus), all rotate the thigh outward, and the first-named muscle, in addition, carries the thigh obliquely backward and outward, in the same way as the posterior fibres of the gluteus medius. In paralysis of these muscles, external rotation is impossible, and the unopposed tone of the internal rotators (anterior fibres of the gluteus medius and minimus) causes the leg and foot to be habitually turned inward.

The *Psoas* (lumbar nerves) and *Iliacus* (anterior crural nerve) flex the hip joint, and, in doing so, cause also a slight rotation outward. In paralysis flexion is lost, and the use of the leg in walking becomes impossible.

The *Tensor of the Fascia lata* has a slight power of flexing the hip, and at the same time rotates the thigh in. It normally counteracts the tendency of the ilio-psoas to rotate outward. If it is paralyzed, there is a tendency for the foot to turn out when it is being brought forward in the act of walking.

Adductors of the Thigh.—The *Pectineus* (obturator nerve) causes an oblique movement forward and inward, *i. e.*, a combined flexion and adduction, as in crossing the legs. It also rotates outward. The *Adductor longus*, and probably the *Adductor brevis* (obturator nerves), have the same action, but the flexion is less than by the pectineus. The *Adductor magnus* (obturator and great sciatic) causes a similar adduction, but while its upper fibres rotate outward, its lower fibres rotate in, and are employed in keeping the foot straight during adduction in riding. This is very difficult, if these fibres are paralyzed. The foot then turns out when the hip is flexed, either in the recumbent posture, or in walking, from the preponderance of rotation out by the other adductors. When all the adductors are paralyzed, not only is adduction lost, but in flexion of the hip the foot is moved forward and outward, instead of directly forward, showing that there is normally a synergic action of the abductors and adductors with the flexors in this movement.

Muscles Moving the Knee.—*Extensors of the Knee: Rectus* and *Vasti* (anterior crural nerve).—The vasti act solely on the knee joint; the rectus also aids in flexing the hip, but chiefly when the knee is bent. In consequence of its passage over the hip joint, moreover, the force with which it extends the knee is increased by the simultaneous extension of the hip. This effect is useful in the propulsion forward of the body in walking.

In paralysis of the extensors of the knee, standing is still possible if the knee is extended, since the arrangement of the articulation renders a contraction of the extensors unnecessary. But secondary shortening of the flexors is apt to occur, and then standing becomes impossible because the knee cannot be perfectly extended. In the same way, walking is possible if the leg is not moved forward beyond the vertical position; if it is, the knee becomes flexed by the weight of the leg and foot, and the patient falls when he attempts to rest upon it. Rising from the kneeling posture in the ordinary way is impossible. In partial paralysis of the muscles, as in pseudo-hypertrophic paralysis (*q. v.*), the extension of the knee, in rising, is facilitated by placing the hand upon it, and so bringing the centre of gravity of the body near the fulcrum of the lever formed by the femur. If the vastus internus and rectus are paralyzed, the vastus externus may dislocate the patella by the obliquity of its traction. The vastus internus never does so in the opposite condition because its action is less oblique.

Flexors of the Knee.—The *Sartorius* (ant. crural nerve) flexes the hip and knee joints, and has a feeble power of rotating the thigh outward and the knee inward. It is a muscle of small importance.

The *Gracilis* (obturator nerve) adducts the thigh more powerfully than it flexes the knee. It rotates the leg inward.

The *Semitendinosus*, *Biceps*, and *Semimembranosus* (great sciatic nerve) are not only flexors of the knee but extensors of the hip joint, and are the muscles that extend the hip during ordinary walking, the gluteus maximus (*q. v.*) being called into action only during special efforts. The leg is rotated inward by the semitendinosus, outward by the biceps.

In paralysis of the flexors the resulting loss of the power of flexion interferes with walking, since the knee joint cannot be bent, in the forward movement of the leg, until the thigh is flexed sufficiently to permit the weight of the foot to flex the knee. To prevent the toes striking the ground the foot is unduly flexed on the leg. The loss of the support that the flexor tendons give to the knee joint leads to an undue strain on the ligaments, which become stretched, and slight retroflexion of the joint may occur.

In paralysis of the muscles that extend the hip there is a tendency to fall forward in walking. To counteract this the trunk is carried backward, and a fatiguing strain on the flexors of the hip results.

In paralysis of the biceps, the leg, during flexion, is rotated inward; when the biceps remains and the other muscles are paralyzed, there is an undue rotation outward. The effect of these abnormal movements on the ligaments of the joint is such that, after a time, the amount of rotation becomes greater than is possible in health.

The *Popliteus* (internal popliteal nerve) has but a feeble power of flexing the knee. Its chief action is to rotate the leg inward when the knee joint has been flexed.

Muscles Moving the Foot.—*Extensors of foot on leg.** The *Gastrocnemius and Soleus* (internal popliteal nerve, from the sciatic) have the same action. They extend the hinder part of the foot and draw down the outer side of the fore part of the foot, but very little the inner side. Hence the foot is rotated, so that the dorsum looks outward, while the whole foot is turned inward on the axis of the leg. The peculiar inversion and adduction that thus accompanies extension is due to the form of the articular surfaces. The gastrocnemius has very little power of flexing the knee, but the extension of the knee increases the effect of the muscle on the ankle joint, especially in walking, just as we have seen that the extension of the hip augments the force with which the contracting rectus extends the knee. For direct extension of the ankle, the peroneus concurs and opposes the inversion. The only difference between the gastrocnemius and soleus is that the latter, having no attachment to the femur, can extend the ankle when the knee is flexed as well as when it is extended. In paralysis of these muscles extension of the ankle (by the peroneus longus and flexor longus digitorum) is extremely feeble, and the foot can scarcely be carried beyond a right angle. Walking is greatly interfered with;

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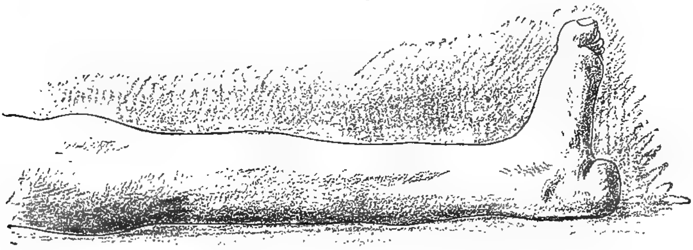
* These muscles have lately been termed "plantar flexors," because they are homologous with the flexors of the wrist. The term is a bad one, since it involves a use of the word "flexor" in absolute contradiction to its proper signification.

standing on tiptoe is impossible. The unopposed peroneus longus causes eversion of the foot, lowers the head of the first metatarsal bone, and deepens the plantar arch. In time the ankle joint becomes overflexed; the heel considerably lowered and the plantar muscles and fascia become shortened. The resulting deformity is termed talipes calcaneus (Fig. 23).

Peroneus longus (musculo-cutaneous nerve, from external popliteal of sciatic) everts the foot, lowering the inner border, narrowing the foot, and increasing the plantar arch. It also turns the whole foot out on the axis of the leg. It has a very feeble power of extending the ankle. It keeps down the inner part of the foot during extension by the calf muscles, as in walking. In paralysis of this muscle the inner part of the front foot is not supported during extension, and yields to slight force. The foot becomes adducted and rotated, so that the sole is directed inward, in consequence of the unopposed action of the sural muscles. The inability to press the inner part of the ball of the foot firmly against the ground leads to overaction of the flexors of the great toe. The plantar arch is lessened; there is "flat foot."

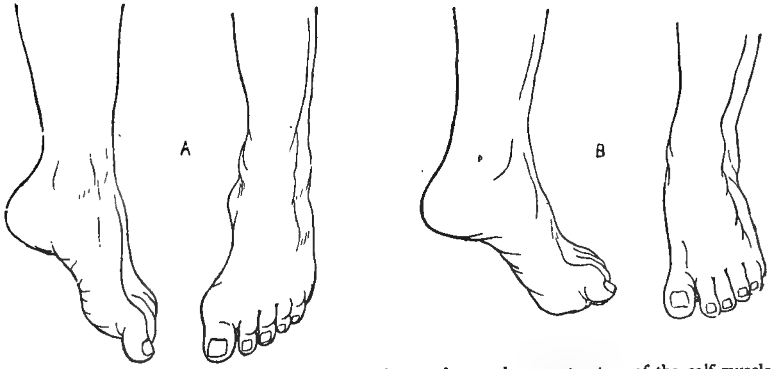
FIG. 23.



Talipes calcaneus from atrophic paralysis of the calf muscles, with flexion of the middle and distal phalanges of the toes from paralysis of the interossei.

Flexors of the Foot.—The *Tibialis anticus* (anterior tibial branch of ext. popliteal nerve) produces simultaneously three movements: it elevates the inner part of the front foot (opposing the peroneus longus); it flexes the ankle joint, and adducts the foot. The *Extensor longus digitorum* (ant. tibial nerve), besides extending the toes, flexes and abducts the foot. The abduction is in consequence of the outward position of its tendons beneath the annular ligament. These two muscles together produce direct flexion of the foot, or flexion with adduction or abduction, as the force of one or the other preponderates. Paralysis of either weakens flexion, and the corresponding lateral movement is

FIG. 24.



Talipes equinus, due to atrophy of the tibialis anticus and secondary contracture of the calf muscles. In A the foot is shown at rest; there is slight equino-varus. In B it is shown during flexion, and the varus is changed to valgus by the action of the peroneus longus. Note the increased extension of the toes in B from the compensatory over-action of the long extensors of the toes. (*After Duchenne.*)

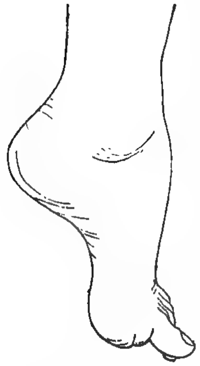
lost, flexion being accompanied by the deviation effected by the muscle that remains. The defect in flexion is greatest in paralysis of the tibialis, and the loss of the instinctive flexion, when the leg is brought forward in walking, causes the foot to catch against the ground. Paralysis of the flexors is followed by secondary contracture of the extensors, and talipes equinus results (Fig. 24), which is the greater the longer the palsy has lasted (Fig. 25). Its occurrence is facilitated, in many cases of palsy, by lessened growth of the bones of the leg, so that the ball of the foot only touches the ground when the foot is extended. There is usually slight rotation inward of the foot at rest, even when the tibialis is paralyzed (see Fig. 24, A), because such rotation is produced by the sural extensors (p. 56), but in this case the slight valgus at rest is changed to varus on an attempt to flex the ankle (Fig. 24, B).

The *Peroneus brevis* (musculo-cutaneous branch of ext. popliteal nerve) abducts the foot and rotates it, raising the outer edge.

The *Tibialis posticus* (posterior tibial nerve from int. pop.) adducts the foot and curves it, rendering the outer border and instep more convex. Its power of adduction is greater than that of the tibialis anticus, and it does not rotate the foot in the same manner.

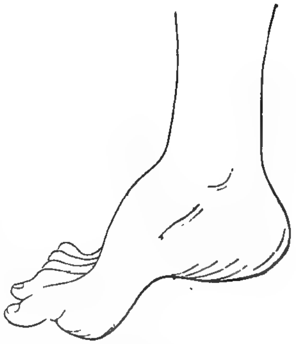
These two muscles alone have the power of adducting and abducting without flexing or extending; and in their paralysis these simple movements are lost. If one only is paralyzed, a deformity develops corresponding to the action of the other muscle,—talipes valgus in paralysis of the tibialis posticus; t. varus in that of the peroneus brevis.

FIG. 25.



Extreme talipes equinus from old-standing palsy of the tibialis anticus (infantile paralysis) and extreme contraction of the calf muscles. No flexor movement was possible.

FIG. 26.



Paralysis of the interossei and the adductor and short flexor of the great toe. The first phalanges are over-extended and the second are flexed, while the hollow of the sole is increased. (*After Duchenne.*)

The *muscles moving the toes* present, in their mode of action, a close correspondence to those of the fingers.

The *Extensor longus digitorum* and the *Extensor longus pollicis* (anterior tibial nerve) extend chiefly the first phalanges, while the *Flexor longus digitorum* and *Flexor brevis* (posterior tibial nerve) flex the last two phalanges. The *Lumbricales* and the *Interossei* (post. tibial nerve by ext. and int. plantar), together with the *Abductor* and *Flexor brevis minimi digiti*, oppose both the other extensor and flexor muscles, flexing the first phalanx and extending the others. This action is of great importance in walking, since they give the last propulsion to the body as the ball of the foot leaves the ground. The *Abductor, Adductor*, and *Flexor brevis pollicis* (plantar nerves from post. tibial), have a similar action on the great toe, but with adduction or abduction respectively. The interossei also produce a lateral movement of the toes, but this action is of little practical importance. In paralysis of the common extensor of the toes, and of the proper extensor of the great toe, the tonic force of the interossei and analogous muscles produces persistent flexion of the first phalanges and extension of the others. If the conditions are reversed, and the latter muscles are paralyzed, the first phalanges are over-extended, sometimes even subluxated, and the two other joints are flexed, so that a claw-like form of foot is the result (Figs. 26 and 23). The final propulsion in walking, above described, is much interfered with, and the attempt is painful because the ends of the toes are turned toward the ground.

PART II.

DISEASES OF THE NERVES.

GENERAL PATHOLOGY.

Structure.—The individual fibres consist of a central "axis cylinder," which is the chief functional element, surrounded by the "medullary sheath," or "white substance of Schwann," composed of myelin (M, Fig. 27). The latter is absent in the "non-medullated" fibres of the sympathetic. A delicate membrane surrounds the white substance, called the "primitive sheath," or the "sheath of Schwann" (S). Nuclei (n) lie at intervals within the sheath, between it and the myelin. The white substance is interrupted at regular distances by what are termed "nodes," sometimes, from their discoverer, "nodes of Ranvier" (N). The end of each portion, or "internode," is enclosed by the sheath, through which the axis cylinder passes. Between the incurved extremities of the sheath is a little clear cementing substance, shown by the fine dotting in the figure. There is one nucleus (n) to each internode, about its middle. Around the nucleus is a little protoplasm, and it is probable that a very thin layer of protoplasm everywhere lies between the sheath and the myelin. Each internode may be conceived as a cell, a fat cell, according to Ranvier, consisting of membrane, nucleus, protoplasm and fatty matter, the cells being arranged end to end, and the axis cylinder passing through them, as a string passes through a series of tubular heads. The internodes are shorter toward the termination of a nerve. It is important not to confound the nodes with other, imperfect, divisions, sometimes termed the "incisions of Schmidt" (IS, Fig. 27). These are oblique, incomplete divisions of the white substance. Many exist in each node. They are believed by some histologists to be of artificial origin, but their uniform character seems conclusive evidence that they depend on structural conditions.

FIG. 27.

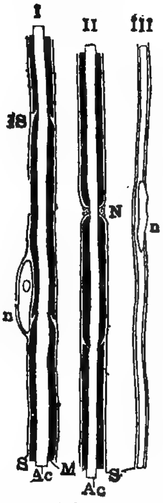


Diagram of the structure of nerve fibres. I and II, medullated, III, non-medullated fibre; S, sheath; M, myelin, or white substance of Schwann; Ac, axis cylinder; n, nucleus; N, node; IS, incision of Schmidt. (*From a preparation of the nerve fibre of a frog, stained with osmic acid.*)

The myelin, Ranvier suggests, must protect the axis cylinder, since its almost liquid consistence will diffuse pressure on the nerve. It may also have an insulating action, which, while not essential for conduction, may render conduction more perfect. The nodal segmentation keeps the almost liquid myelin uniformly distributed along the fibre, and permits nutrient material to reach the axis cylinder.

The "gray fibres," "non-medullated fibres," consist of an axis cylinder, sheath, and nuclei (Fig. 27, III), but contain no myelin. They constitute the sympathetic nerves, but some (probably sympathetic fibres) are found in all the spinal nerves. They are absent from the nerves of special sense, except the olfactory, which contains no other fibres.

The axis cylinder of most nerve fibres, probably of all, is continuous in one direction with the process of a nerve cell, and may be regarded as the prolonged process of the cell, sharing all the changes of nutrition that the cell undergoes. It has been supposed that there are other centres for the nutrition of nerve fibres than the cells from which they spring, but this theory, which rests on no direct evidence, is unneeded to explain the phenomena of disease.

The nerve fibres are united into "fasciculi" by delicate nucleated connective tissue, and these fasciculi are similarly connected into larger bundles, while the whole nerve is surrounded by a dense, connective-tissue sheath. All these tracts of connective tissue are continuous. In them the blood vessels run, and nerve fibres are distributed. These nervi nervorum are derived from the nerve they supply.*

Lesions of Nerves.—*Secondary Degeneration.*—As we have seen, a nerve fibre undergoes the structural changes known as "degeneration" whenever it is separated from the cell with which it is connected. As a rule, the degeneration is in the direction of conduction, *i. e.*, the cell from which the nerve fibre conducts is that which governs its nutrition. An exception is presented by the sensory fibres in the peripheral nerves, which conduct upward, but degenerate downward when separated from the ganglia on their roots. The degeneration is commonly termed "secondary" because it is dependent on a primary lesion of another kind—as division of the nerve. Degeneration also follows many slighter lesions of a nerve, compression, over-extension, and the like, but it is not certain that this is always the same as that which follows a total lesion. The secondary degeneration is often called "Wallerian," from the name of the pathologist who first studied it. It is of great importance, practical and theoretical. The medullary sheath breaks up into segments, and these into smaller and smaller fragments, and the minute globules and granules are ultimately removed from the nerve sheath. This is then empty, the axis cylinder having also perished during the process. The nature of this process of destruction has been studied by Erb and others, but has been chiefly elucidated by the researches of Ranvier, and must be considered in some detail. Ranvier has shown that it is not a mere process of death or decay, but an active process, a destruction of the nerve as such by the protoplasm and nuclei of the internodal cells that constitute it.

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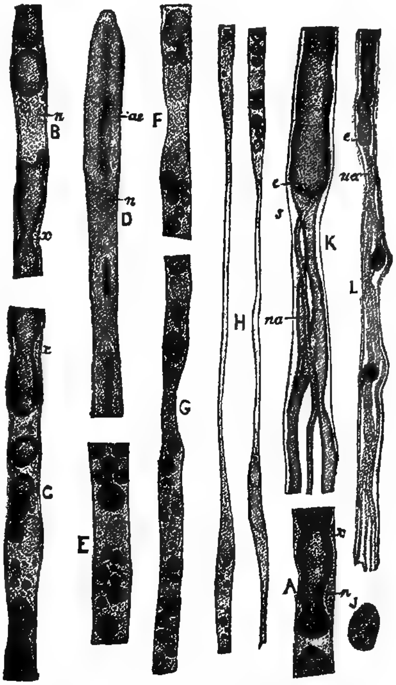
* Horsley, *Royal Med. and Chir. Soc.*, January 22d, 1885.

The nature of the process has been chiefly studied in animals. The most important facts are illustrated in Fig. 28, in which the examples have been selected from Ranvier's figures and reduced to one tint. In the rabbit the first changes are to be perceived at the end of twenty-four hours. The nuclei are increased in size (Figs. A *n*, B *n*), the amount of protoplasm about them is greater than normal and it is granular, and there is in places a local increase in the amount of protoplasm within the sheath, compressing the myelin (A *x*, B *x*, C *x*). The nuclei then become detached from the sheath; the protoplasm everywhere increases, and encroaches on the myelin, until here and there it meets across the tube, completely separating the myelin (A, lower part; B, upper part) and with the myelin the axis cylinder (D). This process then goes on with increased rapidity; the myelin is broken up into smaller and smaller fragments (C, E, F) which are globular in the watery protoplasm, just as oil forms globules in water. The nuclei meanwhile continue to enlarge, and then divide, first the nucleolus and then the whole nucleus (F). The two nuclei may again divide, until (as in G) there are four or more nuclei in each internode, instead of one only, as in health. The small globules of fatty myelin seem to become changed in chemical composition, since they are stained less deeply by osmic acid. Ranvier suggests that their fatty matter may undergo a process of saponification. Ultimately they seem to pass through the sheath, are taken up by connective-tissue cells and lymphatic cells in the vicinity (as in J), and are gradually, for the most part, removed. By the time the myelin is in small globules the nuclei cease to multiply. On the removal of the products of degeneration the sheath shrinks, and looks empty in places, but

FIG. 28.



A–J. Degeneration of nerve fibres (osmic acid and carmine staining). A, from sciatic of rabbit four days after section; B, C, same, fifty hours after section; D, a fibre stained with carmine only, to show the axis cylinder; F, G, fibres from pigeon three days after section; H, two fibres from pneumogastric of rabbit six days after section; J, a lymphatic cell from interfibrillar connective tissue, containing globules of myelin that it has taken up; *n*, *n*, nuclei; *x*, *x*, constrictions of the myelin produced by the growth of the protoplasm; *ac*, axis cylinder. K, L, regeneration of nerve fibres; K, from pneumogastric of rabbit, seventy-two days after section; L, from sciatic of rabbit ninety days after section; *e*, rounded end of white substance of central end of nerve; *s*, sheath; *na*, new axis cylinder; in L are two globules of myelin remaining from the degeneration of the old fibre.

here and there it is enlarged by the nuclei, protoplasm, and a few remaining myelin globules (H). Hence in transverse section many small sheaths are seen with a few of larger size where they have been cut across at these swellings.

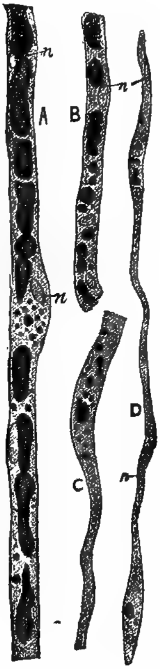
In the rabbit the first changes are visible at the end of twenty-four hours; the first complete interruption of the myelin and axis cylinder occurs about the end of the second day; the process of destruction is considerably advanced at the end of the fourth day, and is finished, and the multiplication of the nuclei ceases, toward the end of the second week. The changes begin simultaneously in the whole extent of the separated portion of the nerve. They progress more rapidly at its peripheral extremity than elsewhere. At the end of the second day, in the rabbit, stimulation of the nerve by electricity no longer causes muscular contraction. Thus the disappearance of the electrical excitability coincides in time with the first complete segmentation of the myelin and axis cylinder. This interruption explains what is termed the "loss of excitability;" it does not follow that the fragments of axis cylinder cannot be stimulated; their stimulation can have no effect. When they cease to be *excitable*, we cannot tell. But it is not certain that "loss of excitability" is only the result of loss of continuity. (See p. 63.)

Thus the process is the result of an active growth of the nuclei and protoplasm of the nerve, *i. e.*, of the cellular elements of which the nerve is composed. Why should this occur? The determining cause is the interruption of the axis cylinder—its separation from the cell of which it is a process. It follows equally destruction of the nerve cell. Ranvier points out that the destructive exuberance of the protoplasm, which follows loss of function in the axis cylinder, suggests that normally this function restrains the vital energy of the cell elements. But it is possible that changes in the nutrition of the axis cylinder precede the segmentation.

A process of destructive cell activity suggests the idea of inflammation. The degeneration may be regarded as a process of parenchymatous inflammation. Several observers have described other indications of inflammation outside the fibres, especially increase of nuclei, and accumulation of leucocytes in the interstitial connective tissue, and even in the nerve sheath, and also dilatation of the blood vessels. Such changes are intense at the primary lesion. Their degree in the nerve below the lesion is very variable, and seems to be influenced, in some degree, by the irritative nature of the primary disease.

The process of secondary degeneration occurs more slowly in the rabbit than in the bird, and seems to be still slower in man, in whom it is probable that complete segmentation does not occur until between the fourth and eighth days. It is certain that an identical process does occur in man. This is placed beyond doubt by the researches of Pitres and Vaillard on the changes in peripheral nerves in the neighborhood of bedsores, etc. These are shown in Fig. 29, a comparison of which with Fig. 28 will show the identity of the process. It is highly probable that after complete division of a nerve in man the changes are the same as in animals. But the most common lesion in man is neuritis, and in this the process of degeneration is probably greatly modified by the severity of the primary lesion; in some cases it occurs rapidly, in others very slowly. In the latter cases, which are chiefly those of focal neuritis, the process cannot be the same as that which follows division of a nerve. The change in irritability, which will be more fully described in the account of the symptoms, is a slow depression, sometimes moderate in degree. The depression may be preceded by an increase in irritability. In such cases, there can be no complete segmentation of the nerve fibres. There must be a gradual alteration in the molecular nutrition of the axis cylinder, changing its excitability. Even in severe cases there is not usually a sudden loss of irritability; the current necessary for stimulation has to be made stronger and stronger, until at last the strongest current the patient can bear fails to cause muscular contraction. But we cannot infer from this that there is ever an actual interruption of the axis cylinder. A stronger current might still excite the nerve. Moreover, as we shall presently see, when a nerve is being regenerated, an axis cylinder may be continuous, and may even conduct, and still not be excitable.

FIG. 29.



Degenerating fibres from cutaneous nerves of man. (*After Pitres and Vaillard.*) A, from near a bedsore in a case of fracture of the skull; B, C, D, from the fifth nerve in a case of neuralgia and ulceration of the lip; *n*, *n*, nuclei; in A the protoplasm and nuclei are increased, myelin is being broken up, the processes having proceeded furthest in the neighborhood of the nucleus in the middle of the fibre; in B the segmentation has gone on to the formation of globules, which in C are, for the most part, small, and many have been removed, so that the fibre is narrow; while in D all the products of degeneration have been removed from considerable tracts of the sheath.

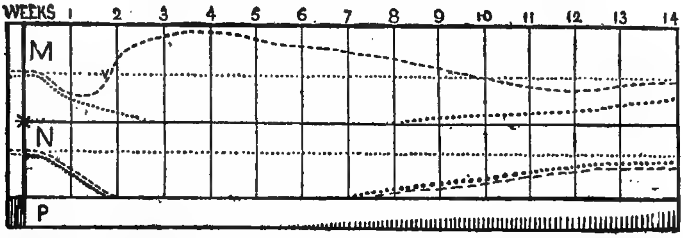
All severe changes in the nutrition of the fibres involve the intra-muscular nerve endings in the same degree. The evidence of this is that the faradaic irritability of the muscles (which depends on the nerves within them) presents changes quite similar to that of the nerve trunk. But this is not always true in slight changes of nutrition of the nerve. We shall presently see that the slight alterations of irritability in the nerve and muscle do not always correspond. The nerves terminate in structures of special nature, and these may well have some slight degree of nutritional independence.

Regeneration may occur in the nerve after the degeneration is over. It is a slow process, occupying the second, third and fourth month after division. According to Ranvier, it occurs always by the growth of new axis cylinders from the central end of the nerve (see Fig. 28, K and L) which ultimately become covered with myelin. One or more new fibres may spring from each central fibre, and these may subdivide. All are enclosed in a sheath which is continuous with that of the central end (Fig. 28, K, *s*.). It is to be presumed that only some of these axis cylinders persist and achieve functional permanence. Sometimes these fibres twist about, and even turn back and grow upward, probably in the direction of least resistance. In animals, new fibres may grow through a considerable extent of cicatricial tissue between the divided ends of a nerve, but in man it is doubtful whether regeneration of a divided nerve occurs unless the extremities are brought in contact, or at least close proximity. Some investigators believe that there is a formation of fibres in the peripheral extremity, as well as a growth of new fibres from the central end. It is highly probable that the process, in the peripheral part of a nerve, is influenced by the connection with other nerves and the recurrent course of anastomosing fibres; because, in man, the suture of a nerve divided some time before, in the peripheral part of its course, has been followed by very rapid restoration of function. In cases of injury which does not involve an actual interruption of the nerve fibres, regeneration occurs more readily, and very readily in slight lesions, in which it is probable, as we have just seen, that degeneration has been incomplete, and the continuity of the axis cylinder has not been broken. The regenerated nerve fibres regain some conducting power while they are still much narrower than normal, and before they can be excited by electricity.

Muscles.—The degeneration of the motor nerves is attended by changes in the nutrition of the muscles. These commence in or after the second week. The muscular fibres become narrower, and may be reduced, ultimately, to one-third of their former width. The transverse striation becomes less distinct, and the striæ seem to be nearer together than in health. The fibres may become slightly granular, but do not, as a rule, present actual fatty degeneration. If no regeneration of the nerve takes place, the transverse striation gradually disappears, and may be replaced by a longitudinal striation, or the fibres may undergo certain chemical changes, in consequence of which they present a peculiar glassy appearance, which has been called "vitreous degeneration." During the progress of the changes in the fibres, the nuclei of the sarcolemma and of the interstitial tissue are increased in number, and cellular elements, either newly formed, or migrated from the vessels, develop into fibres, so that ultimately the muscular fibres are separated by considerable tracts of connective tissue, and a state of cirrhosis results. If regeneration of the nerve occurs, the muscular changes are arrested, and the normal condition of the fibres is slowly restored. The amount of connective tissue in the muscle is often permanently increased, but the size of the fibres is reduced out of proportion to the increase in the connective tissue, so that the muscle is for a long time smaller than normal, and its natural bulk may never be regained. If no regeneration of the nerve occurs, the increased fibrous tissue takes the place of the lost muscular tissue, and slowly contracts, so that permanent shortening may result. Similar shortening sometimes occurs when there is partial recovery of the nerve and muscle. In most lesions of nerves, other than actual division, some fibres recover, even though others are permanently destroyed. The muscular degeneration is apparently the result of that of the motor nerves. All the phenomena can be thus accounted for. (See p. 45.)

Symptoms of Nerve Injury and Degeneration.—The symptoms that attend the lesions of motor nerves and the consequent degeneration are of great importance. The lesion of the nerve causes paralysis of the muscles supplied by it, due to, and in proportion to, the interference with the conducting power of the nerve. The muscles at once become flabby from loss of tone, and to this atony actual wasting is added in the course of a few weeks. The wasting is due to the reduction in size of the muscular fibres. If the sensory nerve fibres are not interrupted, the muscles become tender to the touch, and pain is caused by their strong contraction, due probably to the interstitial inflammation, and the morbid sensitiveness of the sensory nerves that end in the interstitial tissue.

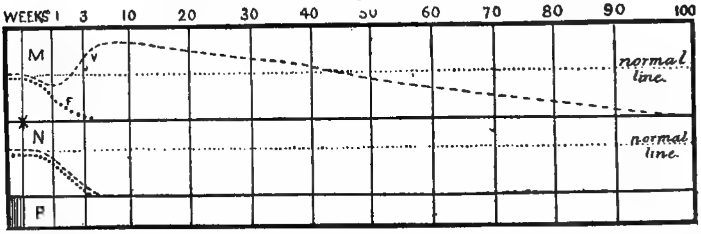
FIG. 30.



Type of degenerative reaction in a case of nerve injury of moderate severity. (*After Erb.*) Muscle irritability lowered from middle of first week; faradaic, extinct in middle of third, reappearing in ninth; voltaic, increased from middle of second week until tenth, then depressed until fourteenth. Nerve irritability lowered from middle of first week, lost at end of second, reappearing at seventh. Power lost until end of fifth week. In this and the following figures the normal degree of irritability (ascertained from the healthy side) is represented by the finely-dotted horizontal line (*n.l.*); faradaic irritability, F, by a line of larger dots; voltaic irritability, V, by a broken line; M, muscle; N, nerve; P, power of voluntary contraction; the asterisk marks the occurrence of the lesion. The vertical lines represent time intervals.

The most important symptoms are those that are afforded by electrical examination of the muscles and nerves, since they enable the degenerative changes to be ascertained and followed during life. The alterations in the electrical reaction, consequent on this degeneration, have been already briefly mentioned (p. 43), but must now be described in greater detail. The rapid degeneration of a nerve, which follows a severe lesion, is attended by a loss of irritability on electrical stimulation, the loss being the same to faradaism and voltaism. After such lesions as are common in man, neuritis, for instance, there is no sudden loss, such as occurs after injury of a nerve in an animal, when the nerve becomes segmented, but there is a more or less rapid diminution of excitability, and this goes on until no stimulation can be produced, even by a strong current. The progressive changes in irritability may be conveniently represented on a chart. Fig. 30 shows the typical course of the changes of irritability in a case of moderate severity. In the muscle (M) a fall of irritability (due to the degeneration in the nerve endings) occurs simultaneously with that in the nerve trunk (N), and the faradaic excitability becomes extinct at the same time in both nerve and muscle. The fall in voltaic irritability is quickly arrested by the change in the muscular fibres through which they soon become more excitable than normal to the voltaic current. This change usually occurs during the second week, and the irritability continues to increase during the third and fourth weeks. At its maximum it may amount to three, four or five cells of the battery, *i. e.*, contraction can be obtained in the paralyzed muscles with a current weaker, by so many cells, than is necessary to cause contraction in a corresponding unaffected part. The further course of the changes in irritability depends on the severity of the lesion and the intensity of the degeneration. In a case of moderate degree, such as is shown on the chart, nerve irritability reappears about the end of the second month, usually after some voluntary power is regained. It is at first low, so that a strong current is required. It gradually increases, but for a long time continues a little below the normal degree. This return of nerve irritability is accompanied by a corresponding return of faradaic irritability in the muscles (*i. e.*, in the intra-muscular nerves). The increase of voltaic irritability often persists long after recovery of power, but it lessens as faradaic irritability returns, and, as shown in the chart, it may fall below the standard of health before it ultimately regains the normal degree.

FIG. 31.



Type of reaction in a case of complete and permanent damage to a nerve. (*After Erb.*) Early course as in the last diagram; no return of power, nerve irritability, or faradaic irritability in the muscle. The early increase in voltaic irritability gradually lessens and at the end of ten months falls below the normal, but is not extinct until nearly two years.

Slight changes in irritability can be ascertained only by a comparison with the corresponding part on the other side in the same individual. Moreover, when we speak of excitability being "lost," we mean that we can obtain no stimulation by any strength of current that can be borne. This strength varies in different persons and in different parts. The more sensitive the part, the earlier irritability seems to be lost, and the later it seems to return. In many cases, if we could use very strong currents, the irritability would be found to be merely much lowered, although it seems to be extinct when we can only test it with currents of moderate strength.

If the lesion is very severe, so that there is no recovery, and no regeneration of the nerve, the loss of nerve irritability, and of the faradaic muscular irritability, is permanent. The increase in voltaic irritability persists for months, and then gradually falls, as the muscular fibres waste, and becomes lower and lower (see Fig. 31), until ultimately no reaction can be obtained, the fibres having perished. It does not become extinct until at least a year has elapsed, and sometimes (as in the chart) only toward the end of the second year. Often, when no contraction can be produced on the first attempt, after two or three separate applications of voltaism, distinct contractions are obtained.

The changes we have hitherto considered are in the *degree* of irritability, "quantitative" changes. But the quantitative increase in the muscular irritability is accompanied by a change in the order of response, according to the pole that is applied and the strength of current that is used—a "qualitative" change. We have already considered its general characters (p. 44), and have seen that it consists in an undue readiness of response at the positive pole (Anode) compared with that at the negative pole (Kathode) (Fig. 32, B and C), the muscle being normally the more sensitive to the latter (Fig. 32, A). Writing Cl. for the closure of the circuit, O. for its opening, and C. for contraction, the normal reaction is:—

1. K.Cl.C.; 2. $\left\{\begin{array}{l}\text{An.Cl.C.;}\\ \text{An.O.C.;}\end{array}\right.$ 3. K.O.C.; in disease we have

1. $\left\{\begin{array}{l}\text{K.Cl.C.;}\\ \text{An.Cl C.;}\end{array}\right.$ 2. An.O.C.; 3. K.O.C.; or even .

1. An.Cl.C.; 2. K.Cl.C.; 3. K.O.C.; 4. An.O.C.

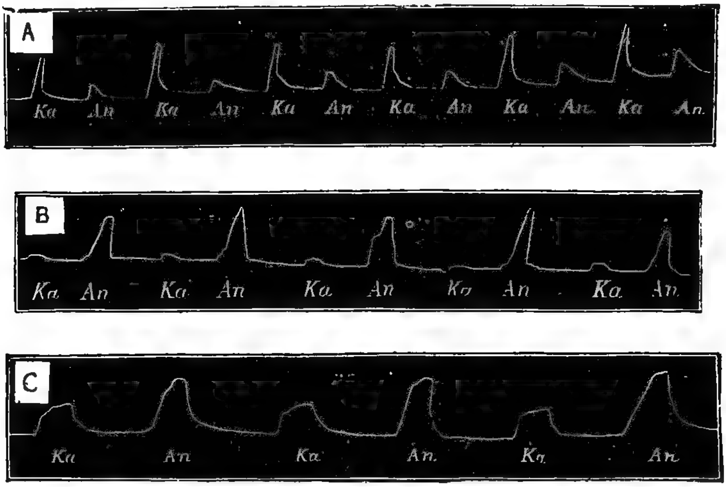
Sometimes a slight abnormal tetanic contraction during the passage of the current also occurs.

This qualitative change is not always present, at any rate in recognizable degree. Even when there is a marked quantitative change, the kathodal closure contraction may still occur first. When the change is present, it is only in the muscles, and it must depend on the muscular fibres themselves. In the motor nerve the kathodal response is always the first, although a qualitative change has been detected in degenerated sensory nerves.

The muscular contractions which occur thus with undue readiness differ from normal contractions, excited through the nerves, in their distinctly deliberate character. Instead of the quick, lightning-like contraction, the movement is distinctly longer in reaching its maximum and longer in its duration. This difference is very well shown in the accompanying tracings of contraction, after Erb (Fig. 32). During the period of increased voltaic irritability, the mechanical excitability of the fibres is often increased. If they are directly percussed, they respond with a distinct slow contraction (Erb).

Such are the changes in irritability which occur in cases of nerve lesion and degeneration of moderate and considerable degrees of severity. Certain variations are occasionally met with, and these are especially frequent cases of neuritis of slight degree. In severe cases the fall in nerve irritability, which usually commences in the middle of the first week, may not take place until the end of this week, although it may then progress so rapidly that no stimulation can be obtained at the end of the second week. An example of this is shown in Fig. 33.* The same chart, and some of those that follow it, illustrate another very common variation from the type above described.

FIG. 32.



Tracings of the muscular contractions in nerve degeneration. (*After Erb.*) *Ka*, Kathodal closure contraction; *An*, Anodal closure contraction. A, tracing in health; *Ka*, much stronger than *An;* contractions sudden. B, tracing in nerve degeneration with moderate current. *An* much greater than *Ka*, the latter scarcely visible. Contraction slower, shown by the more sloping upstroke. C, the same, with a stronger current. *Ka* greater, but still less than *An;* the slow character of the contraction and its long duration well marked.

FIG. 33.

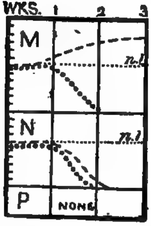


FIG. 34.

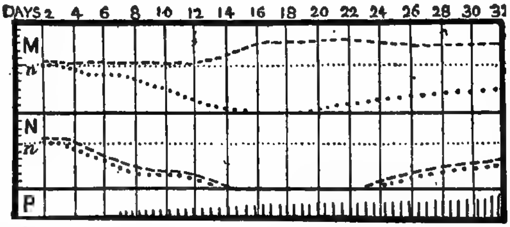


FIG. 33.—Severe neuritis, paralysis complete for months; recovery imperfect. Irritability of nerve, normal during first week; fell rapidly during the second; lost at its close. Simultaneous increase in voltaic irritability. The divisions on the left side represent cells of the voltaic battery, and half-centimetres of the secondary coil of Stöhrer's induction apparatus, in this and the following charts.

FIG. 34.—Slight neuritis; slight return of power on seventh day, slowly increasing; normal in fifth week. Muscle: faradaic irritability lessening from fourth day, lost on fifteenth, reappearing on twentieth. Voltaic irritability normal till twelfth day, then augmented. Nerve trunk: lowered from fourth day without initial increase, reappearing on twenty-third day; changed alike to faradaism and voltaism.

There may be no fall in the voltaic excitability of the muscles before the commencement of the degenerative increase. Indeed, this initial fall is as often absent as present.* The change in the nutrition of the muscle may then coincide with, instead of succeeding, the degeneration of the nerve endings (Figs. 33, 35). Sometimes, if the degeneration is rapid, there is an interval of a few days before the secondary changes in the muscle have reached the degree necessary to produce increased irritability. In a slight degree of degeneration, the increase in the voltaic irritability of the muscles may be postponed for a week or more after the nerve irritability begins to fall, and until the rise occurs, the voltaic irritability of the muscles may remain normal, even when their faradaic irritability falls with that of the nerve trunk. An instance of this is shown in Fig. 34.

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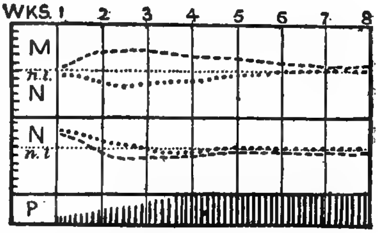
* This and the following charts are from cases of neuritis of the facial nerve.

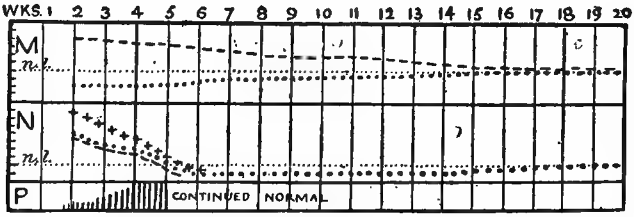
When the nerve lesion is very slight in degree the fall in nerve irritability may be preceded by a rise, which may be far greater in degree than the subsequent fall. The fall may indeed be not only slight, it may be altogether absent, so that the rise constitutes the only symptom. This initial increase in irritability is of considerable interest as the manifestation of the slightest degree of alteration in the nutrition of nerve fibres.† It may last for a few days or for two weeks (Fig. 35), and I have once known it to continue for five weeks (Fig. 36). Although the change in irritability of the nerve is usually the same to both currents, a

Fig. 35.



Slight neuritis; power not entirely lost, and became normal during the third week. Slight deg. react. in muscle developed during second week, and continuing till seventh. Nerve irritability increased during second week, passing during the third into transient depression, the voltaic irritability falling more than the faradaic.

Fig. 36.



Slight neuritis; quick recovery of power during third and fourth weeks. Degen. react. in muscles present at the end of the second week, slowly lessening, but present in slight degree until the fourth month. Considerable increase in irritability in the nerve trunk at the end of the second week, greatest to the faradaic shock (crosses) and lasting until the fifth week.

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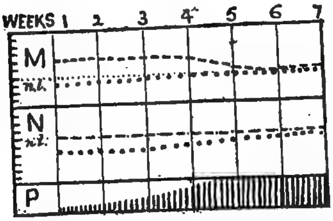
* The fall *seems* to suggest that the voltaic irritability of the muscular fibres is normally below that of the nerve endings, but the fact that there is often no fall seems incompatible with this explanation. It may be that the effect of a very rapid degeneration of the nerve is first to lower the irritability of the muscular fibres, perhaps by an irritative inhibition, and it is only when this has passed away, by the greater degeneration of the nerve endings, that the muscular excitability can be manifested. When the change in the nerve is slight, such inhibitory influence may be absent.

† A similar change is met with in some central diseases, as chorea and paralysis agitans.

partial exception to this rule is presented by the increase in irritability we are now considering. The change is not always quite equal to faradaism and to voltaism, and it is frequently much more marked to the isolated faradaic shocks, than it is to either the voltaic current or the rapid succession of shocks that constitutes the faradaic current. This is shown very strikingly in Fig. 36, and in less degree in Fig. 38. In the former the irritability to the two currents was the same; in the latter that to voltaism was distinctly the greater. I have once met with a slight but distinct and prolonged diminution in faradaic irritability when no change could be found to voltaism (see Fig. 37). Bernhardt has observed lessened irritability to faradaism with distinct increase to voltaism in an ulnar nerve the seat of traumatic paralysis. In one singular case of double facial palsy, with weakness of the limbs, and changes in their muscular irritability, the voltaic excitability of the facial nerves was not lessened, although faradaic irritability was lost to such a current as could be borne. The case resembled in characters diphtheritic paralysis, although there was no history of diphtheria. It was probably either a form of multiple parenchymatous neuritis, or changed nutrition of the ganglion cells. The symptoms gradually passed away.*

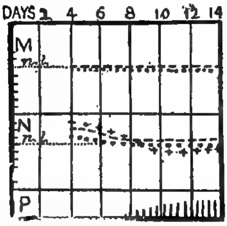
Fig. 37.



Slight neuritis; power regained during the second week. Deg react. in muscle distinct on eighth day, lessening during second and third weeks. Nerve: lowered faradaic, normal voltaic irritability.

Moreover, the law that the faradaic irritability is changed in the same degree in the muscles (nerve endings) and in the nerve trunk, is also not always true of slight cases. The muscle nerves may present no change when the excitability of the nerve trunk is considerably increased (Fig. 38). The probable significance of this difference has been already mentioned (p. 63).

Fig. 38.



Very slight neuritis. No change in muscular irritability; irritability of nerve trunk increased from fourth to eighth day, most to faradaic shock (crosses), least to faradaic current.

The electrical excitability of a nerve and its power of conduction are usually lost together. But when a nerve is recovering from a local lesion, and regeneration is in progress below the lesion, some power of conducting the voluntary stimulus may return in the peripheral portion before it becomes excitable by any form of electricity. Another rare anomalous condition has been noted by Bernhardt and Grünberg, in recent cases of nerve injury. When the lesion arrests all conduction of the voluntary stimulus, a strong faradaic stimulation of the nerve above the lesion may still cause slight contraction in the paralyzed muscles. It is probable that this difference is simply one of strength of stimulus, and that the nerve impulse generated by the electricity is stronger than that descending from the brain.

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* The observations were made with great care and with full knowledge of a frequent source of fallacy—the diffusion of the voltaic current to the muscles themselves. Cyon has observed a similar change after injury to a nerve in an animal.

Recovery of the nerve is attended by gradual restoration of power over the muscles, the nutrition of which slowly improves; but if the wasting has been considerable, a long time elapses before they regain their normal size, and, as already stated, they may be always somewhat smaller than the corresponding muscles on the unaffected side. Curious secondary spasmodic symptoms are common after palsy of the facial nerve (*q. v.*)—spontaneous muscular contractions, and associated over-action of the affected muscles. The associated over-action is very rarely met with after palsy of the limbs, but coarse, fibrillary, muscular contractions are not uncommon, and may persist for years after recovery. Their probable nature is discussed in the account of the pathology of facial paralysis.

Sensory symptoms also result from lesions of the nerves that contain sensory fibres. The interruption of the fibres arrests conduction, and causes loss of sensation in the part supplied by the nerve, just as it causes motor palsy. But a slight lesion of the nerve may cause persistent muscular paralysis, and sensation may be unaffected, or impaired only in slight degree, and for a short time. This result is so frequent that we cannot ascribe it to a difference in the relative damage to the motor and sensory fibres. The probable explanation is that a slight degree of conduction may suffice for the stimulation of the sensory centres in the brain, and not for that of the muscular fibres. In another class of cases, however, we must seek a different explanation of the persistence of sensibility in the area supplied by the nerve. There may be no less even when a nerve is completely divided. This is observed chiefly near the extremity of a limb. It can only be explained by what is termed "recurrent sensibility." Anastomoses exist between the terminal fibres of different nerve trunks, and it would seem that, in some individuals, the connection is so abundant as to suffice for conduction. Thus, of two persons in whom the ulnar nerve at the wrist has been divided, one may have total anæsthesia in the fingers supplied by the nerve, and in the other there may be little or no loss.

The peripheral segments of the sensory fibres degenerate, like the motor nerves. We cannot test them, and ascertain the degeneration in the same way, in severe lesions, because their stimulation cannot be perceived. Nevertheless, an altered polar reaction has been observed in slight cases, analogous to that which occurs in the muscles. A sensation is produced with the positive pole by a weaker current than is required with the negative pole. In very rare cases of nerve injury a delay in conduction of pain has been observed.

Increased sensitiveness in the area of distribution of the nerve, with or without spontaneous pain, is very common in cases of partial lesions of nerves. It must be referred to the morbid changes in their fibres, alterations in the nutrition of the axis cylinder, probably analogous to the slight changes in the motor nerves. Hyperæsthesia, increased sensory excitability, is analogous to the increased motor excitability that we have seen is sometimes present. Pain and tenderness of the nerve trunk are also frequent in the same cases, due no doubt to the increased sensitiveness of the "nervi nervorum" distributed in the sheath. The pains of both kinds are sometimes very severe and persistent.

Reflex Action.—All lesions of nerves abolish reflex action in the area of the distribution of the nerve affected. In the muscles supplied, myotatic irritability is also lost, and this loss may persist long after the other symptoms have passed away.

Vasomotor and trophic disturbance occurs frequently in consequence of lesions of nerves. The vasomotor nerves run in the mixed nerve trunks, and suffer with the other fibres. The general character of the disturbance has been already described. When the lesion is acute and irritative, vascular dilatation and an increase of temperature occur at first, and are followed by passive hyperæmia and lowered temperature. There may be œdema, and an increased secretion of sweat. The changes in the skin, acute and chronic, are those that have been already described (p. 41). They may occur when there is no conspicuous evidence of vasomotor paralysis. The atrophy and smoothness of the skin is especially common. Occasionally, instead of becoming thin, it increases in thickness, the nails often become thick and rough and present transverse furrows. The growth of the hair may be lessened or increased. Sloughing of the skin is far less common than in central diseases, but vesication occurs with extreme readiness. Mustard plasters also blister the skin more readily than in health. It is important to remember this, because the affection is one in which hot applications are often recommended. A gentleman dislocated his shoulder, and either by the displaced bone, or in the reduction, the brachial plexus was seriously injured. The muscles of the hand wasted, and the skin became glossy. He was advised to bathe the hand daily in hot water. One day his wife bathed it in water which to her was pleasantly warm, but the result was that the hand was covered with blisters, and ulcers were left which did not heal for months. The joints often suffer in their nutrition. Acute inflammation, such as occurs sometimes in spinal affections, is rare, but chronic changes in the joints are frequent. Adhesions and alterations in the articular surfaces often limit the movement of the joints. When the muscular wasting is slight, the condition may resemble a primary joint affection, and thus a mistake in diagnosis is sometimes made.

INFLAMMATION OF NERVES: NEURITIS.

Neuritis, or inflammation of nerves, presents various characters, which have led to the distinction of numerous forms. The inflammation usually affects chiefly the outer sheath of the nerve, and this has been rather needlessly distinguished as "perineuritis." It may involve chiefly the connective tissue between the bundles of nerve fibres ("interstitial neuritis"); or the nerve fibres themselves ("parenchymatous or degenerative neuritis").

Our knowledge of the latter is recent, but as an acute and chronic process it has been found to be much more frequent than was suspected a few years ago. Some forms of neuritis have a tendency to ascend the nerve, and this variety has been termed "ascending neuritis" or "neuritis migrans." Usually only one nerve trunk is primarily affected, but sometimes many nerves suffer at the same time. This condition, "primary multiple neuritis," is of great importance, and will be described in a separate section.

Most forms of neuritis may be either acute or chronic. Acute neuritis usually subsides into a chronic stage, and its symptoms may thus persist for a long time. Other varieties that have been distinguished depend upon the cause to which the inflammation is secondary. Thus we may have cancerous, syphilitic and other forms, and these distinctions have some justification in the fact that there are some pathological differences in these forms of secondary neuritis.

Causes.—Any injury to a nerve causes more or less traumatic inflammation at the seat of injury. Such inflammation occurs readily, and follows damage that is slight as well as that which is severe. Neuritis may thus be set up by all sorts of incised and punctured wounds, by contusions, and by over-extension of the nerves. Those that pass by joints are liable to injury from dislocations, either by the displacement of the bones, or from pressure during reduction. The brachial plexus often suffers from this cause. In fractures the nerves may be injured directly, or may be compressed by the callus that is formed. External pressure on the nerve may also produce inflammation ; the musculo-spiral and the sciatic often suffer from this cause. The musculo-spiral and the nerve to the serratus are sometimes damaged by a violent contraction of the muscles through which they pass.

Neuritis may be set up by extension from adjacent inflammation. The nerves near suppurating joints may be involved, and even those that pass by a joint that is the seat of rheumatic inflammation. The intercostal nerves have been affected by extension from an inflamed pleura. The cranial nerves and the spinal nerve roots are involved by extension with especial frequency. Some of the most important symptoms of meningitis are due to the invasion of the nerves.

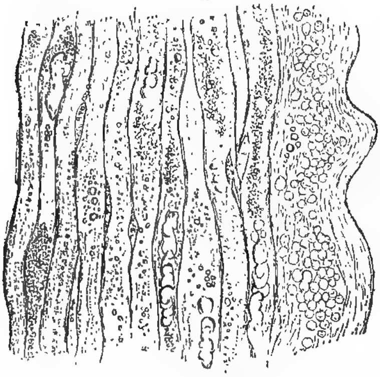
Exposure to cold is another cause of neuritis. It is then often called "rheumatic." The nerve, or rather its sheath, may be primarily affected, or the first effect may be inflammation of the fasciæ, from which the inflammation may spread to the nerve sheath. Persons who are liable to muscular rheumatism, and those who are gouty, suffer thus more than those who are prone to acute articular rheumatism.

Neuritis occurs also from certain general diseases. The chief are gout—from which it may develop without exposure to cold—syphilis, and cancer. In the latter it occurs only in the neighborhood of a cancerous growth, but the inflammation may be apparently simple. It occurs rarely in leucocythæmia, from an infiltration of the nerve with leucocytes. Acute diseases may also lead to neuritis. Smallpox, typhoid fever and others, may cause simple neuritis. Diphtheria causes a special "parenchymatous" form.

Acute degeneration of the nerves has been found in the neighborhood of bedsores in hemiplegia (Pitres). Such pressure neuritis may be the mechanism by which bedsores are produced. Neuritis may result from lead poisoning, although it is not certain that the characteristic paralysis which lead produces is due to neuritis. Lastly, multiple neuritis has been lately proved to occur from many causes, of which the chief is alcoholism, and to form part of many diseases in which it was previously unsuspected.

Pathological Anatomy.—In acute inflammation the affected part of the nerve is red and swollen. The redness depends on distended vessels, which may be visible on the surface. The swelling is due to œdema, or to a sero-fibrinous exudation, sometimes jelly-like in aspect. The microscope shows leucocyte-like corpuscles, surrounding the vessels, infiltrating the sheath (Fig. 39) and accumulating between it and the nerve. There may be even small extravasations of blood. These changes may be limited to the sheath, in what is called "perineuritis," or may extend into the substance of the nerve in "interstitial neuritis." In the latter case the lymphoid corpuscles infiltrate the septa, and may even be seen in the substance of the fasciculi between the nerve fibres. These changes may be continuous along a considerable tract of the nerve (diffuse neuritis), but more frequently they are chiefly marked at certain places, which are separated by portions of the nerve which are so little affected as to appear to the naked eye to be normal (focal or disseminated neuritis). The foci of inflammation are chiefly situated at places where the nerve turns round a bone, or emerges from canals or fasciæ, or divides.

FIG. 39.



Neuritis: degeneration of nerve fibres, the myelin broken up into masses, globules and granules. Accumulation of leucocytes in nerve sheath. From a case of multiple neuritis. (*After Leyden.*)

The extent to which the nerve fibres suffer varies much. They usually present little change, when the inflammation is limited to the sheath, unless the nerve lies in a bony canal, or in rigid fibrous tissue, within which the sheath cannot expand; its swelling then exerts pressure on the fibres. When the inflammation is interstitial the fibres suffer more readily, although not invariably. On the other hand, they are sometimes found much altered, when the connective-tissue elements are but little affected. In this case the inflammation is probably "parenchymatous" and begins in the nerve fibres, the interstitial tissue being secondarily involved. The changes in the fibres are almost the same as those that occur in degeneration (Figs. 28 and 29). It was pointed out how closely the two processes of inflammation and degeneration are connected, and how difficult it is to separate, or even to distinguish, them in many cases. The myelin of the white substance first breaks up into segments, more or less elongated, often with smaller globules of myelin between or beside them. The masses are cloudy or finely granular in aspect. The axis cylinders are interrupted where the myelin is divided. The nuclei of the sheath are increased in number, and the protoplasm around them is increased in quantity. Next the myelin is divided into smaller globules and granules, and the axis cylinder is no longer recognizable. The myelin then disappears in parts of the tubes, while it remains at other parts, but is still more finely divided. Lastly, the sheaths become empty, and very narrow, containing only nuclei at intervals, with here and there a little finely granular material, or sometimes some brownish pigment granules. The process may thus go on to complete destruction of the fibres. These commonly suffer unequally; fibres that have a normal appearance are scattered among those that are much altered.

In interstitial neuritis the axis cylinders often suffer less than the white substance. The latter becomes atrophied, so that the fibres are smaller than normal. If the interstitial inflammation is very intense the fibres may break up as above described. As the inflammation subsides, the new cellular elements that have made their appearance assume the aspect of fusiform cells, and fibrous tissue is developed, either from these cells, which become less numerous, or from the intercellular exudation. This fibrous tissue surrounds and encloses the fasciculi, a condition that has been termed "sclerosis of the nerve." A firm fusiform swelling may remain at the affected spot, and this may be adherent to adjacent structures. Sometimes fat is ultimately formed in the new connective tissue, a condition that has been rather unnecessarily termed "lipomatous neuritis" (Leyden); the fatty deposit does not seem to be related to any special form of neuritis.

In syphilitic neuritis, which affects chiefly the cranial nerves, there is a cellular growth in the sheath and interstitial tissue similar to that which constitutes other syphilitic new formations, with a variable amount of leucocytal infiltration of simpler inflammatory character. The growth in the sheath may amount to a distinct syphilitic tumor, with or without interstitial changes. In cancer, nerves adjacent to the new growth may present simple interstitial neuritis, or may be infiltrated by a growth of cancer elements spreading to the nerve by direct extension.

The destructive changes in the nerve fibres extend down the nerve to the periphery in a process of secondary degeneration, more or less inflammatory in nature, and accompanied by more or less interstitial inflammation. The changes that result are those that have been already described as occurring in secondary degeneration. Usually the central portion of the nerve remains free, the alterations ceasing a short distance above the seat of the primary inflammation. In rare cases an ascending neuritis (n. migrans) passes up the nerve, and may spread, at a plexus, to other nerve bundles and other nerve trunks. The formation of fibrous tissue in the inflamed part, and the cicatricial contraction of this tissue, often prolongs the irritation of the nerve fibres, and the symptoms due to this, and a state of "chronic neuritis" is said to be left.

Symptoms.—The symptoms of neuritis vary extremely according to its intensity, its extent, and the nerve that is affected. The onset of the acute form is sometimes attended by some constitutional disturbance, especially when many nerves are affected (see Multiple Neuritis). The chief symptoms are local. The most prominent is pain, felt in the inflamed part of the nerve, and also often in the part to which it is distributed. The local pain is due to the irritation of the "nervi nervorum," the distant pain is produced by irritation of the proper fibres of the nerve. Sometimes the pain involves the whole limb, and in severe cases it may be most intense, burning, boring, rarely darting, in character; usually worse at night; increased by movement, by postures that involve tension or pressure on the nerve, and by whatever causes passive congestion of the limb, such as the act of coughing. Sometimes it seems to radiate into distant parts, and not rarely pain is also felt in the corresponding region of the opposite limb. The sensitiveness of the whole of the affected region is increased, and even the bone may be tender, so that at first attention may not be directed to the nerve, but when this is pressed great pain is always produced. In slighter cases the pain is usually limited to the nerve and its distribution. If the nerve is accessible to direct examination, it may be felt to be distinctly swollen at the affected part. Occasionally the skin over it has been observed to be red, and rarely there has been slight œdema. Spontaneous sensations may be felt in the region supplied by the nerve, tingling, etc., and the skin may be hyperæsthetic. After a time, as the nerve fibres suffer, sensation may be perverted, or even lessened; complete anæsthesia is usually confined to small areas. The muscles supplied by the nerve become weak, tender, and present fibrillar twitchings, while they may contract occasionally in very painful cramp. Increased perspiration has been observed in the part of the skin supplied by the inflamed nerve, and sometimes, although rarely, eruptions occur. Herpes is not common from ordinary acute neuritis; a curious fact, considering that one form of neuritis seems to be its constant cause. Effusion into joints has been observed in very rare cases. The constitutional disturbance, which may attend the onset, subsides in the course of a few days, but the pain and other symptoms usually persist in undiminished severity for several weeks. They ultimately slowly subside into a chronic stage.

When the affection is chronic from the commencement, initial constitutional disturbance is absent. Pain is the prominent symptom from the first. It may preponderate either in the nerve at the seat of the inflammation, or in the region of its distribution. The affection of sensibility, and the trophic changes, are similar to those met with in the acute form. In both forms the muscles may waste, and present the reaction of degeneration when they are tested with electricity (p. 66). In slight cases the initial increase of excitability in the nerve is often well marked. Trophic changes in the skin are very common, especially the chronic impairment of nutrition that results in the glossy condition. Slight alteration in the nutrition of the joints is also common, and adhesions form, limiting the movement, and fixing the parts in the position corresponding to the muscular paralysis.

When neuritis ascends a nerve, the symptoms gradually extend in area, and if it reaches the plexus from which the nerve proceeds, they may extend to all the nerves of the limb. This "migrating neuritis" is not rare in man, and is readily produced in animals. It may reach the spinal cord, and there produce various disturbances. The inflammation may spread in the tissue outside the dura mater, or may extend to the cord, and cause subacute or chronic myelitis with or without meningitis. The paralysis that occurs secondarily to some visceral diseases, as those of the bladder, and commonly regarded as reflex paralysis, is probably produced through the agency of an ascending neuritis. Lastly, the inflammation may extend to the nerves of the other side, usually to those that correspond to the primary seat of the disease. Such extension may be through the spinal cord or the membranes, but in some cases symptoms occur in the nerve of the opposite side, without any indication of an affection of the centres. Experiments on animals have demonstrated that such implication of the opposite nerve may occur when the centres are unaffected. It has been called "sympathetic neuritis." We have seen that reflected pains are occasionally felt in the corresponding nerve on the opposite side, and it is probable that, as in neuralgia, vasomotor disturbance in the nerve sheath may accompany such pains, and, in a predisposed person, may cause actual neuritis.

The *duration and course* of neuritis vary much. A slight acute neuritis may run its course in a few weeks and then subside. Much more commonly the affection persists in a chronic stage for many weeks, or even months, and slowly passes off. The "rheumatic" forms are, as a rule, much more tedious than those that result from injury, although traumatic neuritis is sometimes very insidious and may last a long time; it may ascend the nerve from its starting-place, and become localized in certain situations, where it may develop afresh, and give rise to symptoms that may seem to be independent of their cause.

DIAGNOSIS.—The diagnosis of neuritis depends, first, on the localization of the symptoms to the distribution of a certain nerve trunk, and, secondly, on the pain and tenderness in the nerve. The diffuse pains that attend the onset may be readily mistaken for the pains of acute rheumatism, or for those due to an inflammation of the bone, but in the course of a day or two the localization of the symptoms declares their nature. The chronic form is easily mistaken for neuralgia, and the diagnosis is often difficult, the more so in that many so-called neuralgias are really due to neuritis. The distinction can only be fully discussed when we have considered the symptoms of neuralgia, but it rests chiefly on the fact that in neuralgia the pain intermits more completely than in neuritis, there is not the same initial tenderness in the nerve trunks, and the tender spots have a more uniform localization. Lessened sensibility, showing organic damage to the nerve fibres, is conclusive evidence of neuritis. The pains in some central diseases, chiefly those of the spinal cord, may be thought to be due to neuritis, but there is not the local tenderness of the nerve trunk, and the pain is not limited to the distribution of a single nerve. As we shall see presently, the diagnosis of multiple neuritis from affections of the cord is far more difficult.

PROGNOSIS.—The gravest form of simple neuritis is that in which the nerve is affected secondarily to a local suppurative inflammation. The prognosis is best, as a rule, in traumatic neuritis, but the rule is one to which the exceptions are occasionally very considerable in degree. The rheumatic form is usually less grave than the traumatic. In all cases the intensity of the symptoms, and the evidence of descending degeneration, furnish a more trustworthy guide to prognosis than does the mere form of the affection. The effects of complete degeneration always endure for some months. Regeneration does not occur until the original cause has ceased to act, and then occupies many weeks in its progress to the restoration of functional competence. In all forms pain is apt to linger on, and in the last third of life it may continue for years.

TREATMENT.—The first consideration in the treatment of neuritis is the removal of its cause, if this can be discovered. A wound or injury or local inflammation adjacent to the nerve must be dealt with by appropriate measures. Any constitutional cause, such as gout, must be treated. For the inflammation of the nerve, it is of the first importance to secure to the part as perfect rest as possible. Movement causes mechanical irritation of the nerve, and involves functional stimulation of its fibres, both of which are injurious. The nerves of a limb run between muscles, and the contraction of these muscles causes pressure on the inflamed nerve, and irritates it, as the pain thus produced sufficiently shows. Pain is indeed a useful indication of the harmful influence of movement, and all movement that causes pain should be avoided. The posture of the limb should be such as to involve no pressure on or tension of the nerve. The general treatment of an acute neuritis must be that suitable for any acute local inflammation, whatever its seat,—an unstimulating diet, an aperient, and diuretics. In gouty cases a brisk purgative may, with advantage, be given. General diaphoresis is useful in cases that are due to cold, and in others local sweating is often of distinct service. The limb may be steamed, or exposed to hot air. This should be followed by hot fomentations applied along the course of the affected nerve, and these by linseed-meal poultices. Leeches may also be employed at the onset of severe cases. When the inflammation results from injury, cold may be applied along the course of the nerve instead of heat; both probably modify the vascular disturbance of inflammation in a similar manner. Counter-irritation may be used at the onset of slight cases, but when the inflammation is severe, this agent is more effective during the subsidence of the inflammation than during its active stage. Blisters, repeated mustard plasters, or stimulating liniments may be used, but care must be taken not to blister skin that is anæsthetic, or troublesome ulceration may be caused. The same caution is necessary in regard to hot applications, as is shown by the case mentioned on p. 72.

Spontaneous pain requires sedatives, of which the hypodermic injection of morphia is by far the most effective. It should be used only for spontaneous pain, and not to enable the patient to use the limb in a way that would produce pain if morphia were not given. Mechanical irritation may be equally injurious, although the pain which it would cause is obviated by the sedative. An injection of cocain, $\frac{1}{8}$–$\frac{2}{3}$ gr., also often relieves the pain. In the rheumatic form salicylate of soda has been given, but it is of doubtful value. Iodide of potassium sometimes seems to be useful, but no agent has so distinct an influence on the process of inflammation in the nerve as small doses of mercury. A grain of blue pill may be given once or twice a day, and if morphia has to be injected at the same time, the mercury is useful also in correcting the constipating influence of the morphia.

In the chronic stage or form, counter-irritation, by blisters or cautery, is of great value. So also is electricity, which has little influence during the acute stage. The voltaic current should alone be used. The positive electrode may be placed over the inflamed part of the nerve, or over the seat of pain, and kept there for ten minutes at a time, the strength of current being slight, such as the patient can just perceive. In very chronic cases a stronger current, sufficient to cause actual pain, is of service, applied in a similar manner, but for a shorter time. It has probably chiefly a counter-irritant influence. Faradaism may be used in the same way. All painful impressions on the skin lessen, for a time, the nerve pain.

The muscles supplied by the inflamed nerve are best left alone, unless their wasting is marked, or the degenerative reaction shows serious damage to the motor fibres. In cases of moderate severity they will recover when the nerve recovers. All that is desirable is that they should be gently rubbed once or twice a day. If the wasting is considerable, however, or there is degenerative reaction, they should be stimulated to gentle contraction by a weak, interrupted voltaic current. They should on no account be faradized during the active stage of the affection, even if they act to faradaism. The acute pain that the faradaic stimulus causes, and the increased tenderness that lasts for hours afterward, are sufficient evidence of its injurious effect.

In all cases attention to the general health is of great importance. Tonics are needed during the chronic stage. Change of air will sometimes remove, in a few weeks, symptoms that have previously been stationary for months.

The modifications of treatment that are rendered necessary by the position of the neuritis will be considered when we come to speak of the affections of the several nerves. The pathological varieties of neuritis only need special treatment in so far as they depend upon special causes, and this point, as already mentioned, must always be one of the first considerations.

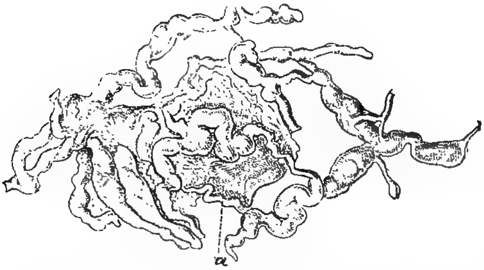
A description of "multiple neuritis," which should properly be considered next, will be better understood after the symptoms of the disease of special nerves have been described.

MORBID GROWTHS IN NERVES: NEUROMA.

The term "neuroma" has been applied indiscriminately to all morbid growths situated on the peripheral nerves. The discovery, by Virchow, that many of these consist of a growth of nerve fibres, while others consist of heterologous tissue such as constitutes morbid growths elsewhere, has led to the distinction of the former as "true neuromata" and the latter as "false neuromata," or "pseudo-neuromata," and even to the limitation of the term "neuroma" to a growth of nerve tissue, whether in the central or peripheral nervous system, the heterologous growths being called by the names they bear in other situations, as "fibroma," "sarcoma," etc. The latter system of nomenclature, although certainly more consistent, has not become current in this country.

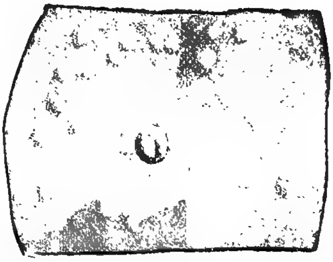
The true neuromata may consist of medullated or of non-medullated nerve fibres, termed "myelinic" and "amyelinic" forms by Virchow. The latter were for a long time regarded as fibrous. The occurrence of ganglion cells has been proved in only one or two instances. There is connective tissue between the nerve fibres, which varies in amount and in character, and hence the firmness of these tumors also varies. This interstitial tissue may be so abundant as to constitute an intermediate form between the true and false varieties. This is probably the condition in most cases of multiple neuroma. The "false neuromata" may be of various nature, but fibrous growths, fibromata, are far more common than any other kind. Myxoma occasionally occurs, the new mucoid tissue growing from the nerve sheath. Glioma is very rare on the peripheral nerves, although occasionally found on the auditory nerve. Various forms of sarcoma have been met with. Carcinoma also occurs, very rarely as an isolated growth, but not uncommonly as a more or less diffused or nodular infiltration of the nerve, arising by extension from a contiguous growth. Syphilitic growths are common on the cranial nerves within the skull, but are rare elsewhere. In lepra anæsthetica the nerves are infiltrated with fibrous tissue, enlarging them to many times their normal size. The enlargement is rarely nodular,

FIG. 40.



Plexiform neuroma from the orbit (*after Marchand*). The connective tissue surrounding the cords has been removed except at *a*, and irregular, cylindrical, nodular cords are seen anastomosing. A nerve passes into the tumor and suddenly becomes enlarged.

FIG. 41.



Cutaneous neuromata from skin of abdomen. (*After Smith.*)

and is perhaps rather a chronic cirrhotic inflammation than a growth. (See Multiple Neuritis.)

A curious variety of neuroma, consisting of interlacing cords, more or less nodular and tortuous, is termed *plexiform neuroma.* About twenty cases have been recorded. The disease commonly begins in fœtal life, and is most common on some branch of the fifth nerve in the orbit or the upper eyelid, but has been met with over the temporal bone, in the lumbar, cervical, brachial and solar plexuses, on the penis and the mamma. It may be quite superficial or deeply seated. The cords, from 1 mm. to several centimetres in diameter, consist of a clear outer zone of concentric fibrillar connective tissue, of inner looser nucleated tissue, and, in the middle, a bundle of nerve fibres, some normal, others degenerating. The cords are connected together by loose tissue, sometimes myxomatous, separated from the proper tissue of the cords by an epitheliated space. The growth of this form is extremely slow, but it may exert compression on adjacent structures.* A remarkable case of extensive myxomatous disease of the nerves of the forearm, congenital in origin, presenting analogies to plexiform neuroma, is recorded by M. De Morgan.†

FIG. 42.

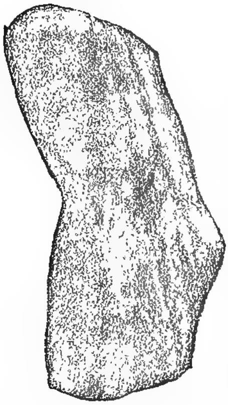


FIG. 43.

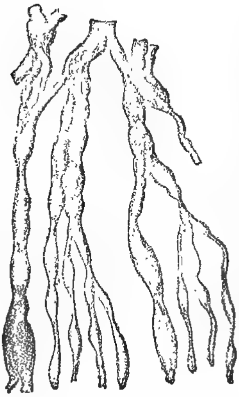


FIG. 44.

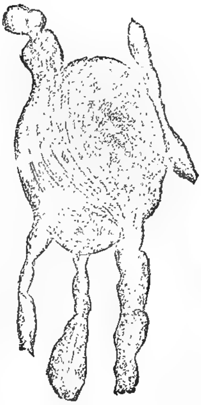


FIG. 45.

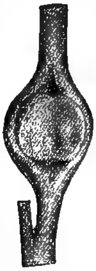


FIG. 42 —Right elbow of a man, æt. 30, with multiple neuromata; those on the nerves of the arm are visible beneath the skin as moniliform series of swellings along the course of the nerves. (*After Smith.*)

FIG. 43.—Part of the brachial plexus and nerves of the arm.

FIG. 44.—Neuroma of the posterior tibial nerve, same case. The tumor was the size of a lemon, and could be felt in the popliteal space. The flattened bundles of fibres of the nerve were separated and passed over the surface of the tumor. The portion of nerve to the right is a piece of the external popliteal. The tumors caused no interference with function. The patient died of enteric fever. One large tumor filled the pelvis.

FIG. 45.—Small neuroma of popliteal nerve laid open. It is seen to lie within the sheath. It caused no symptoms during life. (*After Smith.*)

The subcutaneous extremities of sensory nerves are sometimes enlarged into minute tumors which, when painful, have been termed "tubercula dolorosa." Neuromata in the skin may co-exist with similar tumors on the nerve trunks. Those shown in Fig. 41 are from the same case as the tumors represented in Figs. 42–44.

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* Marchand, *Virchow's Archiv*, Bd. 76, p. 36.

† *Path. Trans.*, vol. xxvi, p. 2.

The size attained by growths on nerves varies from that of a child's head to a nodule only just visible. They rarely exceed the dimensions of the closed fist. The variation in size of multiple neuromata is illustrated in Figs. 42–44, after Smith.* The large tumor (Fig. 42) was the size of a lemon, but in the same case a growth from the sciatic plexus almost filled the pelvis. They may occur on any nerve in the body, whether cerebro-spinal or sympathetic. False neuromata are generally single. When true neuromata are "multiple" they are often extremely numerous, and almost every nerve in the body may be transformed into a chain of growths. In the case figured more than 200 were counted in the right arm alone, and the total number of growths in the body cannot have been less than 1000.

The tumors are almost always within the sheath of the nerve (Fig. 45). Sometimes they are on one side, and the nerve may pass by unchanged. More often the substance of the nerve is involved, and the fibres may be separated and spread out on the surface (Fig. 44). Even then they may not be damaged. They suffer far more in heterologous growths than in true neuromata.

Causes.—The causes of neuroma are generally obscure. Multiple neuromata are sometimes hereditary and are probably due, in most cases, to a congenital tendency of tissue growth. They are said to be sometimes the result of general neurotic predisposition, and evidence of this is found in the occasional occurrence of neuromata in the subjects of cretinism or idiocy, of which some remarkable examples have been recorded. Multiple neuromata are said to be almost confined to men. The isolated form is not uncommon in women. They may occur at any age. Virchow believes that they are unusually common in the phthisical and scrofulous.

Of traceable immediate causes the most frequent is traumatic injury. Pressure, punctured wounds, or division of the nerves may give rise to them, apparently by a perversion in the process by which cicatrization takes place. A well-known instance is the formation of the so-called "amputation neuromata," or "bulbous nerves," round or oval growths, the size of a bean or larger, which form on the divided extremities of the nerves in the stump left after an amputation. It has been supposed that neuromata may arise from a local neuritis, but this is doubtful.

The occurrence of new growths containing nerve fibres may seem to be very remarkable. We must remember, however, how frequently they are connected with traumatic causes, *i. e.*, take origin in cicatricial processes. In such processes the nerve fibres present a remarkable power of growth. Ranvier has shown that from the end of each old fibre several new fibres grow, only one of which probably ultimately persists. Under apparently mechanical influences some of these fibres may twist about and even turn and grow upward. It is thus not difficult to understand that a true neuroma may result from injury to a nerve; it is even surprising that this is not a more frequent result.

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* R. W. Smith, "Treatise on Neuroma," 1849.

Almost any one of the cranial or spinal nerves may be the seat of a neuroma. They have been occasionally met with on the sympathetic.

SYMPTOMS.—These may be entirely absent, especially in the case of multiple (true) neuromata, which are sometimes discovered post-mortem, when their existence has not been suspected. The most frequent symptom is pain referred to the distribution of the nerve, often acute, burning, or darting, and frequently intermittent. It may be increased by pressure on the tumor. In the case of the terminal neuromata (tubercula dolorosa, bulbous nerves) pain may be the only sensory symptom. When the nerve fibres suffer, paræsthesiæ, numbness or formication, may be complained of, and ultimately sensation may be lessened or lost. Paralysis of the muscles supplied by the nerve is occasionally met with, the region paralyzed corresponding to the distribution of the nerve on which the tumor is situated, except in the case of neuromata in the cauda equina, where the nerves may suffer from pressure, and paraplegia may result. In all cases the distribution of the symptoms depends necessarily on the nerve affected. A neuroma of the pneumogastric may cause grave cardiac disturbance.

More common motor symptoms are reflex spasms in adjacent or even distant muscles. For instance, in a case of "bulbous nerves" in the stump after amputation at the shoulder joint, the muscles on that side of the neck were in constant clonic spasm. Occasionally epileptiform convulsions are produced, and have been known to cease when a painful neuroma had been excised. The occurrence of these symptoms has little relation to the size of the tumor.

When the neuromata are in accessible situations they may be felt. Sometimes they give rise to visible tumors, and in multiple neuroma the course of the superficial nerves may be marked out by lines of bead-like swellings, as in Fig. 42. The cutaneous neuromata also cause visible swellings. The whole abdomen was covered with such nodules in the case recorded by Smith (Fig. 41). They are commonly not sensitive, although preëxisting pain may be increased by pressure. Pressure on the nerve above the tumor commonly lessens the pain.

Plexiform neuroma rarely causes other symptoms than the signs manifesting its local presence. The functions of the nerve fibres involved are not usually interfered with. Once or twice anæsthesia of the skin has been observed.

Course.—The growth of neuromata varies greatly in rapidity. When symptoms occur they most commonly last for a long time, increasing in severity, and from the long-continued pain the patient may ultimately be worn out. In other cases the symptoms show little tendency to increase, and in rare instances they may lessen in severity and even disappear.

DIAGNOSIS.—The recognition of the existence of a neuroma depends on its superficial position, or on the production of symptoms of damage to the nerve. The latter, when existing alone, are equivocal, since they may be due to pressure on the nerve from an adjacent growth, or even to neuritis.

The disease may be suspected if symptoms—pain, numbness, weakness—occur, limited to the distribution of a nerve trunk, and no cause of external pressure can be discovered, and the long course of the symptoms makes it unlikely that they are due to neuritis. The diagnosis can, however, only be certainly made when the tumor can be felt.

The diagnosis of the kind of neuroma, whether "true" or "false," *i. e.*, composed of nerve fibres or other tissue, is very difficult. Multiple neuromata are usually true, and of very slow growth; false neuromata are rarely multiple unless they are of infecting nature and rapid in development. The existence of idiocy, or other neuropathic indication, is in favor of the neural nature of the tumors. The lateral position of the growth on the nerve is in favor of its "false" character. The diagnosis of plexiform neuroma is only possible when the tortuous nodular cords can be felt.

Prognosis.—This depends on the existence of symptoms. If these are absent, unless the tumor is of rapid growth, there is good reason to hope that they will not occur. In multiple (true) neuromata the prognosis is better than in isolated growths. If symptoms are present the prognosis is less favorable, and depends on the tendency which they exhibit to increase.

Treatment.—Medicines are useless except in the syphilitic forms of nerve tumor. Extirpation is the only remedy. If the tumor is lateral, and can be shelled out, the removal entails little risk. If, however, the growth infiltrates the substance of the nerve, the affected part must be excised, and the ends brought together. The risk of permanent loss of function in the nerve is of course great, and in deciding on such an operation, the urgency of the symptoms, and the importance of the function of the nerve, must be taken into consideration. After extirpation, the tendency of cicatricial processes in nerves to go on to the formation of neuromata (seen in bulbous nerves) renders the chance of relapse considerable. Electrolysis or caustics are inadmissible. When neuromata are multiple, surgical treatment is out of the question. For pain, sedatives must be employed. In the case of terminal neuromata, whether the cutaneous "tubercula dolorosa" or the amputation neuromata, excision is practicable and unattended with the risks attending interference with a nerve in its continuity. It is also usually successful. In amputation flaps, however, the chance of a relapse is considerable.

In plexiform neuroma, excision is undesirable, and no other treatment is of any use.

DISEASES OF SPINAL NERVES.

The diseases of the cranial nerves may be most conveniently considered in connection with the diseases of the brain. Here, therefore, only the affections of the spinal nerves will be described, in so far as they present special features. The effect of paralysis of the individual muscles has been already described in detail, and need not be here repeated, except in general terms, or in so far as the association of palsies resulting from a nerve lesion presents a particular character.

Phrenic Nerve.—Impaired function of the phrenic nerve is commonly the result of disease of the spinal cord. The roots of the nerve may, however, be damaged by disease of the bones of the spine. The deep position of the nerve trunk protects it from injury, but it is occasionally damaged in wounds of the neck, and in its course through the thorax it may be compressed by tumors, aneurisms, etc. Paralysis sometimes follows exposure to cold, and is ascribed to neuritis. It has been met with in lead poisoning (Duchenne). In disease of the cord and bones, both nerves are usually paralyzed; other causes commonly affect one only. The effect and symptom of paralysis is inaction of the diaphragm (see p. 45). If one nerve only is affected, the diaphragm does not descend on that side, but the movement of the other side lessens the resulting defect of movement, which can then be detected only by close observation. The loss of the action of the diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is readily produced by exertion; the breathing then becomes quick and the voice feeble. Any lung disease, such as an attack of bronchitis, is rendered far more serious by the diminished breathing power. When the diaphragm is paralyzed, the movement of the thorax is often increased, and the expansion of the lower part may draw forward the adjacent abdominal wall. This must not be mistaken for the effect of the descent of the diaphragm. Paralysis has to be distinguished from (1) abnormal nervous breathing. The diaphragm is used little in extraordinary breathing, which is chiefly by the upper part of the thorax, of the "superior costal type" as it is called. Hysterical and nervous patients will often breathe, for a time, only in this manner, even when at perfect rest, especially when they are under observation. Such breathing is no doubt facilitated in women by the fact that the diaphragm is habitually used by them less than by men. Repeated examination may be necessary to determine whether there is any real paralysis of the diaphragm in these cases. A single diaphragmatic inspiration settles the point. The patient's attention should be distracted, and she should not be aware of the object of the examination. (2) Inflammation of the diaphragm may arrest its movement, but is distinguished by the fact that it is usually secondary to adjacent inflammation, commonly of the peritoneum or the pleura. Characteristic pain, due to the inflammation of the serous membrane, is commonly present. (3) A primary and isolated degeneration of the muscular fibres of the diaphragm has been described by Callender and others as common after death, but it has not yet been proved to cause such inaction during life as might be confounded with paralysis. The distinction of paralysis, due to disease of the nerve trunk, from disease of the spinal cord, depends on the fact that in the latter case other muscles always suffer; in the former the diaphragm usually suffers alone. In disease of the nerve roots there are other indications of the position of the disease.

If there is reason to suspect neuritis, counter-irritation should be applied over the lower and inner part of the anterior triangle of the neck. The only other special point in treatment is the application of electricity. The nerve may be stimulated by pressing the rheophore deeply outside the lower part of the clavicular portion of the sterno-mastoid. The other pole may be placed at the epigastrium or over the corresponding half of the diaphragm. But the influence of electricity on paralysis of the diaphragm is not great.

NERVES OF THE UPPER LIMB.

The nerves of the arm and shoulder are derived from the five lower cervical and the first dorsal nerves. These interlace in the brachial plexus in such a complex manner that most of the nerves of the arm are derived from many spinal roots.

The nerve roots form, by their union, three trunks, which we may indicate by Roman numerals. They are formed thus: I, by the branch from the fourth, and the roots of the fifth and sixth cervical; II, by the seventh; and III, by the eighth cervical and the first dorsal. Each trunk divides into two parts, and the union of these divisions forms the three cords of the brachial plexus from which the nerves of the arm proceed. But before the primary trunks divide, certain nerves arise, the origin of which is thus less doubtful. The fifth and sixth cervical roots give origin directly to the posterior thoracic nerve for the serratus, and from the cord formed by their union springs the suprascapular nerve. The three cords of the plexus have the following relations: The posterior is derived from all three primary trunks and gives rise to the subscapular nerve, the circumflex, and the musculo-spiral (or radial, as the whole nerve is sometimes termed). The upper or outer cord is derived from the two upper primary trunks, *i. e.*, from the fourth, fifth, sixth and seventh cervical roots, and from it proceed one anterior thoracic and the musculo-cutaneous nerves, together with the outer head of the median. The inner or lower cord is derived only from the lowest primary trunk, *i. e.*, from the last cervical and first dorsal, and gives rise to the ulnar, the inner head of the median, the internal cutaneous, the intercosto-humeral, and to the second anterior thoracic nerves. It may be convenient to put these coarse anatomical relations in the form of a table.

Nerves.		*Primary Trunks.*		*Nerves.*
		I. 4, 5 and 6 C.	Upper cord.	External ant. thoracic. Musculo-cutaneous. Outer head of median.
Subscapular . . . Circumflex . Musculo-spiral . . .	Post.cord.	II. 6 and 7 C.		
		III. (8 C. 1 D.)	Lower cord.	Inner head of median. Ulnar. Internal cutaneous. Intercosto-humeral. Internal ant. thoracic.

These anatomical facts, however, give us little help in tracing the relation of the nerves to the spinal roots. The investigations of Ferrier and Yeo,* who ascertained the movements produced by faradizing the several spinal roots in the monkey, show us the way in which the movements, muscles and nerves are represented in the spinal roots. Their results are therefore of great interest, although we are not justified in transferring the facts to man except in so far as they receive confirmation from human anatomy and pathology.† Subject to this reservation, the chief results are important and may be thus stated.‡

The roots to which the several nerves are thus traced are as follows: Subscapular, 6 and 7

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* *Proc. Roy. Soc.*, March 21st, 1881.

† In the case of the leg there are some important discrepancies between the results in the ape and the conditions that obtain in man. These will be noticed in their proper place.

‡ Ferrier has since stated that the relations he gave (followed in the text) were all one nerve too high (*Proc. Roy. Soc.*, *1883*, vol. xxxv, p. 229), but this would make the innervation of the intrinsic muscles of the hand from the second dorsal nerve, which is certainly not the case in man.

C.; circumflex, 4 and 5 C.; musculo-spiral, 4, 5, 6, 7, 8 C.; musculo-cutaneous, 4, 5 C.; median, 5, 6, 7, 8 C.; ulnar, 8 C., 1 D.

Still more important are their observations on the relation of various movements of the arm to the nerve roots, and these are as follows:—

Cervical 4.—Elevation and retraction of the arm, flexion and supination of the forearm; by the rhomboids, supra- and infra-spinatus, biceps, brachialis and supinators.

Cervical 5.—Similar to the last, but without retraction of the arm, and with extension of the wrist and first phalanges; by the deltoid, serratus, flexors of elbow, extensors of wrist, and long extensors of fingers.

Cervical 6.—Adduction and retraction of the upper arm, extension and pronation of the forearm, flexion of the wrist; by contraction of the pectoralis, latissimus dorsi, triceps, flexors of wrist, pronators.

Cervical 7.—Adduction and rotation inward at the shoulder joint, flexion of the wrist, and of the fingers at the second phalanx; by the teres major, latissimus dorsi, subscapularis, triceps, and long flexors of the fingers.

Cervical 8.—Flexion of fingers and thumb so as to close the fist; flexion of wrist toward the ulnar side, pronation of forearm, extension of elbow; by the intrinsic muscles of the hand, the long flexors of the fingers and thumb, the flexors of the wrist, and the triceps.

Dorsal 1.—Adduction of the thumb, flexion of the fingers at the metacarpo-phalangeal joints; by the interossei, etc.

Thus most movements are related to many spinal roots. The most important relations are these: that of the deltoid, rhomboids, supra- and infra-spinatus, flexors of the elbow, and supinators, to the fourth and fifth cervical; that of the adductors of the arm and extensors of the elbow to the sixth and seventh nerves; pronation to the sixth and eighth; extension of the wrist to the fifth; flexion to the eighth; extension of the first phalanx to the fifth; flexion of the fingers to the seventh and eighth, and the action of the intrinsic muscles of the hand to the first dorsal. These facts will probably, in the future, find important practical applications.

Paralysis of the nerves of the upper limb may be due to disease of the nerve roots, of the plexus, or of the trunks that arise from these. It is convenient to consider, first, the diseases of the nerve trunks, and afterward those of the nerve roots, and of the plexus. From the roots themselves only one important nerve trunk is derived: the posterior thoracic nerve. The others spring from the plexus.

The morbid influences that affect the nerves of the arm are very varied. The brachial plexus passes close to the shoulder joint, and hence dislocation often damages the nerves, sometimes one only, sometimes several, and occasionally all the trunks. In fractures of the bones, the deeper nerves adjacent may be torn at the time of fracture, or may be afterward compressed by the callus that is formed, or by bandages that are applied. The course of certain nerves is, in some parts of the arm, especially apt to be the seat of wounds of various kinds, while that of others exposes them to pressure against the unyielding bone. Even slight pressure on these nerves is effective when prolonged, as it often is when warning sensations are unperceived in consequence of sleep. Hence, there is a group of "sleep-palsies" of the arm, chiefly of the musculo-spiral and median nerves.

Primary neuritis may involve any nerve, or the brachial plexus itself, but is not common. Secondary neuritis, set up by injury or by adjacent inflammation, is less rare; it sometimes extends upward, and, reaching the plexus, may spread to other nerves. Neuromata occasionally cause paralysis, but are also uncommon. Various morbid processes near the spine and in the neck may cause symptoms in the arms by involving the nerve roots that enter the plexus.

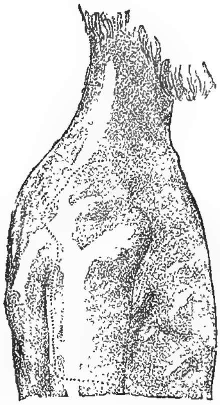
Posterior Thoracic Nerve—Nerve to the Serratus.—In consequence of its position and long course, the nerve to the serratus often suffers. After being formed in the substance of the scalenus medius muscle, by the union of branches of the fifth and sixth cervical nerves, it passes behind the brachial plexus and along the side of the chest to the lower border of its muscle. It is in the neck that the nerve is most often injured. It may be damaged there by direct pressure—as in carrying on the shoulder a heavy sharp-cornered object—or by severe muscular efforts, such as carrying a weight that does not press into the neck, wielding a heavy hammer, or long-continued exertion with the raised arm, as, for instance, in whitewashing a ceiling. In such cases it is not improbable that the nerve is damaged by the forcible or prolonged contraction of the muscle through which it passes. Falls and blows on the neck and shoulder are occasional causes; there is usually much bruising of the adjacent parts. Punctured and gunshot wounds also occasionally involve the nerve. The least frequent cause is exposure to cold, such as a draught on the neck, or sleeping on damp earth. I have once known it to follow parturition, developing four days afterward, doubtless from neuritis excited either by the muscular exertion, or by exposure to cold. The serratus suffers also in central disease, chiefly in progressive muscular atrophy and infantile paralysis; but in these it is always associated with palsy of other muscles. Isolated paralysis of the serratus is nine times as frequent in men as in women. It occurs chiefly in muscular workers, and during the active period of life, twenty-five to forty. The immediate causes sufficiently explain this relation to age and sex, and they also account for another fact—that it is far more frequent on the right side than on the left. It is sometimes bilateral, but the two nerves are never damaged at the same time. One man was affected on the right side after carrying heavy beams on the shoulder; he then carried them on the left shoulder, and the left serratus became paralyzed.

Severe neuralgic pains in the neck and about the shoulder commonly precede and accompany the onset of the affection. The symptoms of the paralysis of the serratus have been already described (p. 47). It is readily recognized by the recession of the posterior edge of the scapula from the thorax when the arm is put forward (Fig. 8, p. 48). In severe cases the muscle is found to have lost faradaic irritability, although it may still contract to the voltaic current. The course of a severe case is always tedious. Months may pass before improvement is manifested, and the paralysis is sometimes permanent. In treatment it is desirable to maintain the nutrition of the muscle, as far as possible, by electrical stimulation, and its superficial situation renders this easy. If counter-irritation is desirable, it should be applied over the position of the scalenus, since the lesion of the nerve is commonly at this place. Severe exertion with the arm should be avoided; an influence ble of inducing the palsy must seriously interfere with the recovery of ıjured nerve. When practicable it is desirable that the elbow should be ed in a sling, and this should be of such a length as slightly to raise the lder.

ne *Suprascapular nerve* arises from the trunk formed by the union of the ı, fifth, and a branch of the fourth cervical nerves, but its fibres come ı the fifth and partly from the fourth. It is not often damaged alone, is sometimes injured in dislocation of the humerus, and by falls on the ılder, usually together with the circumflex. In some of these cases injury may be to the upper part of the plexus (see Combined Palsies). result of disease of this nerve is palsy of the supra- and infra-spinatus 48). The latter is the more important, and causes a loss of the rotation vard of the humerus, interfering with many movements, and, among rs, with the movement of the pen along the line in writing. Undue k is thrown on the deltoid, which becomes fatigued in many actions, ı as sewing (Duchenne). The paralysis of the supra-spinatus is unim-ant unless there is also palsy of the deltoid. It can, in very slight degree, lement the deltoid, and if paralyzed with the deltoid the head of the ierus falls more than it does when the deltoid is paralyzed alone.

'he *Circumflex nerve,* although arising from the posterior cord of the us, seems to derive its fibres from the same source as the suprascapular, fourth and fifth cervical nerves. It supplies the deltoid and teres minor, the skin over the deltoid. The course of the ve renders it very liable to injury from disloca-ıs and falls on the shoulder, and from the pressure ı crutch. Simple neuritis and so-called "rheu-ic palsy" are rare, but the nerve has been affected ome acute diseases, as smallpox, possibly from ssure, and in rheumatic fever, perhaps by inflam-ion extending from the joint. It suffers, with er nerves, from disease of the upper part of the chial plexus.

FIG. 46.



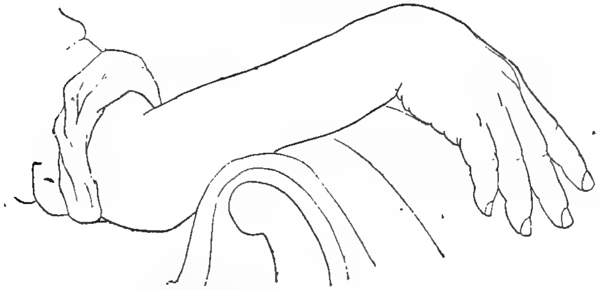
Disease of the circumflex nerve from pressure neuritis during acute illness; wasting of deltoid. The dotted line indicates the area of cutaneous anæsthesia.

'he chief symptom is paralysis of the deltoid (p. , which abolishes almost all power of raising the , a very trifling power of abduction by the supra-iatus alone remaining. The slight nerve supply the fore part of the deltoid receives from one of anterior thoracic nerves is insufficient to mitigate effect of the paralysis of the circumflex, although iay maintain slight power of voluntary contraction he anterior fibres, and this sometimes gives rise n erroneous impression that the paralysis of the umflex nerve is incomplete. The effect of the y of the teres minor is unimportant. The wasting of the deltoid causes hange in the shape of the shoulder. Sensation may be lost in the skin r the lower part of the muscle, and the loss sometimes extends on to the shoulder (Fig. 46). In some cases there is no anæsthesia, even when the muscle is wholly paralyzed, we have seen that this is often the case in nerve lesions (p. 71). Adhesions are apt to form in the shoulder joint, in part probably the result of trophic changes, since the circumflex supplies the articulation as well as the chief muscle that moves it. The diagnosis of the paralysis of the circumflex nerve is easy. The only condition readily mistaken for it is anchylosis of the shoulder joint in a stout individual, in whom the state of nutrition of the muscle is not readily perceived. The risk of error is increased by the fact that arthritis and paralysis may result from the same cause—a fall on to the shoulder. Passive movement at once solves the problem. The scapula moves with the arm in anchylosis, and not in palsy.

The *Musculo-cutaneous nerve* supplies the chief flexors of the elbow and the skin over the radial side of the forearm. It is scarcely ever paralyzed alone, but often suffers, with other nerves, in disease of the brachial plexus. The symptoms correspond to its function; there is paralysis of the biceps and brachialis (see p. 50), the effect of which is especially conspicuous when the arm is supinated and the supinator longus cannot flex the elbow. There may also be anæsthesia of the radial side of the forearm, front and back.

The *Musculo-spiral nerve* is more frequently paralyzed alone than any other nerve of the arm. Arising from the posterior cord of the brachial plexus, it seems to derive its motor fibres from all the nerve roots that enter the plexus except the first dorsal. It supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and fingers, both the supinators, also the skin on the radial side of the back of the hand, the back of the thumb, index finger, and half the middle finger. It is thus the extensor nerve of the arm, and has a more complete relation to a single function than is common among the nerves of the limbs; a fact that has given rise to diagnostic error, since a functional palsy suggests a central cause. The frequency with which this nerve suffers is due to its course. As it leaves the brachial plexus to wind round the bone, its position exposes it to injury in dislocation or from the pressure of a crutch; the most common form of "crutch palsy" is paralysis of this nerve. Lying as it does close to the humerus, it is readily torn by fracture, or compressed by callus, and it suffers gravely from even temporary pressure against the hard bone. Such pressure is often exerted during sleep. The nerve may be paralyzed by the pressure to which it is exposed when the body, with the arm beneath it, rests on the ground or on a hard bed. Thus, a man slept all night on a bench, lying on his right side with his arm beneath him, and woke in the morning with this nerve paralyzed. Curiously, a precisely similar event had occurred to him three years before. In other cases the pressure on the nerve is that of a hard and sharp object over which the arm is placed during sleep, as the edge of a chair or the side of a couch. The continental custom of tying together, behind the body, the arms of a prisoner, often causes paralysis of this nerve, sometimes on both sides. In Russia it is not uncommon in infants, from the popular practice of binding the arms to the body and then laying the child to sleep on its side. I have twice seen paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, and once from throwing a stone with violence. In each the nerve was at once completely paralyzed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps. Neuritis due to cold is often assumed to be a cause and is possibly sometimes effective, but the exposure to cold has usually been during sleep, and, as Panas and others have maintained, it is probable that the paralysis was due to compression rather than to cold. The same influence may also have been operative in cases in which the nerve has been paralyzed during an acute disease. Although, in a case of paralysis during typhus, described by Bernhardt, neuritis was found after death, this was at the spot at which pressure is usually effective, and the inflammation was probably thus excited. The muscles involved in lead palsy are some of those supplied by the musculo-spiral, but we do not know that the poison acts on the nerve itself.

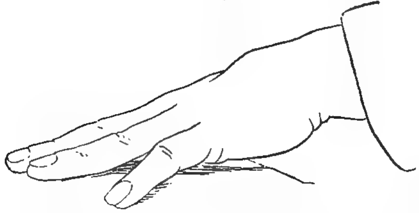
FIG. 47.



Paralysis of the musculo-spiral nerve; maximum extension of wrist and fingers. The extension of the fingers progressively diminishes from the first to the fourth. (*From a photograph.*)

The symptoms of disease of the musculo-spiral nerve are paralysis of the extensors of the elbow and of the wrist, the long extensors of the fingers and thumb, and the supinators (see pp. 49–51). All these are paralyzed by a complete lesion of the nerve near the brachial plexus. When the lesion is near the middle of the humerus, as it is in most cases of compression, the triceps generally escapes, but not always. The supinatus longus is usually paralyzed, but it escapes if the lesion is below the origin of its branch, and may also escape if the injury to the nerve is incomplete. In sleep-palsy it usually suffers, but I have seen it unaffected in a case in which the ext. carpi radialis was also but little affected, and Bernhardt has noted the escape of the supinator in an otherwise complete paralysis from dislocation of the humerus. The extensor palsy causes characteristic wrist-drop, and loss of the power of extending the first phalanges of the fingers and the thumb (see p. 51). There is sometimes a gradation of palsy from the first finger, in which it is least, to the fourth, in which it is greatest (Fig. 47). The same gradation is seen in lead palsy. It is not easy to explain. The action of the flexors is feeble, from the loss of support; in most cases of complete palsy I have found the power of flexion reduced from this cause to one-third of the normal. The loss of the power of supination is a grave inconvenience. If an object is grasped firmly the arm becomes pronated. The patient tries to compensate for the loss by putting the elbow against the side and rotating the humerus. The pronators may ultimately become shortened. The over-flexion of the carpus, and its deficient support by the extensor tendons, leads to a prominence of synovial sacs, and perhaps of the bones, at the back of the carpus (Fig. 48). In severe cases the muscles waste, and the maximum circumference of the limb below the elbow may be a quarter or half an inch less than on the other side. The electrical reactions depend on the severity of the lesion of the nerve; commonly there is well-marked degenerative reaction. The affection of sensibility in the area supplied by the nerve is very variable. The skin of the upper arm rarely loses feeling; in the hand sensation may be normal, although the muscular paralysis is complete. There may be subjective "tingling" in the part, although there is no loss of sensibility. The diagnosis is easy in most cases. The affection is distinguished from lead palsy by its common limitation to one arm and by the affection of the supinator, but these distinctions, although strongly suggestive, are not quite absolute. The palsy of the nerve, moreover, is usually sudden in onset; that from lead is gradual. These characters, and the commonly obtrusive cause, always suffice for the diagnosis. In bilateral wrist-drop, disease of the nerve would not be thought of, unless it followed a cause acting on both sides. It must be remembered, however, that this nerve is one most frequently affected in multiple neuritis; in this disease both arms suffer, the legs are also often involved, pains are conspicuous, and the palsy is rarely limited to a single nerve region. The prognosis in disease of the musculo-spiral nerve depends on the severity of the lesion, as indicated by the electrical reaction. When there is evidence of nerve degeneration the paralysis usually lasts for some months. Recovery ultimately occurs in almost all cases. The treatment is that for neuritis already described, but attention should be paid to the posture of the limb, so as to avoid tension on the affected part of the nerve. For this reason, and also because strong contraction of the triceps may further injure the nerve, extension of the elbow should be avoided in cases of lesion of the nerve as it winds round the humerus.

FIG. 48.



Prominence at back of hand from paralysis of the extensors. The patient was suffering from wrist-drop due to silver poisoning.

The *Median nerve* supplies the pronators, the radial flexor of the wrist, the flexors of the fingers (except the ulnar half of the deep flexor), the muscles that abduct and flex the thumb, and the two radial lumbricales. Its motor fibres seem to have an extensive origin from all the cervical roots that enter the brachial plexus. It subserves sensation on the radial side of the palm, on the front of the thumb, of the first two fingers, and of half the third finger, and also, at least in many persons, on the back of the last phalanx of the index and middle finger; sometimes also on the adjacent part of the back of the ring finger and the back of the last phalanx of the thumb. Isolated palsy of this nerve is not frequent, and generally results from wounds of the forearm, or fractures of the forearm bones, rarely from injuries in the upper arm. The nerve is occasionally the seat of neuritis. I have known it to be paralyzed at the wrist a few hours after a severe sprain of this joint. Webber has recorded a case in which it seemed to be injured by a violent contraction of the pronator teres. When there is complete damage to the nerve, pronation is impossible beyond the mid-position, to which the supinator longus can bring the forearm, and an attempt is made to supplement this by rotating the humerus inward, and separating the elbow from the side. The wrist can only be flexed with a strong inclination toward the ulnar side. The thumb is in persistent extension and adduction (like the thumb of the ape, Figs. 20 and 21, p. 54), and cannot be opposed to the tips of the fingers. The power of flexing the second phalanges on the first is lost, and also that of flexing the distal phalanx of the first and second finger, but this phalanx can still be flexed in the third and fourth fingers, by the ulnar half of the flexor profundus (see p. 51). Interosseal flexion of the first phalanx is still possible, and the unopposed extensor action of the interossei on the middle and distal phalanges tends to cause a subluxation of the articulations concerned. The wasting of the thenar muscles is usually conspicuous. The condition of the hand resulting from these palsies is very characteristic. The sensory loss is variable; it may be absolute or absent. If there is anæsthesia on the palmar surface it will often be found also on the dorsal aspect of the extremities of the first two fingers. Lesions of the median nerve, if rare, are apt to be severe, and their course correspondingly prolonged.

The *Ulnar nerve* comes, through the inner cord of the plexus, from the last cervical and first dorsal roots, and its origin from the lowest part of the cervical enlargement gives it an important relation, since it is the first, of all the brachial nerves, to be affected by disease that ascends from the dorsal to the cervical region of the spinal cord. The nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, some of the lumbricales, the adductor and inner head of the short flexor of the thumb. Its sensory area is the ulnar side of the hand, back and front, more extensive on the back of the hand (two fingers and a half) than on the front (one finger and a half). The course of the nerve, superficial behind the elbow and at the wrist, renders it liable to separate injury. It is often implicated in wounds of the arm, and injured in operations about the elbow joint, by dislocations of the shoulder and elbow, and by fractures of the bones of the forearm. It is occasionally the seat of neuritis, and, like the musculo-spiral nerve, it is sometimes paralyzed by pressure. It is probable that pressure often acts by setting up neuritis. At the elbow, the projecting bones preserve the nerve from direct pressure; although Duchenne describes paralysis in men who, while at work, rest the bent elbow on a hard substance, this mechanism is certainly rare. It is much more common for the nerve to suffer in long-continued flexion of the elbow, without external pressure; and from this cause the nerve is sometimes paralyzed during sleep. I have three times seen a sleep-palsy of the ulnar nerve. The tension may set up neuritis, and is especially effective if the resistance of the tissues to morbid influences is lessened by general ill-health. Many persons must have noticed that if, when out of health, they sleep with the elbow bent, they wake with tingling, and even loss of feeling, in the region supplied by the ulnar nerve, although this may not happen when they are in good health. If the ill-health is profound, as in an acute illness, local neuritis of considerable intensity may be set up. Thus a lady, a few days after childbirth, who was prostrated by a long, exhausting labor, noticed tingling in the side of the hand, and, whenever she bent her elbow, a sensation "as if she had knocked her funny bone." Paralysis of the muscles supplied by the ulnar nerve followed in a few hours, and in a fortnight there was distinct wasting. When I saw her, six months later, the ulnar nerve behind the elbow was distinctly thickened. She had had a similar, but more transient, attack after a previous confinement. Another patient had a similar palsy during the course of typhoid fever. Bernhardt has described a case of palsy of both ulnar nerves during the course of typhoid. In such conditions, not only is the tissue health lowered, but warning sensations are often unperceived.

The importance of the interosseal flexion of the fingers for many actions, such as writing, renders palsy of the ulnar nerve peculiarly disabling. Several illustrations of the symptoms have been given in connection with palsy of the interossei (p. 52). In flexion of the wrist, the hand deviates toward the radial side, and persistent distortion may take place. Adduction of the thumb is lost, and so are most movements of the little finger. The fingers cannot be flexed at the first or extended at the other joints, but the loss is slighter in the first two fingers than in the others, because their lumbricales are supplied by the median nerve. In time the opponents of the interossei, by their contracture, lead to over-extension of the first phalanges and flexion of the others; the "claw-like hand" is produced (see p. 52), but this is less complete than in cases of progressive muscular atrophy, on account of the escape of the first two lumbricales. When the palsy is imperfect it may be possible to extend the second and third phalanges, if the first can be over-extended, and an advantage thus given to the interossei, by the lengthening of the course of their tendons, just as in partial paralysis of the long extensors they may be able to extend the digits if the wrist is flexed, but not if it is extended.

The loss of sensation, as in the case of the other nerves, varies much both according to, and irrespective of, the degree of lesion of the nerve. Subjective sensations are common, with and without anæsthesia, and those who have had neuritis are sometimes unable to bend the elbow for more than a few minutes without feeling tingling in the area supplied by the nerve.

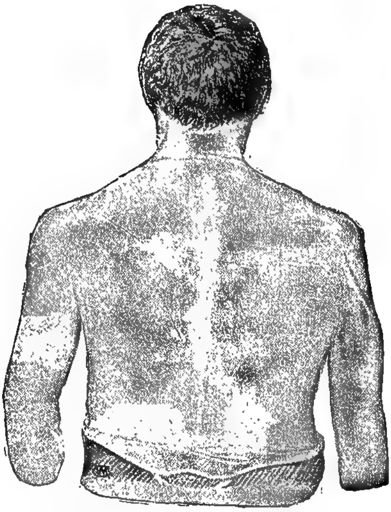
The diagnosis of disease of the ulnar nerve is easy. A difficulty is occasionally caused by the circumstance already mentioned, that disease at the lowest part of the cervical enlargement may cause symptoms limited to the region of this nerve, but a knowledge of the fact is usually sufficient to prevent an error.

Combined Palsies of the Nerves of the Arm.—Paralysis in the region of two or more of the nerves of the arm is very common, and results from many causes. It may be due to disease of the spinal cord, or to disease of the nerve roots within the spinal canal, but with these we are not now concerned. The most frequent causes of such combined palsy outside the spinal canal are the following: (1) Morbid processes in the neck, affecting the nerve roots outside the spine or the upper part of the brachial plexus. (2) Diseases of the plexus itself, neuritis, and especially dislocations of the shoulder. (3) Fractures of the bones of the forearm. (4) Ascending neuritis, "neuritis migrans," which, commencing in a single nerve, may ascend it to the plexus, and there spread to the fibres of other nerve trunks.

We may consider first the cause last named. An injury of a single nerve is sometimes followed by an extension of the motor and sensory symptoms to the regions supplied by other nerves. This can only be explained by an ascending neuritis reaching the junction of the nerves at the brachial plexus, and there spreading, often in what seems a random manner. Thus a lady cut her wrist so as to leave a scar an inch long, over the position, and in the direction, of the median nerve. The injury caused paralysis and wasting of all the muscles in the hand supplied by this nerve. This palsy was followed by a gradual loss of power, with diminished faradaic irritability, in the long extensor of the fingers, the ulnar flexor of the wrist, and also by defect of sensation in the skin supplied by the ulnar nerve. Hence the neuritis must have ascended to the plexus, and there have spread along each root of the median nerve. Again, a woman cut her right hand, with a broken stone bottle, along the hypothenar eminence. Three weeks afterward the muscles of the hand supplied by the ulnar nerve gradually became paralyzed, with wasting and loss of irritability, and in the area of the skin supplied by the nerve there was first tingling and then diminished sensibility. Burning pain spread up the front of the forearm, and inner side of the upper arm, to the axilla; all the forearm muscles lost power, the flexor carpi ulnaris, and the flexor sublimis digitorum in greatest degree; the tingling and lessened sensibility spread to the fingers supplied by the median nerve, and the abductor pollicis wasted. In this case, also, there must have been an ascending neuritis of the ulnar, spreading to the median at the brachial plexus.

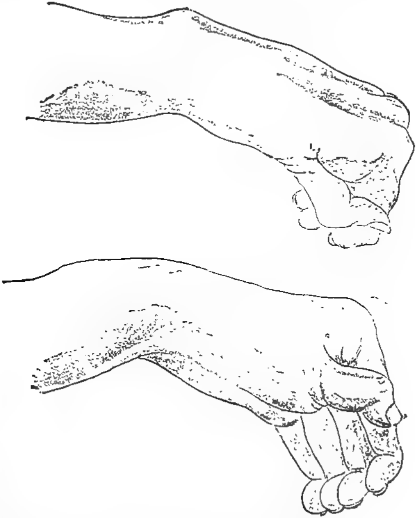
The brachial plexus is occasionally the seat of a primary neuritis, sometimes apparently rheumatic in origin, sometimes developing without any distinct exciting cause. The symptom of such neuritis is pain, acute or burning in character, in the position of the plexus, extending along the course of some of the nerve trunks, and accompanied by various indications of organic damage to the nerves that arise from the plexus. These cases are always most tedious in their course. Fortunately they are rare. A far more common cause of damage to the plexus is dislocation of the shoulder, especially subcoracoid dislocation, in which the displaced head of the humerus necessarily compresses the nerves. The extent of injury varies greatly; only one nerve may suffer, or not one may escape. Every muscle of the arm may be paralyzed, from the deltoid downward. In most cases the injury is severe in degree; there is rapid wasting of the muscles, with the reaction of degeneration, and there is a great tendency to the occurrence of trophic changes in the skin. It was in a case of this character that the forearm and hand were covered with blisters from the application of hot water that seemed merely warm to a healthy hand (p. 72).

FIG. 49.



Combined palsy of deltoid, supra-spinatus, and infra-spinatus, from a fall on the shoulder.

FIG. 50.



Left hand of a patient suffering from a growth beside the lower cervical spine compressing the nerve roots. There was anæsthesia of all parts supplied by the brachial and cervical plexus. The arm was adducted, the elbow flexed, the hand in the posture shown, flexion of first, extension of second, strong flexion of last, phalanges, the first phalanx of the thumb over-extended, the second flexed. Rigidity extreme, and an attempt to overcome it caused great pain. There was also some contracture of the foot (equino-valgus).

Fracture of the humerus may damage both the musculo-spiral and ulnar nerves, rarely the median. Fracture of the bones of the forearm often injures both the median and ulnar nerves.

Injuries to the neck sometimes cause a partial paralysis of the arm of peculiar distribution, the special characters and significance of which were first pointed out by Erb.* A similar paralysis sometimes comes on apart from injury. The muscles affected are the deltoid, often the supra-spinatus and infra-spinatus, the biceps and brachialis anticus, and the supinators. Erb found that there is one spot between the scaleni, corresponding to the sixth cervical nerve, at which electrical stimulation puts all these muscles in action. Hoedemaker, who has described two cases of this palsy, finds the motor point in a line drawn from the sterno-clavicular articulation to the seventh cervical spine, 1.5 centimetres from the edge of the trapezius. The palsy is apparently dependent on disease of the roots of the fifth and sixth cervical nerves, and the fifth, it will be remembered, receives a twig from the fourth.* Besides injuries, this group of palsies may result from non-traumatic processes, probably from neuritis, and also from growths in this situation. Duchenne and Seligmüller have also described paralysis of similar distribution produced in infants during birth, either by pressure from the position of the arm, or by traction on the neck with the finger or hook. Most obstetrical cases slowly recover. Those due to injury in adult life are often severe; the symptoms continue for a long time, and may be permanent.

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* Heidelberg Society, 1874, *Ziemssen's Handbuch, 1874*, Bd. xii, pt. 2, p. 509; see also Bernhardt, *Zeitsch. f. kl. Med., 1882*, Bd. iv, p. 415.

Another series of muscles is paralyzed when the lower nerve roots of the brachial plexus are diseased, the seventh and eighth cervical and the first dorsal. The palsy may be termed the "lower arm type;" those muscles escape that are involved in the "upper arm type." Thus a woman with a deep-seated growth in the lower part of the neck on the right side, presented palsy and wasting of the triceps, forearm flexors and extensors, and hand muscles. The deltoid biceps and supinators escaped.

Morbid processes connected with the bones of the cervical spine sometimes produce combined symptoms of irritation and palsy of the nerves of the arm, pain, hyperæsthesia, anæsthesia, paralysis of muscles, and extreme muscular contractures, often very irregular in distribution (such as are shown in Fig. 50). I have also twice seen purely sensory symptoms in the arm due to what was apparently syphilitic cellulitis about the vertebræ, causing deep-seated induration beside the spine, and quickly dispersed by iodide of potassium.

Diagnosis.—The diagnosis of diseases of the nerves of the arm has been for the most part sufficiently considered in connection with the special nerves. It consists chiefly in an application of the facts there stated. One or two more general considerations deserve, however, a brief mention. Some diseases of the spinal cord are first manifested in the arm, by muscular palsy, wasting, or by anæsthesia. The risk of error is prevented, in most cases, by the absence of any correspondence between these symptoms and the functions of special nerves, by the absence of any indication of morbid processes in the neighborhood of the nerves, and by the presence of other signs of disease of the spinal cord. It has been mentioned that disease in the lowest part of the cervical enlargement may be manifested only in the region of the ulnar nerve, the lowest in origin of all the brachial nerves. Such disease is, however, usually bilateral, and secondary to disease in the dorsal region of the cord, the indications of which have preceded the symptoms in the arm. A knowledge of these facts, and a careful consideration of the distribution and course of the symptoms, will rarely leave the observer in doubt.

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* Erb referred the palsy to disease of the sixth nerve. The experiments of Ferrier and Yeo point to the fifth and fourth roots as those innervating these muscles, but his subsequent correction indicates the fifth and sixth (see p. 86).

A greater difficulty in diagnosis is presented by some cases of functional disorder. The arm is a common seat of neuralgia, and the diagnosis between neuralgia and neuritis is often one of great difficulty, a difficulty that is increased by the circumstance that slight disseminated neuritis may unquestionably result from a primary neuralgia, so that the nerve trunks may become tender. The distinction rests on the variable and intermittent character of the initial pain, on the secondary character of the tenderness of the nerves, and on the absence of interference with the function of their fibres. A similar difficulty is presented by some cases of "occupation neurosis," of which "writers' cramp" is the most common form. Sensory symptoms, common in some degree in these affections, may predominate over the motor symptoms, and may consist of nerve pains and tenderness. The fact that the pain is at first excited only by one kind of muscular effort, the initial limitation of the disability, and the absence of any actual paralysis, motor or sensory, usually suffices for the diagnosis. These cases are considered more fully in the chapter on "Occupation Neuroses."

TREATMENT.—There is little in the treatment of the diseases of the nerves of the arm that requires special mention. The chief measure is the treatment of the cause of the paralysis. Any present source of pressure must be removed as far as possible. The ends of a divided nerve must be sutured. It is remarkable how quickly this has sometimes been followed by the restoration of conducting power. The posture of the arm must, in all cases, be such as to avoid tension on an injured or inflamed nerve. The treatment of neuritis is that already described. Electrical stimulation of the muscles is of great importance in all cases of severe damage to the nerves. Even in old and stationary cases it sometimes is the means of starting some improvement.

NERVES OF THE LOWER LIMB.

Disease of the nerves is far less common in the lower than in the upper limb, with one important exception—the primary disease of the sciatic nerve that goes by the name of "sciatica." This affection is reserved for separate description in the next section.

The nerves of the leg are derived from the lumbar and sacral plexuses. The *lumbar plexus*, consisting of the first three and a half lumbar roots, supplies the skin of the lower part of the abdomen, of the front and sides of the thigh, of the inner side of the lower leg and foot. It supplies also many muscles—the cremaster, and those that flex and adduct the hip joint, and those that extend the knee. Its branches for the leg are the obturator and anterior crural nerves. The *sacral plexus* consists of the fifth lumbar root and half the fourth (lumbo-sacral cord) and the first four sacral nerves, of which, however, only the upper three have to do with the leg. This plexus innervates the extensors and rotators of the hip, the flexors of the knee, and all the muscles that move the foot, together with the skin of the gluteal region, the back of the thigh, the outer side and back of the lower leg, and most of the foot. Its chief nerves are branches to the outward rotators of the hip, the gluteal nerve, and the small and great sciatic.

The results obtained by Ferrier and Yeo (see p. 86) on stimulating the roots of the lumbar and sacral plexuses in the monkey, may be thus summarized:—

Lumbar I and II. Lower abdominal muscles (not cremaster).
III. Psoas and iliacus, sartorius, extensors of knee (flexion of hip and extension of leg).
IV. Glutei, adductors, extensor cruris, peroneus longus (extension of hip and knee, elevation of outer side of foot).
V. Glutei, hamstrings, and all the muscles in front and back of lower leg (rotation of thigh outward, flexion and rotation inward of leg; extension of foot with elevation of outer edge; flexion of distal phalanges of toes).
Sacral I. Hamstrings, calf muscles, long flexor of toes, intrinsic muscles of foot (slight outward rotation of thigh, flexion of knee, extension of foot, adduction of great toe, flexion of first phalanges of all toes and of both phalanges of great toe).
II. Intrinsic muscles of foot ("interosseal" flexion of toes, similar to the last).

These results cannot be simply applied to man. For instance, it is certain that in man, one flexor of the hip, the psoas, and the cremaster, are largely innervated from the second lumbar, but no indication of the action of either of these could be observed on stimulating this root in the monkey.

The *Lumbar plexus* itself is sometimes damaged by growths in the abdomen (especially by those that spring from the lumbar glands) and by psoas abscess, and the nerve roots may suffer in disease of the bones of the vertebræ. The plexus may also suffer when inflammation ascends the lumbo-sacral cord from the sacral plexus, and it is occasionally the seat of spontaneous neuritis. The *obturator nerve* is rarely affected alone; it has been sometimes damaged in the course of parturition. The *anterior crural nerve* has suffered from the same cause, and is sometimes injured by wounds of the groin or thigh, or by dislocation of the hip joint.

Of the interference with movement that may result, the paralysis of the flexors of the hip depends on the position of the disease. This paralysis is total only when the lumbar plexus is damaged. Disease of the anterior crural nerve, within the abdomen, does not affect the psoas, but may paralyze the iliacus, and so weaken, without abolishing, the power of flexion. The chief symptom of disease of this nerve is the loss of power in the extensors of the knee, and the wasting of these muscles, together with loss of the knee-jerk which results from the interruption of the reflex arc. The effect of these palsies on movement is very serious (see p. 55). Anæsthesia involves the whole of the thigh (except a strip along the middle of the back), the inner side of the leg and foot, and the adjacent sides of the first and second toes. In many cases the paralysis is incomplete, and the symptoms present corresponding variation. Irritation of the nerve may cause severe pain in the region supplied by it. This is sometimes an early symptom of a growth near the spine. The pain in neuritis of the plexus may extend along the course of the ilio-hypogastric, ilio-inguinal, and genito-crural nerves, to the lower part of the abdomen and groin.

Paralysis of the obturator nerve causes a loss of the power of adduction of the thigh, so that the patient cannot put one leg across the other. Rotation of the thigh outward is interfered with, but in slighter degree than adduction. The effect of these palsies (described on p. 55) is far less serious than is the disability which results from disease of the anterior crural.

The *Superior gluteal nerve* occupies an intermediate position between the two plexuses, arising, as it does, from the lumbo-sacral cord, which descends from the fourth and fifth lumbar roots. Its disease, which is very rare in isolated form, causes paralysis of the gluteus medius and minimus, with a loss of abduction and circumduction of the thigh (see p. 55).

The *Sacral plexus* is sometimes damaged by growths in the pelvis, by pelvic inflammation of various kinds, and by compression during parturition. It is also often the seat of neuritis, which, however, less frequently begins in the plexus, than ascends to it from the sciatic nerve. Apart from spontaneous neuritis, which will be separately described, the sciatic nerve outside the pelvis is occasionally injured by wounds, rarely by dislocations of the hip, often by disease of the bone, and by adjacent morbid growths. It is also a relatively frequent seat of neuroma. Of the terminal branches, the external popliteal, by its superficial course, and proximity to the hard fibula, suffers from traumatic lesions of various kinds; it is also prone to spontaneous neuritis. This nerve is homologous with the musculo-spiral nerve of the arm, and presents an analogous liability to disease. The posterior tibial nerve is more secure in its deeper course, but may be damaged by fracture of the bones.

The symptoms of palsy of the sciatic nerve vary much in their character, according to the position of the disease. The *small sciatic* is implicated only when the mischief involves the pelvic plexus, and it scarcely ever suffers alone. The effect is palsy of the gluteus maximus, which interferes with rising from a seat more than with walking (see p. 55). There is, also, anæsthesia of the skin in the middle third of the back of the thigh, and in the upper half of the calf. A lesion of the sciatic nerve, near the sciatic notch, paralyzes the flexors of the leg (which are also extensors of the hip; see p. 56), and all the muscles below the knee. If the lesion is below the upper third of the thigh, the flexors of the leg escape. The anæsthesia that results from a lesion of the nerve below the origin of the small sciatic, involves the outer half of the leg, the greater part of the dorsum of the foot, and all the sole.

The symptoms of disease of the branches of the sciatic are as follows:—

That of the *external popliteal nerve* causes paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. The effect of this is a loss of all power of flexing the ankle and of extending the first phalanx of the toes (see p. 57). The foot cannot be raised from the ground in walking, and talipes equinus ultimately results (Fig. 24, p. 57), which may be attended with persistent flexion of the first phalanges of the toes from contracture of the unopposed interossei. There is also anæsthesia on the outer half of the front of the leg, and on the dorsum of the foot.

Disease of the *internal popliteal nerve* paralyzes the popliteus, calf muscles, tibialis posticus, and long flexors of the toes, as well as the muscles of the sole. In addition to the disability which characterizes paralysis of the plantar muscles, there is loss of the inward rotation of the flexed leg, if the disease is so high as to involve the branch to the popliteus, and there is also loss of the power of extending the ankle joint. Talipes calcaneus results (Fig. 23, p. 57). The sensory loss is anæsthesia on the outer part of the back of the leg in the lower half, and on the sole.

The *plantar nerves* rarely suffer alone. A lesion of the *internal nerve* causes anæsthesia on the inner part of the sole, and plantar surface of the three inner toes and half the fourth, together with paralysis of the short flexor of the toes, the plantar muscles of the great toe (except the adductor), and of the two inner lumbricales.

Disease of the *external nerve* produces anæsthesia of the skin on the outer half of the sole, the little toe and half the fourth, paralysis of the flexor accessorius, the muscles of the little toe, all the interossei, the two outer lumbricales, and the adductor of the great toe. The effect of the palsy (see p. 58) is serious, since the toes cannot take their proper share in propelling the body forward in walking, and they gradually become flexed at the last two joints and extended at the others, from the contracture of the opponents of the interossei,—a position of the toes that causes serious inconvenience in walking.

DIAGNOSIS.—The diagnosis of diseases of the nerves of the leg is determined by the same general principles as those that have been mentioned as applicable to the nerves in general, and to the nerves of the arm in particular. The limitation of the symptoms to the functional areas of individual nerve trunks, the evidence afforded by nutrition and irritability that the muscles are separated from the spinal cord, the implication of the sensory functions, and often the tenderness of the affected nerves, indicate, in most cases, the seat of the disease. To these signs are often added other indications of a local cause, corresponding, in its incidence, with the nerve to which the symptoms point.

The relation of nerve trunks to nerve roots, although by no means simple, is certainly less complex in the case of the nerves of the leg than in those of the arm. This is especially the case in the lumbar plexus, and it leads to an occasional difficulty in diagnosis. Pressure on the spinal cord, for instance, at the level of the origin of the fourth lumbar roots, may cause symptoms identical with those of a partial lesion of the anterior crural nerve. Paralysis of the extensors of the knee, loss of the knee-jerk, anæsthesia in the front of the thigh, were present from this cause in a case of meningeal gumma. But other evidence of a spinal lesion is rarely absent under such circumstances, and, in this patient, a foot clonus, due to the pressure on the pyramidal fibres, left no doubt as to the situation of the disease. Another difficulty arises from the long course of the nerve roots in the cauda equina, disease of which may simulate that of the nerves of the leg. But the symptoms are commonly bilateral in consequence of the proximity of the nerve roots of the two sides. In all cases in which the symptoms are bilateral (unless there is evidence of a disease known to cause symmetrical lesions, such as multiple neuritis), the suggestion is that the disease is situated where the motor or sensory paths of each side are so near that they can be affected by a single lesion, *i. e.*, that the disease is within the spinal canal. But here, as in other cases, we cannot reverse our diagnostic rules. Disease of the spinal cord does not always cause bilateral symptoms. A limited lesion of one anterior cornu may be so placed as to paralyze the muscles supplied by a single nerve, and a doubt may be felt as to the central or peripheral origin of such palsy. The muscles supplied by the anterior crural nerve, and the muscles in the front of the lower leg supplied by the external popliteal, are those of which the central palsy sometimes leads to doubt. But the mode of onset, the presence or absence of sensory symptoms, the rarity of acute spinal palsy except in childhood, and of nerve lesions except in adult life, the wider initial prevalence of the palsy in acute, and its later extension in chronic, cornual disease,—these suffice as a rule to remove any doubt that may at first be felt.

It is important to remember that the pressure of a growth may cause either a chronic or an acute affection of the nerves. The chronic symptoms result from pressure; the acute from a neuritis set up by the pressure and irritation. Thus neuritis, while it does not suggest, does not exclude, a cause outside the nerve.

TREATMENT.—The treatment of diseases of the nerves of the leg must depend, in each case, on the nature of the lesion and the character of the symptoms. It does not differ from that of disease of the nerves of the arm, and its general principles have been already stated. More care, perhaps, is needed to avoid increasing present mischief, or inviting a relapse, by exposure to cold, or by fatiguing exertion. More care is also needed to obviate the tendency to secondary contractures in the case of palsies of long duration, and in those attended by pain, in which the patient seeks ease in postures to which the muscles only too readily adapt themselves. The contraction of the hamstrings, from constant flexion of the knees, is that which occurs most readily, and is most serious when the patient recovers from the palsy, or loses the pain, which led to the adoption of the position. The contracture of the calf muscles, which occurs when the flexors of the ankle are paralyzed, also constitutes a serious obstacle to walking after recovery. A little timely care, in preventing these contractures, will often save a vast amount of later trouble. That of the calf muscles, however, which is due to the extension produced by the weight of the foot, as the patient lies, cannot always be altogether prevented.

SCIATICA.

As the word sciatica is commonly used, it is a general designation for all affections of which the chief symptom is pain in the region of the sciatic nerve. It is thus often applied to the pain which results from pressure on the nerve in the pelvis, as by a tumor. In a stricter use of the word, however, it is applied to painful affections of the nerve not due to any cause outside it. The two varieties may be distinguished as secondary and primary sciatica. Primary sciatica is commonly regarded as a neuralgia. This view is probably in the main erroneous; the vast majority of cases of sciatica are really cases of neuritis of the sciatic nerve. The reasons for this opinion are given in the section on the pathology of the disease.

CAUSES.—Sciatica is far more frequent in males than in females; the proportion has been very variously estimated. It is difficult to obtain statistics on the malady that are free from sources of fallacy, since comparatively few cases enter general hospitals. The incidence of severe sciatica seems to be fairly presented by the 137 cases that were treated in the Devonshire Hospital, Buxton, during the year 1883, and there is no reason to suppose that slight cases differ, in this respect, from those that are severe. The percentage of males is 82; of females, 18; giving a ratio of 4 to 1, which is probably very near the truth. The disease is unknown in childhood, and rare in the second decade of life. It is most frequent between forty and fifty, next between fifty and sixty, and next between thirty and forty. The percentage distribution of the 137 cases at Buxton is as follows: Ten to twenty, 3 per cent.; twenty to thirty, 9 per cent.; thirty to forty, 19 per cent.; forty to fifty, 29 per cent.; fifty to sixty, 26 per cent.; sixty to seventy, 13 per cent.; over seventy, 1 per cent. The influence of age is nearly the same in each sex.

Two constitutional conditions are potent factors in the production of sciatica,—gout, and that form of rheumatism which affects chiefly the fibrous tissues of the muscles. These two constitutional states cannot always be entirely separated, but many of the sufferers are of opposite types; some are stout, plethoric, distinctly gouty; others pale, weakly, and rheumatic. It is, I think, most common among those who are gouty, but do not suffer from articular gout. Such individuals often have many attacks. Gouty inheritance is sometimes to be traced in those who suffer early from the disease. Syphilis has been supposed in some cases to be the cause of a sciatic neuritis, but cases in which the syphilitic nature of the disease is certain are extremely rare.

An exciting cause is to be traced only in a minority of the cases. Exposure to cold is the most common. It is usually local exposure, as by wet boots, standing in water, etc.; sometimes, however, a general chill of the body determines an attack. The exposure to cold may be even more direct, as by sitting on wet grass. I believe that draughty water-closet seats are answerable for some cases. The neuritis occasionally arises by the extension to the nerve of adjacent rheumatism. Thus I have known an attack of that form of lumbago which is localized in the fibrous attachments of the muscles at the back of the sacrum, to pass into sciatica, distinctly by the rheumatic affection of the fibrous tissues, extending along the fasciæ to the nerve sheath in the neighborhood of the sciatic notch. Mechanical causes also sometimes excite the disease, and still more often coöperate with other influences. The pressure of the edge of the chair, in those who sit much, is the most frequent mechanical cause. Muscular over-exertion has been thought occasionally to excite the disease. If the nerve is already tender, a strong contraction of the muscles at the back of the thigh, especially when the knee is flexed, and the muscles can freely shorten and widen, may produce acute pain in the nerve, evidently by its compression. It is conceivable, therefore, that this cause may be effective in a predisposed person. Various morbid processes within the pelvis may cause symptoms of sciatica, usually by acting on the sacral plexus. Thus rectal and other tumors, pelvic inflammation, and injury during labor are occasional causes. A loaded rectum has been thought to be capable of producing it. Mechanical congestion of the plexus of veins which lies over the sacral plexus of nerves is usually regarded as an occasional cause, but this is somewhat hypothetical, and its efficiency can scarcely be demonstrated. Lastly, the sciatic nerve may be secondarily involved in mischief that is outside the pelvis. The most frequent cause of this is disease of the bone, and especially disease of the hip joint. In all these forms of secondary sciatica the symptoms are due partly to pressure on the nerve, partly to inflammation excited in it by the pressure, or extending to it from the adjacent disease.

Pathological Anatomy.—Sciatica being a disease that does not itself cause death, observations of the morbid changes are few. In most cases that have been examined, distinct evidence of neuritis has been found, chiefly involving the nerve sheath, but extending in some cases to the interstitial tissue. The changes are those that have been already described in the account of neuritis (p. 74). In recent cases there is swelling and redness of the sheath, sometimes there are small hemorrhages, and, in severe cases, similar but slighter alterations in the interstitial tissue, with secondary damage to the nerve fibres. The signs of inflammation are most intense at the sciatic notch, and opposite the middle of the thigh. They may be limited to one or both of these places, or they may be greatest there, and extend, in slighter degree, over a considerable tract of the nerve.

Symptoms.—The chief symptom of sciatica is pain along the course of the nerve trunk, often also along that of its branches, and sometimes in the area of its distribution. The affection may begin suddenly, especially in cases of rheumatic origin,—as suddenly as lumbago. More frequently the onset is gradual; slight pain is felt along the back of the thigh, on movements and in postures that make the nerve tense, or cause pressure upon it. As the disease increases, the pain is more readily produced, and becomes greater in degree, until at last the patient is free from induced pain only when at rest and the leg is in a certain position. Any movement that makes the nerve tense causes extreme suffering, and to avoid this, the knee, in walking, is kept slightly flexed, and the leg held stiffly so as to avoid painful changes in the tension of the nerve. As the pain on movement increases, spontaneous pain is added, at first chiefly felt in the nerve trunk, but soon spreading to its branches and distribution. It is usually most intense in certain spots—(1) above the hip joint, near the posterior iliac spine, (2) at the sciatic notch, (3) about the middle of the thigh, (4) behind the knee, (5) below the head of the fibula, (6) behind the external malleolus, (7) on the back of the foot. The pain may radiate over the whole distribution of the nerve, but it is often so distinctly limited to the course of the trunk and branches, that the patient points these out with exactness, when he indicates the course of the pain. It may be dull or acute, and is often burning in character. It may seem to dart downward, often starting from the highest point behind the iliac bone. As the pain on movement increases, the nerve trunk becomes extremely tender to pressure, especially along the back of the thigh. Even before the tenderness becomes considerable, it may often be elicited in the following manner. Let the patient sit on a chair with the knee at a little more than a right angle. If the finger is then pressed into the popliteal space, so as to make the nerve a little more tense, a pain is felt, shooting up the course of the nerve at the back of the thigh. This is a very useful test of sensitiveness of the nerve, and is of diagnostic value in slight cases. Sometimes there is great pain behind the hip, and radiating pains in the distribution of the nerve, but very little tenderness of the trunk; in these cases the chief disease is within the pelvis. It is not common for tender points to be developed in the periphery.

Abnormal sensations other than pain are often felt in the area of distribution of the nerve, tingling, formication, and the like, and in severe cases there may be irregular spots of anæsthesia on the back of the thigh, on the leg, or the foot. The affection of sensibility at the back of the thigh indicates that the disease extends up the nerve, above the sciatic notch, to the origin of the small sciatic, or that this is involved in a simultaneous neuritis. In severe cases the muscles supplied by the nerve become flabby, tender to the touch, and sometimes distinctly weak and wasted. This is chiefly noticeable in the calf muscles. There may be slight alteration in the electrical irritability, but this amounts to a distinct degenerative reaction only in very severe cases. Slight fever and corresponding constitutional symptoms may attend an onset that is acute, or an inflammation that is intense. Chronic cases, however, are usually not attended by elevation of temperature.

The duration and severity of the affection are extremely variable. It may be trifling in degree, causing pain on movement only, and this may pass away in the course of a few weeks. On the other hand, the spontaneous pain may be so continuous and intense that sleep can be obtained only by the help of narcotics, and the disease may last for many months, and even for a year. Improvement is shown by the subsidence of spontaneous pain, followed by the slow diminution of the pain on movement, and of the tenderness of the nerve. The muscular wasting, that occurs in severe cases, may last long after the active stage of the disease in the nerve, and fibrillary contractions in the muscles that have been affected may continue for years, and are sometimes accompanied by a tendency to painful cramp. The disease is prone to relapse and still more prone to recur after recovery. A second attack may occur in the same or in the other leg, but both legs are scarcely ever affected at the same time.

The cases of secondary sciatica depending on disease outside the nerve, compressing or irritating it, differ, in some respects, from the primary form. Pain is felt less in the nerve trunk than in its distribution. The difference is not absolute; it is one of degree. There is rarely, however, the tenderness of the nerve that is so common in primary sciatica. The course of the secondary cases is also different, and depends on the course of the original disease.

Complications of sciatica are rare, but one or two deserve mention. Cutaneous eruptions, usually herpetic in character, have been occasionally met with. These sometimes heal slowly, but in themselves they are unimportant. A graver complication, but happily a rare one, is a tendency of the mischief to ascend the nerve. Thus symptoms may spread from the region of the sciatic to that of the lumbar plexus; the pain may spread to the front of the thigh, and the extensors of the knee may become flabby and weak. This is probably due to the passage of an ascending neuritis up the lumbo-sacral cord. In extremely rare cases, the morbid process has apparently reached the spinal cord, and indications of cord disease have thus succeeded those of a primary and apparently simple sciatica.

PATHOLOGY.—The nature of the disease has been already indicated. The evidence that the disease is a neuritis, and not a neuralgia, is chiefly threefold. The conditions under which the disease occurs are very different from those that attend unquestionable neuralgia in other situations. This has been remarked with surprise, even by those who regard it as a neuralgia, but who have failed to see the significance of the difference. Secondly, the neuritic nature of the affection is clearly indicated by the facts of pathological anatomy, scanty though these are. Thirdly, the symptoms in severe cases are proof of the existence of neuritis, the wasting of the muscles and the anæsthesia indicate structural damage to the nerve fibres, and preclude any other explanation. There is every gradation between these severe forms and those which are slight, and the symptoms in the latter are identical with those of the early stage of the severe cases.

The symptoms find their explanation in the character of the morbid process. The pain in the nerve and its tenderness must be due to irritation of the nervi nervorum. The pain referred to the distal portions of the nerve is due to the irritation of the proper fibres by the interstitial inflammation, while their greater damage explains the anæsthesia and muscular wasting.

It may seem strange that, when there is irritation of the nerve roots or sacral plexus by pelvic disease, or of the trunk by hip disease, any pain should be felt along the course of the nerve. It might be thought (and has indeed been asserted) that all the pain should be referred to the peripheral distribution of the nerve. The explanation is that the "nervi nervorum" are derived from their own trunk. The nerve itself is part of the area of distribution of its own fibres.

Diagnosis.—The diagnosis of sciatica rests on the position of the pain—on the relation of this to the trunk of the nerve, and to its area of distribution. But the recognition of this relation does not alone suffice for the diagnosis. We have to consider the distinction from other pains in the sciatic area, to discriminate, if we can, sciatic neuritis from sciatic neuralgia, and to decide whether the affection of the nerve is primary, or is secondary to mischief outside it.

Branches of the sciatic nerve and sacral plexus ramify over the hip bone, and pain in sciatica may be felt near the hip joint; hence disease of this joint and sciatica may be confounded. The pain in simple disease of the hip joint does not extend down the back of the thigh in the course of the sciatic, and there is no tenderness of the nerve. The danger of this error is not great, if a careful examination is made, and has been much exaggerated by writers unfamiliar with the characteristic symptoms of true sciatica.

The distinction between a sciatic neuritis and a sciatic neuralgia is sometimes difficult, although less frequently than might be inferred from current accounts of these diseases, in which the history of the neuralgia has been written from the symptoms of the neuritis. If we recognize that all cases of sciatica with persistent tenderness of the nerve are really neuritic, cases of sciatic neuralgia become extremely rare. The two diseases occur usually under different conditions; the subjects of neuralgia have often suffered from neuralgia elsewhere, and are generally weakly and anæmic. The pain is from the first spontaneous; posture has little influence upon it; movement is not itself painful, although it may excite paroxysms of pain. The pain is referred to the branches and distribution of the nerve rather than to its trunk, and tenderness of the nerve, if it exists, is altogether subordinate to the spontaneous pain.

Secondary sciatica is usually produced by disease of bone about the hip joint, or of the joint itself, or by disease in the pelvis. In the former case, a careful examination (never to be omitted in any case of sciatica) at once reveals the mischief. When the disease is within the pelvis, the tenderness of the trunk of the nerve is slight in proportion to the pain, and this circumstance should always lead to a careful search for any indication of pelvic mischief. In any case of doubt, a rectal examination should be made.

Sciatic pain occurs in some diseases of the bones of the spine, in lesions of the cauda equina, and occasionally in disease of the spinal cord itself. In these cases we have no tenderness of the nerve; the pain is chiefly peripheral, and very often bilateral. Double true sciatica is almost unknown, and bilateral pain should always suggest disease of the nerve roots (see p. 102). The pains of locomotor ataxy are often felt in the sciatic area, and occasionally follow the course of the sciatic nerve, but their wider range, their fugitive character, and their association with other symptoms of tabes, should prevent an error.

Prognosis.—The prognosis of sciatica not dependent on disease outside the nerve, is always good as regards ultimate recovery. As a general rule, the probable duration of the disease is proportioned to the severity of the symptoms. The practicability of adequate rest is an important element in the prognosis. Irritating exertion may lengthen the duration of the disease by many months.

TREATMENT.—The principles of the treatment of sciatica are those of neuritis already described, and only the points of special importance need be here repeated. In all cases rest to the limb is of the utmost importance, and its urgency is proportioned to the severity of the symptoms. A slight attack may pass away, in spite of a moderate amount of standing and walking, but many slight cases are converted into severe ones by unwise exertion. All postures and all movements which increase pain should be avoided as far as possible. The same principle applies to mechanical compression of the nerve by hard seats, and by strong contractions of the flexors of the knee. In more positive treatment, the possible causes of the disease must be remembered. In gouty cases saline purgatives are often of signal service, and are distinctly useful in preventing attacks in those who are liable. In the acute stage of a severe attack, hot linseed-meal poultices should be applied along the course of the nerve, and small doses of mercury may be given with advantage. Counter-irritation is of great value, and cannot be employed too early. A commencing attack may often be cut short in a few days by rest, and a series of mustard plasters or small blisters applied over the seat of pain, as this changes under their influence, chasing it, as it were, from one spot to another, until it disappears. Spontaneous pain can only be relieved by sedatives. Morphia is the surest, but often an injection of half a grain of cocain relieves the pain as effectually, and is far less objectionable. Cocain must be injected at the seat of the pain. Its influence in many cases is great and more prolonged than might be anticipated; apparently the arrest of impressions from the periphery facilitates the subsidence of the morbid irritation. The injection of morphia may be made, with advantage, over the inflamed part of the nerve, so as to combine slight counter-irritation with the sedative influence. Simple acupuncture along the course of the nerve has been recommended; it gives temporary relief to the sciatic pain, as does any superficial pain, however produced, but the cases are very few in which it has a permanent effect. Sedative liniments and ointments may also be applied along the course of the nerve; the most useful are belladonna liniment mixed with an equal part of chloroform liniment, and aconite ointment, rubbed in until distinct tingling is produced. Electricity is chiefly useful in the later stages; its method of use has been described in the account of the treatment of neuritis. In very obstinate cases, nerve stretching has done good; sometimes, perhaps, by releasing the nerve from adhesions, but probably more often by effecting an energetic counter-irritation, and enforcing a beneficial rest.

MULTIPLE NEURITIS.

The term "multiple neuritis," or "polyneuritis," is applied to the condition in which many nerves are inflamed simultaneously or in rapid succession. It has also been called "disseminated neuritis," but this is an inappropriate name, because a single nerve may be the seat of disseminated inflammation. The discovery that certain combinations of symptoms formerly thought to depend on disease of the spinal cord, are really due to disease of the peripheral nerves, is one of the most important steps in the recent advance of neurology. It has profoundly modified many of our conceptions, not only of the processes of disease, but of the range of action of certain morbid influences.

The occurrence of multiple neuritis was first demonstrated by Duménil, of Rouen (1864), although the leprous form had been previously described by Virchow. Graves, indeed, long ago suspected that many cases of paralysis were due to disease of the nerves, but he based his opinion on the normal aspect of the spinal cord; and in most of the cases he describes it is probable that modern methods of examination would have revealed central disease. Duménil's observations attracted little notice, and it was not until fresh facts were brought forward by Joffroy (1879), Leyden (1880), and Grainger Stewart (1881), that attention was generally directed to the subject. During the last five years a large number of observations have been published, especially by Eichorst, Eisenlohr, Pitres, and Schulz.*

Multiple neuritis presents the same varieties that have been described in the general account of inflammation of the nerves. The morbid change may be interstitial, or parenchymatous and degenerative, or both. The pathological forms that occur cannot yet be adequately defined, but five chief classes may be distinguished. (1) Diphtheritic neuritis; acute parenchymatous degeneration of the nerves occurs after diphtheria. The changes are not, however, confined to the nerves, and the condition is considered in a later part of this volume. (2) Tabetic neuritis; in locomotor ataxy degenerative changes are common in the peripheral nerves; they are usually very chronic, and it is perhaps doubtful whether it is correct to regard them as inflammatory, although they are identical in character with forms which, when acute, are commonly so regarded. Except in their relation to some other forms of neuritis, the tabetic changes will not be considered in this section. (3) Leprous neuritis; the slow interstitial overgrowth of connective tissue which occurs in anæsthetic leprosy. (4) Endemic neuritis, such as occurs in the curious and terrible endemic disease of Japan called kak-ké. This form appears to be due to endemic influences, and it is probable that some other cases of neuritis may be due to a similar cause. (5) We must at present place together the other forms of multiple neuritis common in this country. They differ somewhat in pathological characters, in the acuteness of their course, in the symptoms they produce, and the causes to which they are due. Nevertheless, no constant varieties have yet been ascertained. This group will probably ultimately be subdivided; at present we can only distinguish the tendencies of its varieties and the difference of its causes. In the following account the statements made apply to this group generally;

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* A useful summary of our knowledge, with many original cases, will be found in Dr. Buzzard's Harveian Lectures, published in the *Lancet* for November and December, 1885.

endemic and leprous neuritis will be separately described at the end of the section.

Etiology.—Primary multiple neuritis affects both sexes, but, in this country at least, females are more liable to suffer than males. The disease is one of adult life, and is scarcely ever met with until the age of twenty-five is passed. It is most common between thirty and fifty, but has been met with as late as sixty. Its most frequent cause, beyond all question, is chronic alcoholism. This lesion is the common cause of alcoholic paralysis. For some reason, at present unknown, females suffer from this cause far more frequently than males. I have notes, for instance, of eight cases of characteristic alcoholic neuritis in females, and not of one in a male. The fact is remarkable considering that male drinkers are certainly more numerous than female drinkers. Apart from alcoholism, the disease is probably more common in males, but the influence of this cause is so great as to determine a preponderance of females when all cases are taken together. In cases in which this cause is not operative others can sometimes be traced; exposure to cold, extreme and long-continued fatigue, exhaustion after other maladies, acute and chronic. In a few instances the symptoms have followed acute or subacute articular rheumatism, but the disease has sometimes been ascribed to rheumatism on insufficient grounds, since rheumatoid pains, referred in part to the joints, are often an early symptom of neuritis. Nevertheless, true rheumatism has been an apparent cause in some cases, and, as we have already seen, single nerves may be affected from the same influence. Many sufferers from multiple neuritis have died from phthisis, but the nature of the association of the two diseases is obscure.

It is probable that some blood states allied to septicæmia may cause multiple neuritis. Thus, a man, thirteen days after a stab wound in the abdomen, which healed well, had an attack of parotitis with facial palsy; on the fortieth day there developed paralysis of the tongue, vocal cords, and limbs, and on the sixth day after the onset of these symptoms he died, from respiratory palsy. Extensive peripheral neuritis was the only nerve lesion.* In some instances more than one cause is operative; exhaustion and exposure to cold may concur with alcoholism.

Pathological Anatomy.—The changes in the nerves are those already described in the account of neuritis. They vary, however, very much in different cases. In some, the inflammation chiefly affects the sheath and interstitial tissue; in other cases both the connective tissue and nerve fibres are involved; as in Fig. 39, p. 74. This was the condition in the case of presumably septicæmic neuritis mentioned above. In others again, the fibres are affected chiefly or exclusively; they present the degeneration which when acute is regarded as indicative of parenchymatous inflammation. We cannot, at present, connect these pathological varieties with any differences in causation. The more acute the case, the more probable is it that the connective tissue as well as the nerve fibres will be involved.

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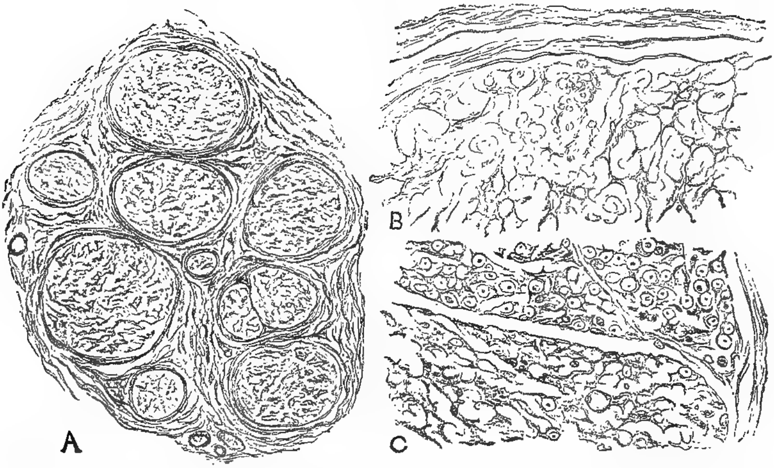
* Roth, *Correspondzbl. f. Schweizer Aerzte, 1883*, No. 13.

Naked-eye changes are present only when the connective tissue and sheath are inflamed in considerable degree. In recent and acute cases of this character, the nerves may be found reddened, swollen, and sometimes small hemorrhages are visible. In older cases they may or may not be swollen, but are usually softened, and even pulpy. When the changes are confined to the nerve fibres, diminished consistence may be the only alteration that can be recognized with the unaided eye. In the former cases, the microscope shows the sheath to be infiltrated with lymphoid cells (probably leucocytes), and in old cases many spindle cells and fibres increase its bulk. The same changes may be traced in the septa between the fasciculi, and in the secondary sheaths that surround the latter. The walls of the vessels are also thickened (Fig. 52, A). In recent cases, the small vessels may be surrounded by leucocytes. The fibres themselves are always damaged in some degree, and as in the case from which Figs. 51 and 52 are drawn, they may suffer chiefly, the connective tissue presenting comparatively little change. In this case, the patient was a woman, aged 33, alcoholic; the course of the disease was subacute; the symptoms reached a considerable degree in a few weeks, and the patient died, chiefly from coincident liver disease, five months after the onset. The symptoms were characteristic, and continued up to the time of death. The condition found after death is a good illustration of the parenchymatous or degenerative form, but in all forms of neuritis the process in the nerve fibres is the same, whether the connective tissue is or is not much affected. The fibres undergo acute degeneration, essentially the same as that which occurs after an injury to a nerve. It has been represented in Figs. 28 (p. 61) and 29 (p. 63), and, associated with inflammation of the sheath, in Fig. 39. Examination in the recent state reveals abundant products of the degeneration of the fibres,

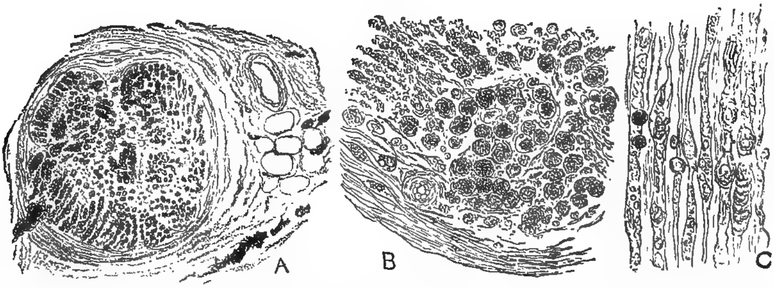
FIG. 51.



Multiple alcoholic neuritis: sections stained with carmine and cleared. A. Transverse section of part of sciatic nerve; low power. B. Part of a fasciculus of same more highly magnified. C. Part of a less affected fasciculus from a musculo-spiral nerve.

granule corpuscles, etc. After hardening, a transverse section, stained with carmine, and cleared in the usual way, presents the appearance shown in Fig. 51, A. There is a slight increase in the connective tissue between the fasciculi; the area of these is occupied by tracts of connective tissue, which under a higher magnifying power (B) have a branching form, enclosing spaces which have been occupied by nerve fibres, and in some of which healthy fibres still remain (C). The space between this tissue is occupied by the products of degeneration of the fibres, but these are rendered invisible by the clearing process, and only faint indications of their outline can be seen (B). In sections stained with osmic acid and mounted in glycerine (Fig. 52) these products of degeneration, stained dark, are conspicuous. Under a low magnifying power (A), a fasciculus is seen to be studded with black granular spots, elongated where the fibres are divided obliquely; the increase in the tissue between these degenerated fibres is distinct; aggregations of myelin are seen in the connective tissue between the fasciculi. Under higher powers (B), the dark spots are seen to be the sections of degenerated nerve fibres, in some places two or more being blended into a larger mass. In a longitudinal section (C), the process of degeneration of the fibres is more distinctly seen, and from it the appearance of the transverse section can be better understood. The white substance is broken up and enlarges the fibre at some places, while at others it has been removed, and the sheath is left empty. Round masses of myelin lie (on the left) outside the fibres. The nuclei of the fibres are enlarged; one, of considerable size, is seen in the centre of the figure. Connective-tissue fibres and cells lie between the nerve elements, and near the sheath are round and spindle cells containing myelin granules and globules which they have taken up (B).

FIG. 52.



Sections from the same sciatic nerve, stained with osmic acid and mounted in glycerine. A. Fasciculus under low power. B. Part of a fasciculus more highly magnified. C. Longitudinal section of nerve fibres. (*From sections prepared by Dr. Beevor.*)

The distribution of the changes in the nerves varies much in different cases. The signs of inflammation of the sheath may exist only on the larger and medium-sized nerves of the limbs, and in the smaller nerves the changes may be confined to the nerve fibres. These may be traced down into the intra-muscular nerves and their endings. In some of these cases, the latter may be secondarily degenerated. On the larger nerves, the signs of inflammation of the sheath are most intense at certain spots, especially (as in simple neuritis) where a nerve turns round a bone or passes through a fascia, or divides. When the trunks are traced upward, the evidence of inflammation of the sheath, as well as the change in the fibres, becomes slighter, and in all forms the anterior roots are usually normal.

In cases in which the affection is chiefly degenerative, the changes are always intense in the peripheral parts of the nerves, and usually lessen when the larger trunks are reached, becoming progressively slighter, and the proportion of normal fibres larger, until the morbid appearance ceases. The centripetal extent of the disease, the degree in which the larger trunks are affected, varies according to the duration and severity of the case. In the case from which the illustrations are taken, even the sciatic nerve presented few healthy fibres. But, as usual, the anterior roots were healthy.

The nerves of the limbs alone suffer in the majority of cases. The corresponding nerves on the two sides are usually affected. The musculo-spiral nerve in the arm and the anterior tibial nerve in the leg usually suffer first and in greatest degree, but often all the nerves of the limbs are involved. The nerves of the trunk are affected only in severe cases. Very rarely the phrenic, pneumogastric, facial and hypoglossal have been inflamed. The other cranial nerves escape.

The *muscles* present changes of the same character as those which result from simple degeneration (see p. 64). They are paler than normal and smaller in bulk. The fibres are reduced in size, and pale; the transverse striation may be preserved, or they may be granular; normal fibres may be found side by side with those that are degenerated. The nuclei of their sheaths may be increased in number, and granular, and pigment masses may accumulate between the fibres.

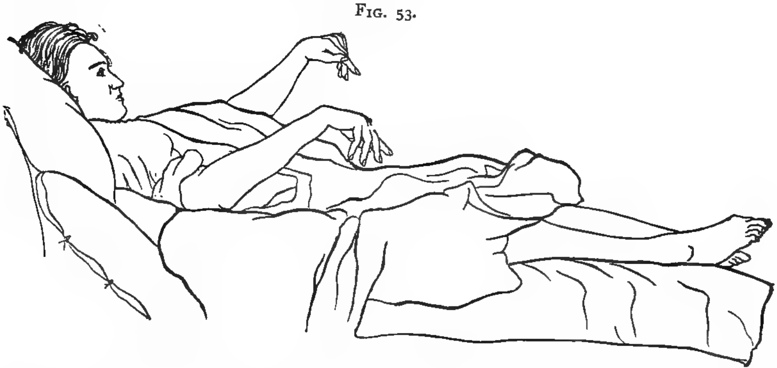
The *spinal cord* has been found healthy in most cases. The ganglion cells, in some instances, have presented abnormal appearances, a vitreous appearance or vacuolation, and this when the anterior nerve roots were not diseased. Now and then more considerable changes, usually those of chronic myelitis, have been found in the cord. They are probably merely coincident.

Morbid appearances in other organs are common in the cases of alcoholic origin. The brain may present signs of chronic meningitis. The liver is frequently, and the kidneys are occasionally, found diseased.

SYMPTOMS.—The symptoms of multiple neuritis resemble in their characters those produced by inflammation of single nerves, differing, however, in their wide range and special distribution. The manifestations of the disease are, in most cases, very characteristic, in spite of the fact that the extent of the symptoms obscures that relation to individual nerve areas which constitutes so important a feature of the inflammation of single nerves. The early and dominant symptoms are those of motor palsy and sensory irritation. The distribution of the palsy is especially determined by the tendency of the musculo-spiral and anterior tibial nerves to suffer most. In consequence of this there is a symmetrical paralysis of the extensors of the hand and flexors of the foot, with resulting wrist-drop and foot-drop (Fig. 53). The sensory irritation causes pain and tenderness, usually localized in the nerve trunks. The progress of the disease is attended by more profound indications of nerve damage, wasting of muscles and impairment of sensation in the distribution of the most affected nerve trunks.

The onset of the symptoms may be acute or subacute, but is rarely so slow as to deserve the name of chronic. An acute onset may be attended by considerable constitutional disturbance, rigors, fever, etc., and this may last for from one to three or four weeks. The initial pyrexia may amount to 103° or 104°. The first definite symptom is usually a sensation of tingling, or "pins and needles," in the extremities, chiefly in the fingers and toes. It may or may not be preceded by vague rheumatoid pains, but to it pain is soon added. This is commonly at first dull in character, but speedily increases to a considerable degree, and becomes more acute—sharp, darting or burning in character. It may be felt along the course of the nerve trunks or in the area of their distribution, and is sometimes especially severe in the fingers and toes. It is often increased by movement. Occasionally, in very chronic cases, the amount of pain is trifling. With the pain there develops considerable tenderness of the limbs, increased sensitiveness of the skin and deeper structures. The nerve trunks usually become extremely tender, and sometimes the more accessible nerves, such as the ulnar, may be felt to be distinctly swollen. The muscles become very sensitive to pressure, whether they are the seat of spontaneous pain or not. This tenderness of the muscles is even more constant than that of the nerve trunks. Muscular weakness is soon added to the symptoms of sensory irritation, although for a time it may be masked by the interference with movement caused by the pain. The loss of power usually begins in the arms as deficient power of extension of the wrist and fingers. Both arms are affected together or in quick succession. The palsy resembles that which results from lead poisoning, but the supinator longus is usually involved. With, or soon after, extension of the hand, the power of flexion of the

Fig. 53.



Multiple alcoholic neuritis; palsy of extensors of wrist and flexors of ankle. (*From a photograph by Mr. Hyde Marriott, B. Sc.*)

ankle and extension of the toes becomes impaired. Sometimes the legs suffer before the arms, and occasionally the symptoms are confined to the lower limbs. The flexors of the hand and extensors of the foot suffer less than their opponents, but in cases of greater severity they also become weak. In these cases, also, the muscles moving the elbow and knee, and even those of the shoulder and hip, become feeble, but lose all power only in very severe cases, and in these the trunk muscles may be involved. The diaphragm may be paralyzed; constant frequency of pulse and paralysis of the vocal cords may indicate a neuritis of the vagus (Roth, Vierordt); and the tongue and face may become affected (Strube). The affected muscles soon become flabby and waste. The nerves lose their electrical excitability, and the muscles present, in most cases, the reaction of degeneration. The increase of voltaic irritability may be slight in the cases of chronic course, and even in acute cases the early increase gives place to depression much sooner than in cases of focal neuritis or of infantile paralysis. It is often very difficult to make a satisfactory electrical examination, on account of the extreme sensitiveness of the limbs. Besides motor weakness, there is sometimes distinct incoördination of movement, which may closely resemble that of locomotor ataxy.

The hyperæsthesia of the skin and deeper structures usually continues for a long time, often through the entire course of the disease. It is extensive in most cases, but has been known to be limited to the extremities, and even to the palms and soles. As the nerve fibres suffer in greater degree, the sensibility of the skin is usually impaired, that to touch first and chiefly. Increased sensitiveness to pain often coexists with anæsthesia. The loss of sensation is always greatest on the extremities, but may extend up the limbs, first along the outer side of the leg, and the radial side of the forearm. It is rare for sensibility to pain to be lost, but its conduction has been delayed in some cases.

Reflex action in the affected limbs is always lost. The myotatic irritability disappears early. The knee jerk may be lost even before the weakness of the extensors of the knee has reached an appreciable degree. Trophic changes in the skin, nails, and hairs are common, similar to those met with in ordinary neuritis. Œdema of the limbs is frequent; probably, in alcoholic cases, the blood state conduces to its occurrence. In one case the œdema existed only over the nerve trunks, in the neighborhood of joints (Leyden). Effusion into joints has been observed, but there is some doubt whether this was the consequence of the neuritis, or was rheumatic and primary. Slower changes in the nutrition of the joints are common, and may lead to adhesions. The sphincters are affected only when there is also disease of the spinal cord.

In rare cases the motor weakness is very slight, the sensory symptoms exist chiefly in the periphery, there is little tenderness of nerve trunks, and the incoördination, already described as occasionally present, is a conspicuous symptom. Such cases present a close resemblance to the symptoms of locomotor ataxy, and the condition has been termed "alcoholic pseudo-tabes,"* or "alcoholic ataxia."†

The *course* of the affection varies according to its acuteness and severity. In cases of moderate degree, the symptoms increase during four or six weeks, then become stationary, and, after one or two months, slowly improve. The pains lessen, but the hyperæsthesia may continue for some time after the pains have ceased. Power slowly returns, first in the muscles affected last and least, and afterward in those paralyzed and wasted in greater degree. In these the weakness lasts for many months, and their opponents are apt to become shortened. The contracture of the calf muscles, secondary to palsy of the flexors of the ankle, constitutes a grave hindrance to the use of the legs. The palsy of the arms usually lasts longer than that of the legs. Often, for instance, at the end of six or eight months, the legs have recovered and the extensors of the wrist and fingers are still feeble. Some times power returns first in the arms. Improvement goes on for a long time, but occasionally there is some permanent loss of power. It is remarkable, however, how much improvement may take place, even after the paralysis has been considerable for a year. Relapses are apt to occur, but only when the cause of the neuritis is still in operation.

On the other hand, the disease may continue to increase for several months, or may develop with such acuteness and intensity that at the end of ten days or a fortnight there is complete paralysis of the limbs; the intercostals share the weakness of the muscles of the trunk, and, if the diaphragm also becomes paralyzed, death may occur from the failure of breathing power. Such cases have been known to result in death at the end of six and ten days. In other, slightly less intense, cases the patient has lost all power of movement, and has died at the end of a few weeks or months, worn out by continued pain and bedsores, or suffocated by a trifling bronchitis in consequence of the partial palsy of the respiratory muscles. In many cases, however, the immediate cause of death has been associated disease, either phthisis, or some other consequence of alcoholism, especially disease of the liver. I have twice known rapid and unexpected cardiac failure to cause death in alcoholic cases. It may have been paralytic in nature, but as each patient had recovered from the graver symptoms, it is more likely to be due to degeneration of the muscular tissue of the heart.

Of *complications*, the most important, beyond those that have just been named, is mental derangement. This is confined to the alcoholic cases. It may be acute or subacute, and consists in failure of mental power, often accompanied by delusions. It is ultimately recovered from, if the patient's habits can be reformed. When there is no pronounced mental derangement, there is often in women a marked incapacity for endurance, a tendency to exaggerate suffering, and an intolerance of remedial measures, which increases very much the difficulty of dealing with these cases.

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* Krucke, *Deut. med. Zeitung*, *1884*, No. 72.

† Dreschfeld, *Brain*, July, 1884, p. 201.

Pathology.—The changes found in the nerves afford a complete explanation of all the symptoms of the disease. The ataxy observed in some cases may be due to damage to the sensory muscle nerves. The relative affection of the nerve sheath and nerve fibres cannot at present be explained. The relation to alcohol suggests that the neuritis is the result of a direct toxic influence on the nerve elements themselves. It has been indeed assumed that the influence is exerted on special trophic centres in the spinal cord which govern the nutrition of the nerve fibres (Erb), but the existence of such centres is not only not proved, but is improbable. The inconstancy of the slight changes in the ganglion cells of the spinal cord, and the normal state of the anterior roots, preclude the assumption that the intense degeneration of the nerve fibres can be the result of those alterations, and make it probable that these central changes, when present, are an associated effect of the same cause. The severe general symptoms that occasionally attend the onset has suggested the idea that the malady is often of the nature of an acute specific disease. The gradation that exists between these and more chronic forms is an objection to this theory; nevertheless, the hypothesis must not be thrown altogether aside, since multiple neuritis in the Japanese kak-ké and in leprosy seems to form part of a specific endemic malady, and some other blood states may produce it. On other grounds the occurrence of multiple degeneration in the peripheral nerves from the influence of alcohol is a fact of great interest in connection with the pathology of locomotor ataxy. We have seen that the symptoms may closely resemble those of tabes. In this disease the morbid influence acts on the sensory nerves only. It seems probable that some influence may act chiefly on the motor or on the sensory nerves, the incidence being perhaps determined in part by predispositions, at present obscure.

Diagnosis.—The diagnosis of multiple neuritis depends on the combination of motor and sensory symptoms above described, on their localization in the distal parts of the limbs, and on the tenderness of the nerve trunks and of the muscles. The early pains are often mistaken for those of rheumatism, but their seat is not that of rheumatic pain, and their association with tingling in the extremities should excite a suspicion that they are of nerve origin, even before muscular weakness renders their nature clear. The symptoms resemble most closely those of certain diseases of the spinal cord, acute and subacute inflammation of the gray matter, polio-myelitis. Until recently, indeed, all cases of multiple neuritis were looked upon as spinal. In each disease there are a febrile onset, muscular wasting with the reaction of degeneration, initial rheumatic pains, and a tendency to the spontaneous recovery of the least affected parts. The distinction rests on the symmetrical localization of the neuritic palsy, while that of polio-myelitis is characteristically random in distribution; on the persistence and severity of the neuritic pains; on the tenderness of the inflamed nerve trunks; and on the anæsthesia in the area of their distribution, a symptom never present in polio-myelitis. Pachymeningitis, damaging the nerve roots, may cause both palsy, wasting, and anæsthesia, but in this all four limbs are not affected, and the legs rarely suffer; the anæsthesia extends on to the upper part of the limbs and trunk; there is no tenderness of nerve trunks, and there is usually distinct evidence of damage to the spinal cord itself. A mysterious disease, termed "acute ascending paralysis," may resemble the most rapid form of multiple neuritis, but, in it, the symptoms ascend the trunk to the arms, and do not begin in the hands and feet at the same time, as does the usual form of multiple neuritis. Moreover, there is no anæsthesia in the spinal malady. Diphtheritic paralysis, when severe in degree, may also be attended by muscular wasting and anæsthesia, but there is no considerable pain, and weakness in the limbs succeeds that in the palate and ciliary muscle. The distinction of the pseudo-tabetic form from true tabes is very difficult unless muscular weakness is distinct. There is no affection of the internal ocular muscles, no loss of reflex action in the iris, no affection of the sphincters, and no optic nerve atrophy, but these symptoms may be absent in true tabes. A history of alcoholism, with these indications, affords grounds for a presumptive diagnosis, but it must also be remembered that true tabes may occur in an alcoholic subject.

Prognosis.—In the early stage of an acute case there is danger to life in proportion as the upper parts of the limbs and trunk are paralyzed, and the respiratory muscles are thus threatened or involved. When the disease has become stationary, the danger to life is not great, except in the severe cases in which the pains are intense, the patient helpless, and an affection of the sphincters or a girdle pain makes it probable that the mischief has extended to the spinal cord. The prognosis depends, however, as much on the complications as on the disease of the nerves. Organic disease of the liver, kidneys, or lungs, always renders the prognosis grave. The extent to which the habits of the patient can be controlled is an important element. If the danger to life has passed, recovery in the parts least affected may be expected to occur in from three to six months; in those in which the muscular wasting is greatest, not for nine or twelve months, and some permanent weakness may be left. It is remarkable, however, how great and how prolonged a loss of power may ultimately be recovered from.

Treatment.—At the onset of an acute case, the treatment should be that suitable for a local inflammation or a morbid blood state. Absolute rest is of great importance. The excitation of the nerves involved in movement is likely to intensify the morbid process in them. Light nutritious food should be given, and the bowels gently opened. If alcohol has been taken in excess, it must be reduced to a small quantity, or, better still, if practicable, it should be given up altogether. This point in the treatment of alcoholic cases is of extreme importance, and often of extreme difficulty. In the case of every female drinker, deception should be considered not only possible, but probable. Servants are corrupted, and alcohol is often taken in secret, and absolutely denied. When a case does not present the improvement that might reasonably be expected, this influence will generally be found to be at work. Hence it is of great importance that these patients should be under the immediate care of trained nurses, and not of their own servants or relatives. The special treatment of the disease is the same as that for inflammation of single nerves already described. Warm poultices or fomentations should be applied to the limbs, and a daily warm bath, for twenty minutes or half an hour, is often of great service. The general rules already mentioned govern the use of counter-irritation and electricity. A very gentle voltaic current may be applied to those muscles which present indications of nerve degeneration, but not until the most acute stage of the disease is over. Rubbing is very useful, and may be employed almost from the first; in the early period it should be very gentle; afterward attempts should be made, by passive movement, to lessen the tendency to contractures. The extreme sensitiveness of the skin and muscles, and the intolerance of the patients, often render local treatment very difficult. Attention to posture will do much to prevent the occurrence of contractures, but it is often impossible to induce the patient to lie in any other position than that which favors the occurrence of shortening of the muscles. These contractures hinder progress during convalescence. Tenotomy is, however, seldom needed. It is remarkable how much muscular shortening will gradually give way.

During an acute febrile onset, the treatment by drugs should be that of the constitutional state: citrate of potash, nitric ether, and compound tincture of bark are the most suitable, and salicylate of soda is said to do good in the cases which follow exposure to cold. Afterward iron is useful, with quinine and small doses of strychnine or nux vomica. Mercury is not so efficient as it is in cases of simple inflammation of a nerve sheath, and iodide of potassium has not seemed to me to have any special influence on the disease. In chronic cases, or in the chronic stage of acute cases, arsenic may be given with seeming advantage. Cod-liver oil is beneficial in patients whose general nutrition is impaired. The pains may need opium or morphia, by the mouth or injection, but these should be given as sparingly as possible in alcoholic cases. Cocain sometimes relieves local pain, and hypodermic injections of a 2 per cent. solution of carbolic acid have been found, by Caspari, to give relief. Any complications that may be present, in the nerve centres or other organs, must receive appropriate treatment.

Endemic Neuritis.—Multiple neuritis appears, from recent researches, to play the chief part in the production of the nervous symptoms in the terrible malady known as *Kak-kè* in Japan, and as *Beri-beri* in other parts of the East Indies. The inflammation of the nerves involves both the connective-tissue elements and the nerve fibres. Rapid degeneration of the muscles occurs. The spinal cord also presents changes, but these are very variable in degree, and are thought by most observers to be either secondary to those in the nerves or merely coincident. The disease varies much in severity, and in the most acute cases, almost every tissue in the body suffers. The chief nervous symptoms are paralysis, muscular wasting, pains, and "numbness" or "burning" in the skin; all these symptoms being chiefly seated in the legs. In the acute cases, the nervous symptoms are subordinate to those of the general blood state. Some form of malarial poison is probably the agent in the production of the disease. It occurs far more frequently in men than in women, in young adults than in children or the old, and in summer than in winter. It is said to be especially prevalent when the first heat of summer falls on a soil saturated with moisture.

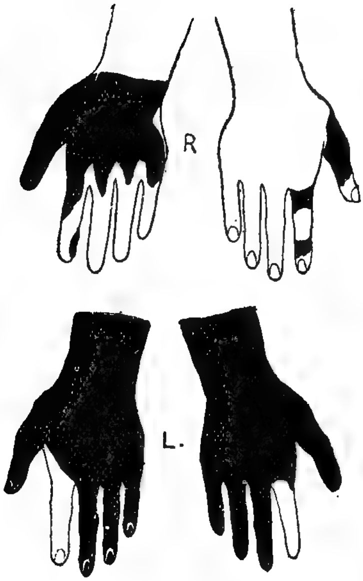
Two cases of paraplegia have come under my notice which were possibly due to malarial neuritis. In each case the disease developed in a fever district, one in India, the other on the East Coast of Africa. The paralysis in each case involved chiefly the muscles in front of the leg, flexors of foot and extensors of toes; these presented well-marked reaction of degeneration; there were pains, but there was no anæsthesia. In each case there was steady improvement under treatment (tonics, electricity, etc.), although secondary contracture in the calf muscles hindered, for a time, the recovery of walking power. The facts that the paralysis was confined to the legs, and that there was no anæsthesia, seem opposed to the opinion that the lesion was neuritis, but these characters obtain also in beri-beri, and it is possible that the neuritis which results from malarial influences may affect chiefly the motor-nerve fibres.

Leprous Neuritis.—Far removed as leprosy may seem to be from the malady last described, both by its general character and extremely chronic course, it is certainly also an endemic disease, and high authorities have sought its cause in malarial and miasmatic influences. The pathology of leprosy has been carried a step further than that of most endemic diseases, since the researches of Carter, Hansen, and Neisser have proved the constant presence of a specific bacillus in all the new formations met with in the disease.

The nervous symptoms of leprosy depend almost exclusively on inflammation of the nerves. They are muscular wasting and anæsthesia, greatest in degree toward the extremities of the limbs. There may be unpleasant tingling sensations, but pains are trifling, perhaps because the process is extremely chronic. Fig. 54 shows the distribution of anæsthesia in the hands in a well-marked example of this disease which came under my observation some years ago. The patient was a creole of Mauritius, who had come to England in early childhood, and presented the first distinct symptoms at twelve years of age—changes in the pigmentation of the skin, anæsthesia, and muscular wasting. They steadily increased during the next four years, the anæsthesia extending each half year from one finger to another. There was also scattered anæsthesia of the legs below the knee. As the figure shows, the loss of sensation does not correspond in area to nerve distribution. This depends on the fact that all the nerves, even those of the fingers, are diseased, but in none are all the nerve fibres commonly destroyed. The muscular wasting was considerable, although only in the small muscles of the hands was it comparable to that of progressive muscular atrophy. The electrical excitability of the muscles was greatly lowered to each current.

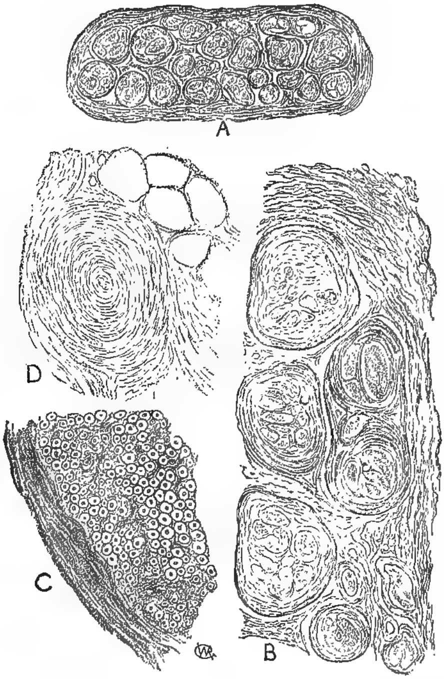
The neuritis of leprosy is typically interstitial (Fig. 55). The primary sheath and the secondary sheaths of the fasciculi are greatly increased in thickness, and consist of nucleated fibrous tissue arranged concentrically (A, B). From the sheath, tracts extend into the interior of each fasciculus (B), isolating the groups of nerve fibres. The increase of tissue even extends between the fibres themselves (C), and these undergo slow wasting; many of the fibres in the figure are seen to be distinctly narrower than normal. The concentric growth and fibrous tissue may even invade the whole area of the fasciculus, all the nerve fibres perishing before it (D). The characteristic bacillus of leprosy is found abundantly, in recent cases, in the new tissue of the nerves. Peculiar cells are met with, infiltrated with the organisms. As the fibrous tissue develops and contracts, the bacilli seem to perish, and ultimately can no longer be discovered. The fact that the connective tissue of the nerves should be thus clearly one of the structures for which an organized virus has a special affinity, is of extreme interest in connection with the action of the varied causes of multiple neuritis.

FIG. 54.



Distribution of anæsthesia in a case of leprosy. The black areas indicate the loss.

FIG. 55.

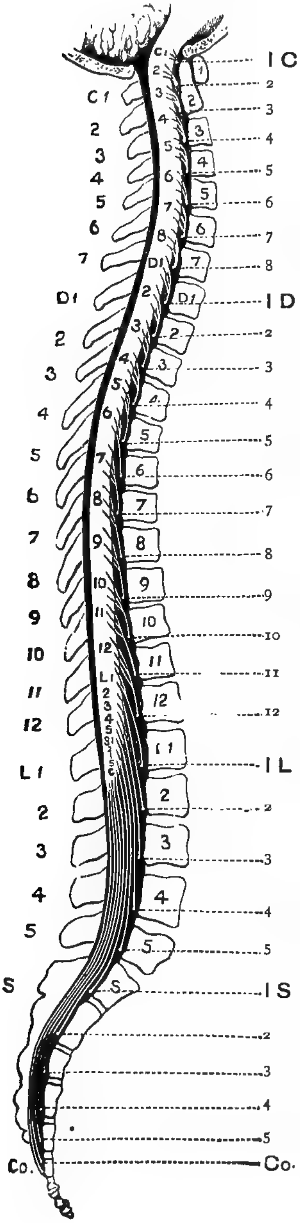


Sections of nerves from a case of anæsthetic leprosy, under the care of Dr. Buzzard. A, median nerve at wrist × 5; B, portion of same more highly magnified; C, part of a less diseased fasciculus from the ulnar nerve; D, a small fasciculus from median in which the concentric fibres have invaded the whole area of the fasciculus.

PART III.

DISEASES OF THE SPINAL CORD.

FIG. 56.



INTRODUCTION.

ANATOMY OF THE SPINAL CORD.

THE spinal cord, it will be remembered, is much shorter than the spinal canal, reaching only to the second lumbar vertebra. Hence the nerve roots descend to their foramina of exit. The lower they arise, the longer is their intraspinal course. All those below the second lumbar pair leave the canal below the lowest portion of the cord. It is convenient to speak of the portion of the spinal cord from which each pair of nerves arises as the corresponding "segment" of the cord. The segments are longest in the dorsal region, and shortest in the lumbar enlargement.

The only part of the spinal column that we can feel are the vertebral spines. These are not all on a level with the bodies of their vertebræ. It is important, therefore, to know the relation of the spines to the bodies of the several vertebræ, and of these to the origin of the nerves. These relations are shown in the accompanying figure (Fig. 56) with sufficient clearness to render a detailed description unnecessary.*

Of the *membranes*, the pia mater closely infests the surface, and is continuous with the tracts of connective tissue that pass within the substance of the spinal cord. It is also prolonged along the nerve roots as their sheaths. The arachnoid forms a much less close investment. The dura mater is not applied to the bones, as it is within the cranium, but a layer of fat and a plexus of large veins intervene between the two. An extension of the dura mater passes along each nerve root, and blends with its sheath.

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* The details of the chief relations are enumerated in my "Diagnosis of Diseases of the Spinal Cord," 3d ed., p. 6.

Structure of the Spinal Cord.—The general form of the cord, the enlargements it presents, and its constitution of white and gray substance, are too well known to need description here. The *white substance* surrounds the gray, except at the two points at which the posterior horns come to the surface (Fig. 57). It consists of medullated nerve fibres, running longitudinally. The posterior cornua isolate the posterior columns from the rest of the white substance. These posterior columns are divided by a median septum of connective tissue, and a little distance from this another incomplete septum (posterior intermediate septum), and corresponding to a depression on the surface, marks off a portion next the median septum, the "postero-median column," or "column of Goll," from the part next the posterior horn, "postero-external column," "column of Burdach," or "posterior root zone." The latter name is given because the fibres of the posterior root pass through it. The rest of the white substance is divided, in the middle line in front, by the anterior median fissure, down which the pia mater and blood vessels pass, and at the bottom of which is the anterior or white commissure. Between the anterior median fissure and the posterior cornu, the white substance is continuous and undivided, extending round the front and side of the cord. It is artificially divided into an anterior and lateral column, the line of division being the outermost of the anterior nerve roots, which pass through the front of the cord, but there is no corresponding distinction of structure or function. The white substance varies in amount in different parts of the cord, and, as a whole, lessens gradually from above downward (see Fig. 58). To this progressive diminution in size certain parts constitute exceptions.

FIG. 57.

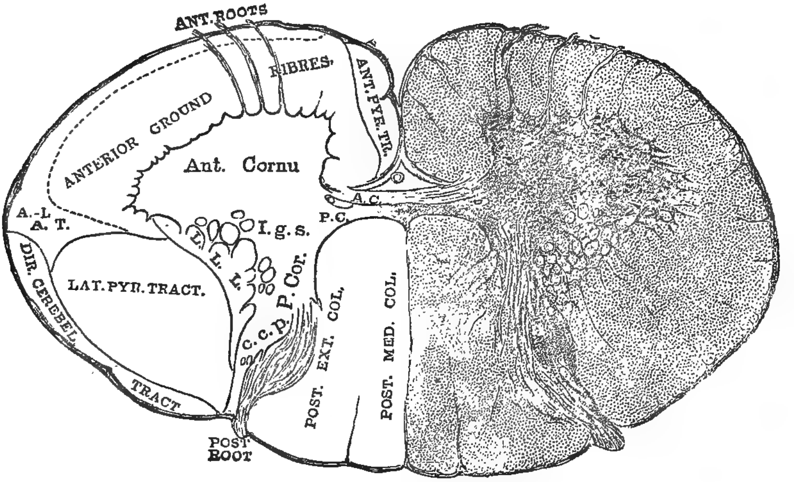


Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate gray substance; P. cor., posterior cornu; C. C. P., caput cornu posterioris; L. L. L., lateral limiting layer; A.-L. A. T., antero-lateral ascending tract, which extends along the periphery of the cord.

The white substance is everywhere composed of medullated nerve fibres, which vary considerably in size. In carmine-stained sections the axis cylinder is seen within each fibre, not always in the centre, and around this are concentric cloudy lines due to the irregular refraction of the white substance. Between the fibres is a peculiar substance, the "neuroglia" or nerve cement, the exact nature of which has been the subject of much investigation and discussion. It appears to consist of an albuminous matrix; embedded in this are fine fibres, which resist acetic acid, and these form a network. At their intersections are peculiar cells consisting of a nucleus and small cell body ("glia cells," "cells of Deiters"). These were formerly regarded as stellate, and are still believed to be so by many authorities, the fibres being looked upon as their processes, but, according to Ranvier, they are flattened cells, without processes. The nerve fibres, thus connected, lie in the meshes of a coarser network, formed by branching processes of finely fibrous connective tissue, proceeding from the pia mater on the outside, and from the gray matter on the inside. In these septa blood vessels run, and some are occupied by nerve fibres that have a horizontal course from one element of the cord to another. These tracts of connective tissue unite the pia mater so closely to the cord that the membrane cannot be readily detached, but in the intervals between the septa, the pia mater and cord are separated and connected by a very narrow layer, similar in structure to that which caps the head of the posterior horn, the so-called "gelatinous gray substance." This also continues the posterior horn to the surface of the cord (Fig. 57). In the dorsal region, where the head of the posterior horn proper is some distance from the surface, this tract of gelatinous substance is of considerable length. It fills up a narrow fissure, which extends inward from the surface.

FIG. 58.

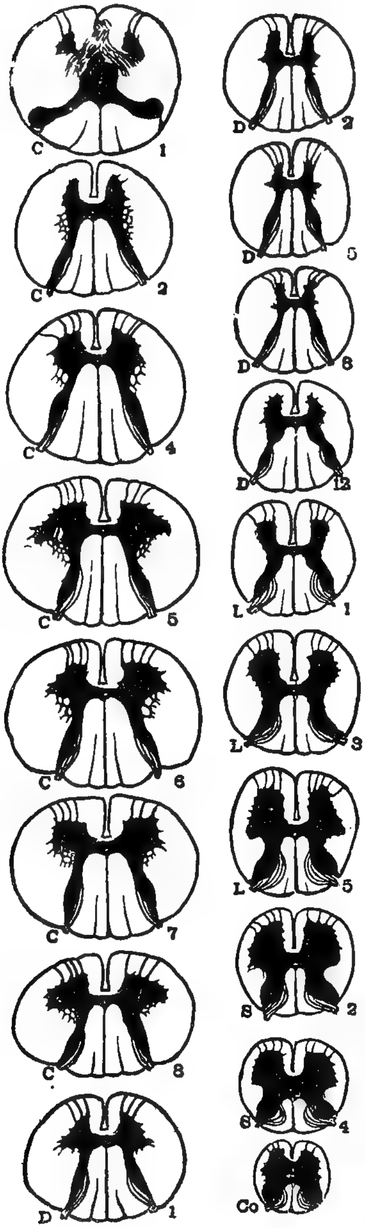


Diagram showing the relative size and shape of the cord and gray matter at different levels.

FIG. 59.

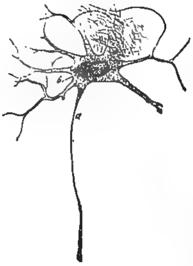


FIG. 60.

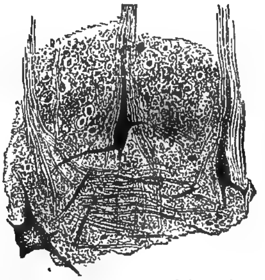
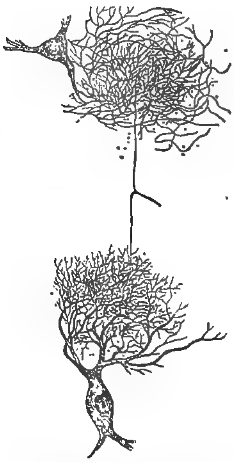


FIG. 59.—A nerve cell from the anterior cornu of the spinal cord of man; *a*, unbranched process becoming the axis cylinder of a nerve fibre; *b*, pigment mass. (*After Gerlach.*)

FIG. 60.—Nerve cells of the anterior cornu sending a process into the anterior root. (*After Henle.*)

FIG. 61.



A fine nerve fibre dividing into two parts, each of which joins the plexus of fibrillæ formed by the branching processes of a nerve cell. From the spinal cord of an ox. (*After Gerlach.*)

The *gray substance* varies in shape and size in different parts of the cord, being largest in the cervical and lumbar swellings, and corresponding to the number and size of the nerve roots given off. The variations in shape and size are indicated in the accompanying figure (58), and will be readily understood by an examination of this. Its division into anterior and posterior horns or cornua is familiar. The part on each side that intervenes between the two horns may be conveniently termed the "intermediate gray substance." It is customary to divide the gray matter into two varieties, "spongy" and "gelatinous." The latter forms a cap on the posterior horns, and is named on account of its naked-eye aspect. The spongy substance, which forms the rest of the two cornua, consists mainly of an excessively fine felty network of nerve fibrillæ, with some cementing neuroglia and small cellular bodies, which vary in size and shape. Many are angular with processes, and are probably of nervous function. Through it course many larger nerve fibres, medullated and non-medullated, and in it lie many nerve cells of various sizes. Those in the anterior cornu are, for the most part, large "ganglion cells," nucleated, with many processes. One process has the aspect of the axis cylinder of a nerve fibre (Fig. 59), and can be sometimes traced into a fibre of an anterior root (Fig. 60). The other fibres divide and subdivide, their ramifications being lost in the sponge-like matrix of the gray substance. Nerve fibres divide and join this ramification (Fig. 61). These cells lie in groups, between and through which course tracts of fibres, chiefly from the anterior roots. Other fibres come from the anterior commissure, white columns, and posterior horn. The ganglion cells are certainly motor in nature, and give origin to at least most, and perhaps all, of the motor fibres of the spinal nerves. Birge, in a careful investigation, found that in each segment of the spinal cord of the frog the number of nerve cells in the anterior cornu corresponds to the number of fibres of the anterior roots arising from that segment.

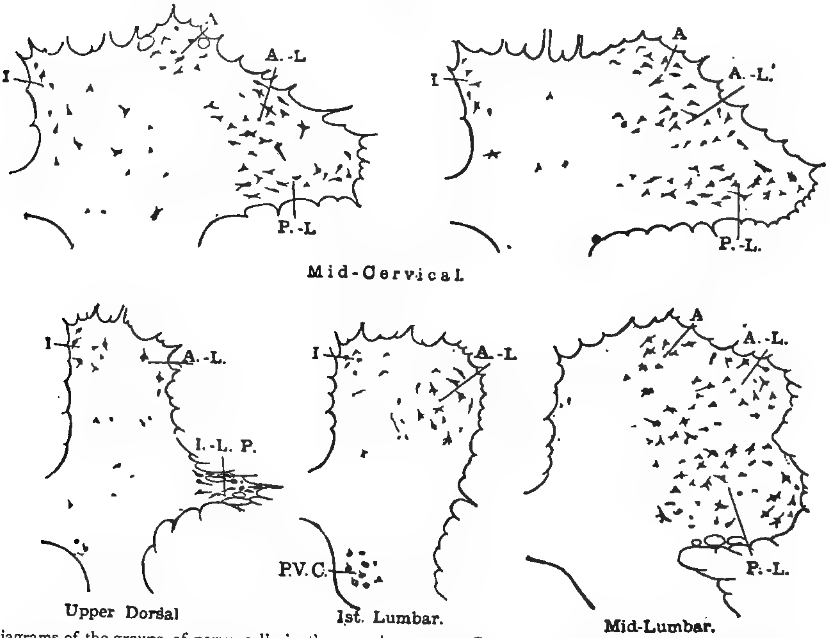
The arrangement of the nerve cells of the anterior cornu is of some practical importance, on account of the frequency with which this part is the seat of limited lesions. They form, as already stated, certain groups, but these groups vary in different regions of the cord, partly under the influence of the shape of the horn, partly irrespective of this, and they even vary in parts of the cord that are near together. They are influenced by the course of the tracts of fibres of the anterior roots, which may pass through a group and break it up into smaller groups, although in a neighboring section it is undivided (see Fig. 62, the cervical groups). Hence very different descriptions have been given of these groups, and the process of distinction has sometimes been carried too far. The groups that can most readily be recognized are the following: In the inner anterior angle of the cornu is a small group, the *inner or medial group*. This is one of the smallest, and is absent in some parts of the cord, especially in the lumbar region. A much larger group lies near the anterior edge of the horn, in the middle, or a little to the outer side of the middle, the *anterior group*. External to this, in the outer extremity of the front part of the horn, is anothor group, the *antero-lateral group*. These two groups are often blended, so that the anterior group cannot be separately distinguished, as in the right hand mid-cervical figure. A third group, usually the largest, lies in the outermost part of the horn, behind its front, usually in the posterior outer angle; it may extend inward, halfway across the horn. It is called the *external* or *postero-lateral group* (P.-L., Fig. 62). These three are the most important groups. There is in some parts also a *middle group*, occupying nearly the centre of the horn. In the small cornu of the dorsal region often no well-defined groups can be made out, but when any can be recognized, they are generally the anterior and external.

Similar cells, usually smaller in size, and isolated, are scattered through the intermediate gray matter, and a group of cells of some size occupies a projection which enters outward into the lateral column in the lower cervical and upper dorsal regions. It was termed by Lockhart Clarke the "*intermedio-lateral tract*," but (on account of other associations with the words "lateral tract") the projection is better designated the "*intermediate process*," and the cells the "*intermediate group*" (I.-L. P., Fig. 62). Isolated ganglion cells, medium and small in size, lie in the posterior horn. These are often fusiform, with many processes, and are supposed to be connected with some of the fibres of the posterior roots. They extend back into the caput cornu posterioris.

In the lower dorsal and upper lumbar cord, from the ninth dorsal to the third lumbar nerves, a group of nerve cells lies in the inner part of the neck of the posterior horn, the "*posterior vesicular tract*" of Lockhart Clarke* (P. V. C., Fig. 62; see also Fig. 71). Most of these cells are fusiform in shape, but are placed vertically, so that they appear round or oval in transverse section. Some, however, present processes that run forward or backward. Nerve fibres pass into and by it, which we shall consider subsequently. Although this tract is chiefly developed in the lower dorsal and upper lumbar cord, a few nerve cells of similar character are met with in the same position in other parts, and are occasionally sufficiently numerous to form a small group.

The "gelatinous" gray matter which forms a cap on the posterior horn, *caput cornu posterioris* (C. C. P., Fig. 57) differs considerably in structure from the rest. It resembles closely the granule layers of the retina in microscopical aspect, and apparently in chemical nature. It has been described as made up of granules, but, according to later researches, consists of a peculiar, translucent material forming an excessively fine network, which behaves to reagents like horny material. Numerous nerve fibres pass through it, from the posterior roots, and these, coursing forward, seem to divide it into columns, as it is viewed in transverse section. Although it is developed from the same embryological elements as the true nerve structures, it has not, according to this view, a nervous function, but is to be regarded rather as a supporting structure, especially developed in connection with the sensory nerve roots. Its extent and position are, it must be admitted, difficult to reconcile with this view of its nature. A few small ganglion cells lie in this gelatinous substance, chiefly on the inner side. Bundles of vertical fibres are also seen in transverse section, and these are often numerous just above the caput, especially on the inner side of the horn.

FIG. 62.



Mid-Cervical.

Upper Dorsal

1st. Lumbar.

Mid-Lumbar.

Diagrams of the groups of nerve cells in the anterior cornu. Groups: I., inner or medial; A., anterior; A.-L., antero-lateral; P.-L., postero-lateral; I. L. P., intermediate lateral process; P. V. C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighboring part of the cord.

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* It is often termed "Clarke's column."

Most of these are fibres of the posterior nerve roots that have a vertical course for a short distance. In Fig. 71 the axis cylinders of these fibres are seen to be changing from the horizontal to the vertical direction, and their relation to the posterior root fibres can be distinctly traced. These fibres are sometimes so abundant that some of them probably belong in part to the postero-external columns (posterior ground fibres). The gelatinous substance contains many vessels, running for the most part vertically.

Fig. 63.

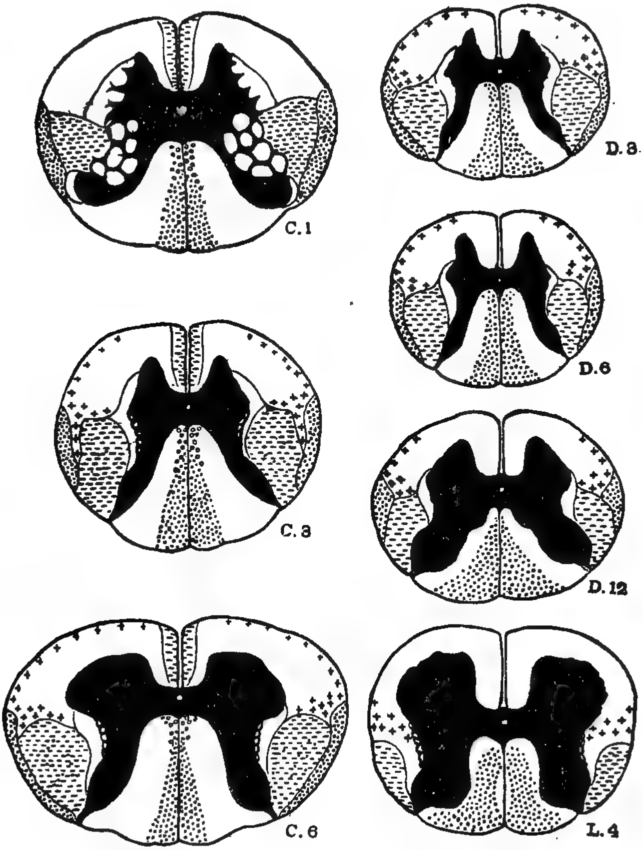


Diagram of the elements of the white substance at various levels of the spinal cord. (*Modified from Flechsig.*) The pyramidal tracts are shaded by short horizontal lines; the direct cerebellar tract by dots; the posterior median column by dots as it degenerates in disease of the lowest part of the cord or of the nerves of the cauda equina; the small circles in the forepart of this column indicate the area which degenerates in disease of the cervical enlargement. The antero-lateral ascending tract is shown by crosses. It is not represented in the first cervical section because there are no facts, at present, to show its exact position here; it probably lies chiefly in front of, and between, the direct cerebellar and pyramidal tracts, as C. 3. The anterior ground fibres, lateral limiting layer, and postero-external column are left white. Fig. 57 should be compared with this.

White Substance.—We may now examine, in greater detail, the constitution of the white columns of the cord, and the probable course and function of the fibres they contain. The analysis has been greatly aided by two facts. The first (discovered by Türck) is that in certain tracts in the white substance the nerve fibres undergo secondary degeneration when separated from their cells of origin, a degeneration analogous to that which, as we have already seen, occurs in the nerve fibres outside the cord. The process is analogous in general character, but we do not know that it is similar in histological nature. As we have seen, the degeneration in the peripheral nerve fibres seems to vary in its character in different cases. Secondly, in the developing cord, the fibres of different tracts acquire their white substance at different periods, and the study of these differences (by Flechsig especially) has not only confirmed, but also extended, the indications afforded by disease. The knowledge we possess is still incomplete. Some facts ascertained have not yet found a practical application, but many others are of the utmost importance on account of the light they throw on the processes of disease.

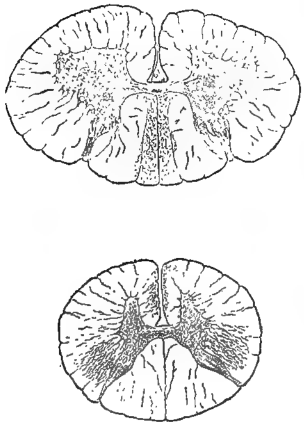
It will facilitate the study of the subject if we consider first those tracts which undergo secondary degeneration, as our knowledge regarding these is the most complete. These degenerations are both ascending and descending. Only one set of fibres degenerates downward, through a considerable extent,—those that continue through the cord the anterior pyramids of the medulla. They commence still higher, in the motor region of the cortex of the cerebral hemisphere, and are the conductors of voluntary impulses. From their relation to the anterior pyramids they are termed the *pyramidal tracts.* They are shaded by short horizontal lines in Fig. 63; compare also Fig. 57.

At the decussation of the pyramids about three-quarters of the fibres usually cross to the other side. These pass down the cord in the lateral column, and constitute the *lateral* (or *crossed*) *pyramidal tract.* Those which do not decussate, pass down their side of the cord, in the inner part of the anterior column, at the side of the anterior median fissure, and constitute the *anterior* (or *direct*) *pyramidal tract.* Flechsig has found that the decussation is subject to many variations. In the majority of cases, the decussating fibres are between 70 and 80 per cent. of the whole. Sometimes they constitute a still larger proportion, and in one case (of 60 examined) all the fibres crossed. Sometimes fewer fibres decussate, only one-half or even less than one-half; in one case 35 per cent., in another only 10. No case has yet been met with in which no fibres crossed.

The *lateral pyramidal tract* occupies the posterior half of the lateral column, outside the posterior cornu. It extends down to the end of the cord, even when it is originally small, and in consequence most pyramidal fibres do not decussate. Hence the fibres that most constantly decussate at the medulla are those for the leg. Through the greater part of the cervical and dorsal regions, this tract is separated from the surface by a narrow layer of fibres, the "*direct cerebellar tract.*" In the upper part of the cervical region (third cervical segment), this tract lies farther forward, so that the pyramidal tract comes up to the surface close to the posterior cornu (Fig. 63, C. 3), and here, if the tract is small, there may be a depression on the surface. In the lower part of the dorsal cord there is a similar movement forward of the cerebellar tract, so that the pyramidal tract again comes in contact with the surface posteriorly (Fig, 63, D. 12), and as the cerebellar tract ceases at the last dorsal nerve, the pyramidal tract extends up to the surface throughout the lumbar enlargement (Fig. 63, L. 4). The inner side of the tract is in contact with the hinder part of the posterior cornu, near the surface, throughout the entire length of the cord, but further forward it is separated from the base of the cornu and intermediate gray substance by a layer of nerve fibres, termed by Flechsig the *lateral limiting layer* (L. L. L., Fig. 57). Among the fibres of the pyramidal tract, in the dorsal and cervical regions, are scattered a few other fibres that belong chiefly to the cerebellar tract. The lateral pyramidal tract diminishes in size from above downward. Its fibres curve inward along the septa that extend from the gray matter into the lateral column, and they enter the gray matter between the anterior and posterior cornua. They pass inward and forward in the anterior cornu, and are then lost in the complicated structure of the gray matter. Their probable termination will be considered presently.

The *anterior* or *direct pyramidal tract* (called also the "column of Türck") descends the cord in the inner part of the anterior column, adjacent to the anterior median fissure. Its exact form and size vary; usually it bounds the fissure on each side; sometimes it forms only a small tract at the posterior part of the fissure; when large, it not only reaches forward to the front of the cord, but causes a prominence on the surface bounded by a distinct sulcus. Its extent down the cord, always less than that of the lateral tract, varies according to its size. It steadily lessens in area, and, if originally small, may not extend beyond the middle of the cervical enlargement; if originally large, it may be traced as far as the lumbar enlargement (Fig. 64); when bearing the usual proportion to the lateral column, it ceases about the middle of the dorsal region. Hence it seems that this tract contains chiefly fibres for the arm, a conclusion which harmonizes with the fact that the lateral tract, even when small in the cervical region, extends down to the lumbar enlargement. But microscopical examination, even of the developing cord, throws no light on the way in which the fibres of this tract end. Flechsig could find no indication of their passage into the anterior commissure; at the same time his observations do not negative such an ending, and it is certain that many fibres of the anterior commissure come from anterior columns and from the neighborhood of this tract. The probability that the fibres decussate in the cord is strong, because most of them seem destined for the arm,

FIG. 64.



Ascending and descending degenerations from myelitis of the mid-dorsal region: the upper section is from the lower cervical, and shows the ascending degeneration of the post.-median column spreading out against the commissure. The other figure is from the lower dorsal, and shows degeneration of the pyramidal tracts, anterior and lateral.

FIG. 65.

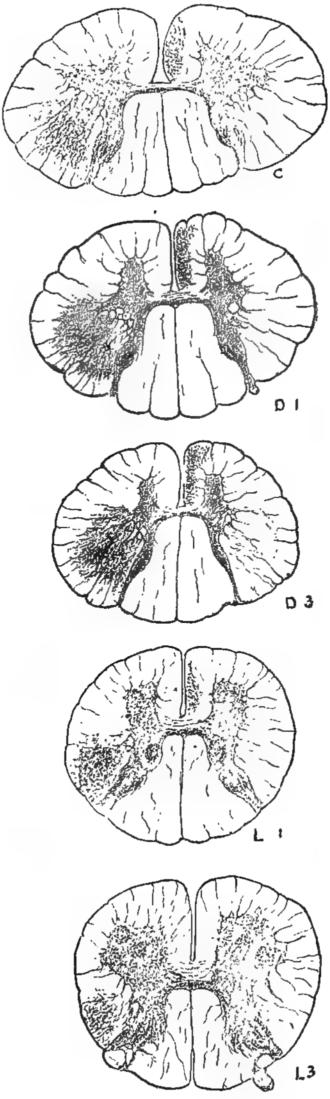


FIG. 66.

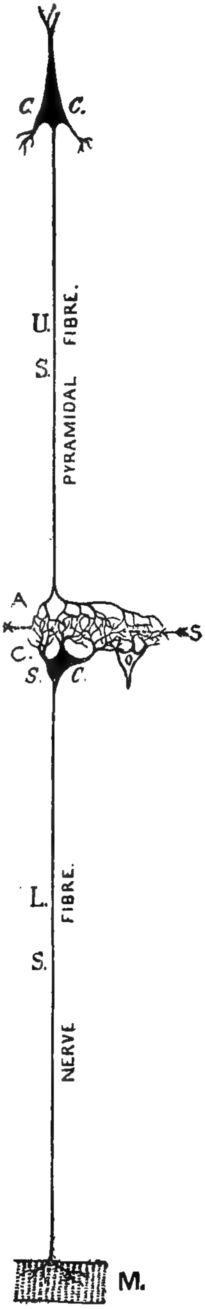


FIG. 65.—Descending degeneration of the pyramidal tracts in a case of hemiplegia from disease of the right cerebral hemisphere. (*From sections prepared by Dr. Mott.*)

FIG. 66.—Diagram of an element of the motor path. U. S., upper segment; L. S., lower segment; *C. C.*, cortical cell; *S. C.*, spinal cell; A. C., anterior cornu; M., muscle; the dotted line indicates the junction of the two segments; S., path from sensory nerve roots.

and the relation of the arm is almost exclusively to the hemisphere of the opposite side. It is therefore probable that most of the fibres pass through the anterior commissure to the opposite gray matter.

Disease of the brain destroying the motor cortex, or the fibres which descend from it to the pyramids, causes descending degeneration, limited to the related pyramidal tracts, anterior on the same side, lateral on the opposite side (Fig. 65). But in some cases slight degeneration has been also found in the lateral tract on the same side, extending into the lumbar region. Hence it is probable that some fibres of each anterior pyramid find their way to the lateral tract on the same side, and descend to the corresponding leg. This degeneration of the lateral tract on the same side as the lesion, is very marked just below the decussation of the pyramids, and therefore cannot be due to any lower decussation in the cord.* We shall afterward see that there is other evidence that each hemisphere of the brain is connected with both legs, although chiefly with that of the opposite side.

How do the fibres of the pyramidal tract end? They seem all to pass into the intermediate gray matter, and forward into the anterior cornu, among the motor nerve cells. There they are lost in the plexus of nerve fibres. There is the strongest indirect evidence that they divide and subdivide, and that their ramifications join the plexus of nerve fibrillæ constituted by the dividing processes of the ganglion cells. The evidence of this is twofold. As far as is at present known, only one process of a ganglion cell becomes an axis cylinder; the others divide. The axis-cylinder process passes into an anterior root. It can be seen to do so in the case of some ganglion cells (Fig. 60), and, as we have seen, the number of ganglion cells and anterior root fibres is the same in the frog, and therefore, presumably, in man. If so, since the pyramidal fibres are certainly connected with the ganglion cells, the connection can only be by the ramifying processes of the cells, and this involves a division of the nerve fibre. The dividing fibre isolated by Gerlach, and shown in Fig. 61, may have been a branch of a pyramidal fibre. The second point in evidence of division is the fact that the nerve cells and root fibres are many times more numerous than the fibres of the pyramidal tract. But all (or at least most) of the motor root fibres may be stimulated through the pyramidal fibres, and therefore the latter must be connected with all (or most) of the ganglion cells. This also involves a terminal ramification.

It is worth while to consider for a moment the whole motor path, from the cortex of the brain to the muscles. We may consider it as composed of two segments, an upper and lower (Fig. 66). Each consists of a ganglion cell above, a nerve fibre, and the terminal ramification of the latter. The upper, "cerebro-spinal," segment consists of the cortical ganglion cell, and the pyramidal fibre which proceeds from the cell, passes through the brain and cord, and ends by dividing in the spongy substance of the anterior cornu. The lower, "spino-muscular," segment consists of the spinal ganglion cell, and the fibre proceeding from this, passing through the anterior root and nerve trunk to the muscle, where it divides and ramifies on the muscular fibre. The elements of the two segments do not probably correspond in number. Each cortical-spinal element is connected with many spinal-muscular segments.† It will be found that this conception of the motor path conduces to clearer ideas of many phenomena of disease.

Fibres degenerate upward in both the lateral and the posterior columns. We may consider the latter first.

The *posterior column*, as we have seen, is divided into two parts by the imperfect "intermediate septum," a narrow "postero-median column," and a wide "postero-external column" (Fig. 57). Secondary degeneration in part confirms this distinction, but shows, at the same time, that the distinction must not be made the ground for a division of the two. If the posterior columns are interrupted anywhere in their course, ascending degeneration results, and this, a short distance above the lesion, is confined to the posterior median column (Figs. 64, 67, 68), in which the degeneration continues upward to the medulla oblongata, and ends there in the gray matter of the "post.-pyramidal nucleus." The median columns degenerate in the same way when the lesion is not in the cord but in the posterior nerve roots of the cauda

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* Pitres, *Arch. de Physiologie, 1874*. Haddon and Sherrington, *Brain*, Jan., 1886. An illustration of this bilateral degeneration will be found in the section on Diseases of the Brain.

† The terminal ramification of the lower segment is a complex "end organ," containing minute cells, and it is possible that the termination of the upper segment is also an analogous structure, containing cellular elements.

Fig. 68.

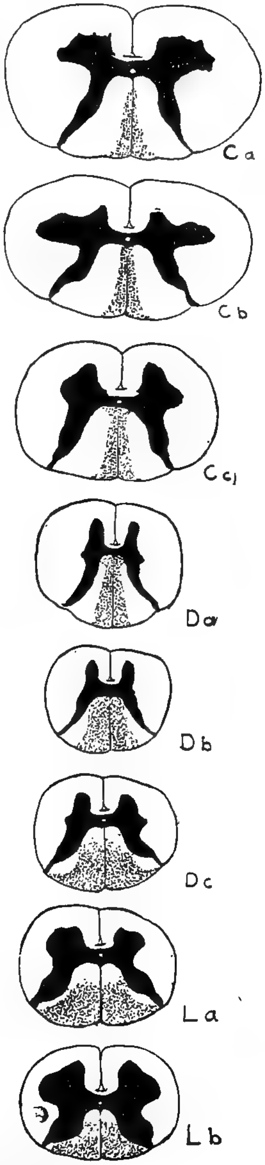

Fig. 69.

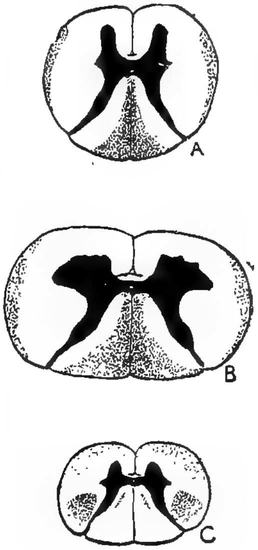

Fig. 67.

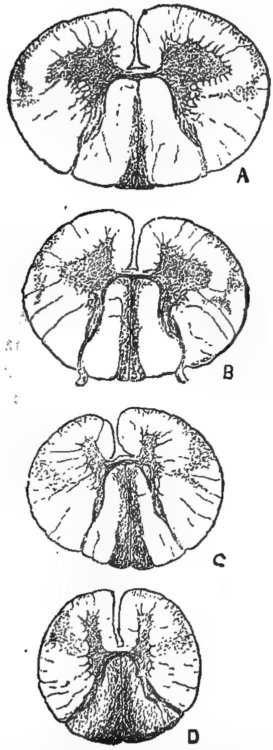

Fig. 70.

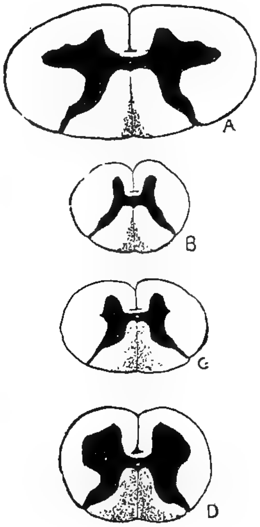

Fig. 67.—Ascending degeneration in the postero-median column and antero-lateral ascending tract. The cord was crushed at the first lumbar segment.

Fig. 68.—Degeneration after injury to the cauda equina. C a, b, c, upper middle and lower cervical; D a, upper; b, c, lower dorsal; L a, upper; and L b, middle lumbar region. (*After Schultze.*)

Fig. 69.—Degenerations after a lesion of the cord in the lower part of the cervical region. A, highest cervical; B, middle cervical 1½ cm. above lesion. Degeneration of the posterior median columns, spreading slightly in A and widely in B into the post. ext. col. The circumferential degeneration is probably composed of the direct cerebellar tract and the antero-lateral ascending tract. C, upper dorsal 2 cm. below lesion. Degeneration of the lateral pyramidal tracts; "comma-shaped" degeneration in front of the post. ext. col.; slight degeneration of the anterior ground fibres.

Fig. 70.—Ascending degeneration after injury to the lowest part of the spinal cord and sciatic nerve roots arising from it. A, cervical; B, lower dorsal; C, junction of dorsal and lumbar regions; D, middle of lumbar enlargement. (*After Schultze.*)

equina (Fig. 68). Hence, it is clear that these columns contain long fibres that are continuous with some of the fibres of the posterior nerve roots.* The contrast between these degenerated columns and the undegenerated post.-external columns, through the greater part of the cord above the lesion (Fig. 67), is very striking, and justifies the distinction between the two. But just above the lesion the degeneration is not confined to the median columns; it spreads out into the post.-external columns, especially toward the posterior surface, and the more extensively, the nearer to the lesion, until close above this the whole extent of the posterior columns is diseased, except a small area close to the posterior cornu, sometimes only in the anterior part. This is true whatever be the seat of the lesion, whether it be in the cervical (Fig. 69), dorsal, or lumbar (Fig. 70) parts of the cord, and if it is in the cauda equina, the degeneration spreads out in the same way in the lumbar enlargement (Fig. 68). This seems to show that all the way up the cord, fibres pass to the post.-median column from the post.-external column, and that these are fibres from the posterior roots is highly probable in the lower part of the cord, and therefore probable also in all parts. As the fibres ascend the cord in the median column, they come to lie posteriorly in proportion as their source is lower. Those from the sciatic nerves, for instance, in the lower cervical cord, only occupy the posterior half of the columns (Fig. 70). Those from the whole lumbar enlargement, or whole cauda equina (Fig. 68), reach forward to the commissure in the lower cervical region, spreading out a little near the commissure, where, however, the degeneration is much less dense than it is behind. Above the middle of the cervical enlargement, the degeneration from a lesion in the dorsal or lumbar regions does not extend forward beyond the junction of the anterior and middle third of these columns, but if the lesion is in the cervical region, the degeneration extends up to the commissure, even at the level of the second cervical segment (Figs. 69, A, 64), showing that the fibres from the cervical roots occupy the anterior portion of these columns.

The root fibres that pass to the post.-median column through the hinder part of the post.-external column apparently do not decussate. This is not, however, the only source of fibres to the post.-median column. Fibres pass to it (1) from the neck of the posterior horn, across the anterior part of the post.-external column, curving backward (Fig. 71, *x*); many of them extend almost to the postero-median septum. They come from the layer of fibres that lies on the inner part of the neck of the cornu, on the inner side of the post.-vesicular column, where this exists. Some of them may be distinctly seen to turn backward in the neck as if they came either from the posterior roots or from cells near the caput cornu. But it is possible that some of them may be fibres from the post. commissure. (2) From the posterior commissure in the middle line. These are very numerous and conspicuous in the lumbar enlargement. They course backward in the septum and then pass outward on each side into the adjacent column. Flechsig believes that these are posterior root fibres. They may easily decussate in the septum, but such decussation has not been proved.

In the lower lumbar region there is a narrow tract of fibres close to the posterior median septum that is apparently of a different nature from the rest of the column. It is lenticular on transverse section and is indicated by the clear area in L. 4, Fig. 63, and faintly in L b, Fig. 68. It is distinguished by a difference in time of development (Flechsig) and by freedom from the secondary degeneration that involves the rest of the column.

It is very doubtful whether all the fibres that enter the postero-median columns continue in these to the brain. Some certainly do, but the upward increase in size of the columns seems to be far too small for the accommodation of all the fibres that seem to pass to them. At the same time we have, at present, no indication of the mode in which fibres leave these columns.

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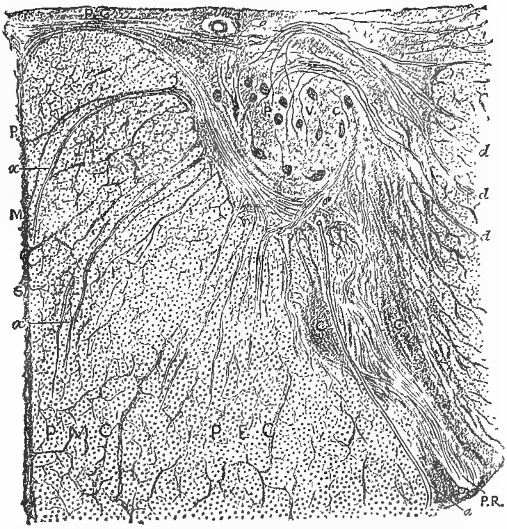
* This inference has been recently contested by Betcherew on the ground that secondary degeneration sometimes passes nerve cells. But in the central nervous system of man, as a general rule, the degeneration ceases where cells interrupt the fibres. This fact is so constant in so many parts that continuity of degeneration must be held as strong presumptive evidence of continuity of fibre, until the interruption has been demonstrated in the individual case.

At the medulla oblongata this column becomes filled with nerve cells, the "*post.-pyramidal nucleus*," so called because the highest portion of this column has been termed the *posterior pyramid* of the medulla. The discovery that the fibres of this column are continuous with the nerve roots invests the post.-pyramidal nucleus with considerable importance, since its nerve cells are the first with which these root fibres are connected. The upward degeneration of the post.-median fibres implies upward conduction; but their precise function is still uncertain. It will be further considered in connection with the localization of function in the cord.

FIG. 71.



Posterior cornu and column at the last dorsal segment. P. M. C., postero-median column; P. E. C., postero-external column; P. M. S., posterior median septum; P. C., posterior commissure; *v*, commissure vein; P. V. C., posterior vesicular column; C. C., caput cornu; P. R., posterior root; *a*, an artery; *d*, *d*, *d*, adjacent to a strip of the lateral column, indicate the tracts of fibres passing from the vicinity and interior of the posterior vesicular column along the septa of the lateral column, to form the direct cerebellar tract; *x*, *x*, tracts of fibres passing from the neck of the horn, near the post. vesic. col., to the post.-median column.

The postero-external column ("column of Burdach," "posterior root zone," Charcot) consists chiefly of vertical fibres. Many of the posterior root fibres pass through it, horizontally or obliquely (Fig. 57). Most of these curve inward to the posterior horn, but the secondary degeneration just described seems to show that some must also pass obliquely upward and inward to the median column. Across the anterior part fibres also pass, as just described, between the neck of the posterior horn and the median column. The other vertical fibres of the external column, "posterior ground fibres" of Flechsig, have apparently only a short course, since they only degenerate for a few centimetres above or below a lesion. The longest descending degeneration is of a comma-shaped bundle, in the middle of the anterior third of the column in the dorsal region, which degenerates downward for four or five centimetres (Fig. 69). These vertical fibres, of short course, may connect the gray matter of the posterior horn at different (but adjacent) levels. This column is larger in the enlargements than it is in the dorsal region, partly in consequence of the larger number of root fibres in the former. Above, it also ends in a gray nucleus.

The function of this column is still unknown, although much interest attaches to it as the seat of the special central lesion in locomotor ataxy. How far the incoördination of this disease depends on the damage to the root fibres that pass through the column, or to fibres that connect the gray matter at different levels, is still unknown. We shall return to the question when discussing the disease.

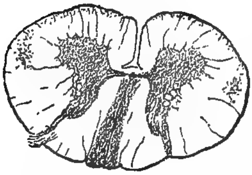
The "*direct cerebellar tract*" is another column of fibres which degenerate upward. It forms a layer at the periphery of the lateral column, outside the pyramidal tract (Fig. 57), but does not extend through the whole length of the cord, ceasing at the level of the first lumbar nerve (Fig. 63). The anterior part of the tract (as seen in section) does not extend forward beyond the anterior level of the lateral pyramidal tract, but, as we shall presently see, there are other fibres in front of it that also degenerate upward, and these have given rise to an erroneous opinion that the cerebellar tract extends farther forward than it actually does. But at the level of the second cervical nerves, and also in the lowest part of the dorsal region, the tract lies a little anterior to its position in the rest of the cord, and hence at these places, as we have seen, the pyramidal tract comes to the surface behind the cerebellar tract, close to the posterior nerve roots, up to which elsewhere the cerebellar tract extends. The tract increases somewhat in size from below upward, and hence receives fibres throughout its course, but the chief bulk of its constituent fibres enter it at the level of the lowest dorsal and first lumbar nerves, *i. e.*, at its lowest part. These fibres come from the gray substance, coursing to it through the lateral column, along the septa that cross the pyramidal tract from the gray matter. In the latter these fibres are conspicuous objects at this level (Fig. 71), passing transversely and obliquely from the front of the posterior vesicular tract. Into this many of them can be traced; others change their direction and become vertical, probably passing into the vesicular tract at a different level. It is probable that the fibres come from the tract; the cerebellar tract is chiefly formed at that part of the cord at which the posterior vesicular column is chiefly developed, and the cells of this column have been found atrophied when the cerebellar tract is degenerated.* Besides the fibres that are gathered into the compact tract, others, belonging to its system, ascend among those of the adjacent lateral pyramidal tract.

The cerebellar tract only degenerates when a lesion of the cord is at, or above, its place of origin, the junction of the dorsal and lumbar regions. If the lesion is in the lumbar enlargement, as in the case shown in Fig. 67, the tract does not degenerate. At the level of the first cervical nerve (where the pyramidal tract leaves the lateral column to cross into the anterior pyramid of the medulla) the cerebellar tract lies in front of the "gray substance of Rolando" (formed from the caput cornu posterioris), and passes up, in the restiform body, to the cerebellum. We have no definite knowledge of its function. It certainly conducts upward, and Flechsig thinks it may conduct impressions from the muscles of the trunk.

We have seen that the lateral pyramidal tract, although in contact, behind, with the head of the posterior cornu, does not extend quite up to the neck of the horn or to the intermediate gray substance, the two being separated by "*lateral limiting layer*" of vertical fibres, in part broken up by processes from the gray matter. In the upper cervical cord, and also to a less extent in the dorsal cord, this layer extends forward outside the anterior horn. It consists of fine fibres that pass into the gray matter, forming curves, some with the convexity downward, others with the convexity upward. This course, and the fact that the tract does not degenerate through any considerable extent, suggests that its fibres have but a short course, and connect the gray matter at different levels.

The rest of the lateral column in front of the pyramidal tract is termed by Flechsig the *anterior mixed lateral zone*, because its fibres vary in size, course, and date of development. Secondary degeneration, however, as I pointed out some years ago,† enables us to distinguish a band of fibres lying immediately in front of the pyramidal and cerebellar tracts, which degenerates upward throughout the cord. The zone extends across the lateral column, as a band which is largest in area near the periphery of the cord, where it fills up the angle between the pyramidal and cerebellar tracts, and it reaches the surface of the cord in front of the latter tract; it then extends forward in the periphery of the anterior column, almost to the antĕrior median fissure. It may be conveniently termed the *antero-lateral ascending tract*. (Figs. 57; 63, crosses; and 67.) It has often been confounded with the cerebellar tract, and it is this which has given rise to the impression that the cerebellar tract extends farther forward than it really does. In the lumbar region, the tract lies across the lateral column on a level with the posterior commissure, from which its fibres probably come (Fig. 63, L. 4). In the upper cervical (*Ib.*, C. 3) it extends also between the pyramidal and

FIG. 72.



Cervical section. Ascending degeneration in post.-med. col. and ant.-lat. ascending tract, secondary to a lesion in the dorsal region. (*From a section prepared by Dr. Mott.*)

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* *e. g.*, by Minkowski, *Deut. Arch. f. kl. Med.*, Bd. xxxiv, p. 433. Slight degeneration of the pyramidal tracts was the only other lesion.

† "Diagnosis of Diseases of the Spinal Cord," first ed., 1879.

direct cerebellar tract, and probably occupies a similar position at the level of the first cervical. This tract has recently been found by Betcherew to undergo development at a different period from the rest of the lateral column. It is almost certainly a sensory tract, and physiological facts seem to show that its fibres are connected with the posterior roots of the opposite side. Fibres probably pass to it through the whole length of the cord and these are mingled together, so that the degeneration from a lesion of the lower part of the cord is not dense.*

Some fibres of the anterior part of the lateral column are large in size, and curve upward and downward into the anterior horn. They must be of short course, and may be fibres of the anterior nerve roots, which ascend and descend to nerve cells on a different level from that of the roots by which they leave the cord.

The fibres of the anterior column, excluding the anterior pyramidal tract, are termed by Flechsig the *anterior ground fibres* (Fig. 57). They are not separable, structurally or by development, from those of the lateral limiting layer, and it is doubtful how far the distinction of the two is warranted. The ground fibres do not degenerate through any considerable extent of the cord, and some probably connect the anterior cornua at different levels. Moreover, some of the fibres pass to the anterior commissure, and thus, by the agency of these columns, a connection may be established between the two anterior cornua at different levels.

As far as investigations have hitherto gone, the direction in which fibres degenerate, in the central nervous system, is the direction in which they conduct. We may therefore feel reasonably sure that the fibres that degenerate downward from the brain, or upward to the brain, conduct as they degenerate, and through the same extent. But we cannot affirm that the tracts which do not degenerate through any considerable extent, do not form part of conducting paths to or from the brain. All we can feel sure of is that they do not constitute a continuous conducting path, such as the pyramidal tracts and the tracts that present a continuous ascending degeneration.

The *commissure* of the spinal cord lies between the bottom of the anterior fissure and the posterior columns, and in it alone is there a passage of fibres from one-half of the cord to the other. It consists of two parts, an anterior or white, and a posterior or gray commissure. The *white commissure* varies in thickness in different parts of the cord, and is largest in the lumbar region. It is composed of medullated fibres, which cross in the commissure in such a manner that the anterior fibres on each side pass out in the posterior part of the commissure on the other side. The fibres in front pass into the anterior white column, those behind into the gray substance; hence they appear to establish a connection between the anterior column of one side and the anterior cornu of the other. Whether they go to ganglion cells or to the anterior nerve roots is not known. It is not likely that any considerable number of fibres have the latter termination, but some are thought to pass from the roots of one side to the inner group of nerve cells of the opposite cornu (Laura and Pick). Some fibres, moreover, pass across the intermediate gray substance, and, reaching the lateral column, assume a vertical direction. It is probable that some fibres are decussating fibres of the direct pyramidal tract, so that part of the anterior commissure is to be regarded as a continuation of the decussation of the pyramids. Lastly, a few fibres of the anterior commissure have been thought to come from the posterior nerve roots.

The *gray commissure* contains very fine medullated nerve fibres, a large number of which pass backward in the posterior horn of each side, and appear to be continuous with the fibres of the posterior roots. It is highly probable that, by this means, a considerable number of the posterior root fibres cross (or fibres from cells in which the root fibres end), and that thus the partial decussation of the sensory path in the cord is effected, which is known to take place a short distance above the entrance of the nerves. If the gray commissure consisted only of root fibres, it has been estimated that it could contain only about one-half of those of the posterior roots. Some of the fibres that thus cross doubtless find their way to the antero-lateral ascending tract in the opposite lateral column. Others leave the commissure in the middle line, and pass backward to the posterior median column, but whether these decussate or not we do not know.

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* Other illustrations of the degeneration of this tract will be found in the section on Locomotor Ataxy. For an example of its degeneration and references to previous observations, see Tooth, *St. Bartholomew's Hospital Reports, 1885*, p. 137.

Within the gray commissure is the central canal of the spinal cord, lined with epithelium, and surrounded by granular material, similar in nature to that which caps the posterior horn. It is often filled up in adult cords.

The *course of the root fibres* remains to be considered. The anterior roots pass through the anterior columns in bundles which are distributed through an area of the column approximately corresponding to the width of the anterior surface of the cornu. In the gray substance, fibres from most of these bundles pass in three different directions, outward, inward, and directly backward in the middle of the horn, and thus cross each other and the fibres from the anterior commissure. Many of the fibres enter the groups of nerve cells and certainly end in these cells. Some of those that pass outward, enter the lateral white column ("mixed zone") and probably, after a short course in this, reënter the gray matter at a higher or lower level. Some of the fibres that pass inward may pass through the commissure to the opposite anterior horn.

The *posterior root fibres*, although arranged in a continuous vertical series at their surface attachment, divide into two sets, one of which passes through the "*caput cornu posterioris*" (Fig. 71), the other through the adjacent part of the post.-external column (Fig. 57). The former course through the gelatinous substance, sometimes in a compact bundle (Fig. 71), sometimes in curves of which the concavity is turned toward the centre of the head. Some of the fibres bend into a longitudinal direction above the caput (Fig. 71) and are seen in section as rounded bubbles. After a short vertical course, upward or downward, these fibres again pass forward into the horn. Of the fibres that pass through the postero-external column many pass inward and enter the posterior cornu in front of its head. These have not all a horizontal course; some pass obliquely upward or downward. Some may even have a vertical direction for a short distance. It is not likely that any fibres of the posterior roots pass far upward in the postero-external column, but it is probable that some pass inward, through this column, to the postero-median column, since the secondary degeneration of the posterior median column extends, below, into the outer column, when it is due to disease of the roots outside the cord.

In the posterior horn, many fibres of each set pass to the posterior commissure; others are believed to go to the posterior vesicular tract (where this exists), while some pass forward into the anterior cornu, chiefly toward the outer group of nerve cells, and a few pass toward the anterior commissure. It is probable that the fibres that pass to the posterior vesicular tract are connected with its cells, whether directly or indirectly through their ramifying processes and the spongy plexus, we do not know. Those that pass toward the posterior commissure are probably interrupted by nerve cells in the posterior cornu.

Blood Supply to the Spinal Cord.—The arterial supply is constituted by small branches, derived from the vertebrals, intercostals, and other arteries, which reach the cord by the anterior and posterior roots. The anterior pass for the most part inward, to the anterior median fissure, where they are connected by vertical branches, continuous in direction, which are described as the "anterior spinal artery." Branches also pass backward into the anterior median fissure, at the bottom of which they divide, and the small arteries enter the cord, passing outward through the anterior commissure, and again dividing in the gray substance. Small branches reach the lateral column (Ross). The fibres of the anterior commissure are displaced by the vessels, and hence, in section, the commissure often appears to be interrupted. The posterior spinal arteries give branches to the caput cornu posterioris, which course upward and downward in it (*a*, Fig. 71), and then divide into small branches which extend outward and inward, ramifying in the pia matter. From this ramification, arterioles pass inward into the white substance along the connective-tissue septa, send off minute twigs among the nerve fibres, and end in the gray substance. The branches into the lateral column do not reach the gray substance; the branches in the gray substance extend into the inner part of the lateral column.

The *veins* of the cord pass in two directions. Some, especially those of the white substance, pass outward along the septa, and those on the surface of the sides and back of the cord are gathered into a "posterior spinal vein." Others, especially of the gray matter, pass inward to a large vertical vein that lies in the gray commissure, a little distance from the central canal on each side (*v*, Fig. 71, compare also Fig. 57), from which branches pass forward to the anterior median fissure, and through this to an "anterior spinal vein." The anterior and posterior spinal veins deliver their blood, through communicating veins, into the huge venous plexuses that lie outside the dura mater, and which receive blood also from the bones, and from the structures and skin behind the spine. But the veins of the spinal cord cannot be injected from these plexuses, not because there are valves in the connecting branches, but apparently because they form so trifling a proportion of the total connections. From the plexuses blood passes to the various vertebral, cervical, and intercostal veins.*

Although there is a vertical continuity of the vessels of the cord, it is probable that the course of the circulation is, in the main, horizontal. From the very tortuous course of the path by which arterial blood enters the cord, it is evident that the pressure to which the arteries of the cord are exposed must be far lower than that in the arteries of the brain, and hence they are far less liable to degeneration and to rupture. On the other hand, the conditions that prevent an injection of the plexus of veins outside the dura mater from passing into the veins of the cord, must save the latter from the extreme over-distention to which they would otherwise be liable, when there is a hindrance to the return of blood from the plexus.

FUNCTIONS OF THE SPINAL CORD.

We are now in a position to consider the functions of the spinal cord, and the structures by which they are subserved. The functions are these: (1) The conduction of motor nerve impulses from the brain and sensory impulses to it. (2) The spinal cord constitutes a series of centres for reflex action. (3) It contains certain centres that ultimately govern nervous action in structures under the immediate control of the sympathetic system of nerves, the bladder, rectum, blood vessels, etc. (4) It influences the nutrition in all parts to which its nervous control extends.

Motor Conduction.—We have already seen that the path of motor impulses is through the pyramidal fibres (in the anterior and lateral pyramidal tracts), the anterior cornua, and the anterior nerve roots. The ganglion cells form part of the path, and so also probably does that part of the plexus of fibrillæ in the cornu which intervenes between the pyramidal fibres and the ganglion cells. We have also seen that the path may be regarded as consisting of two segments, an upper, cerebro-spinal, and a lower, spino-muscular (see p. 130). The conduction in the cord is chiefly on the side opposite to the hemisphere of the brain in which the impulses originate, to a less extent on the same side in the upper part of the cord (anterior pyramidal tract), the path crossing lower down by the anterior commissure.

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* A fuller account of the vessels of the cord will be found in some papers by Dr. Ross, in *Brain*, vol. iii, p. 80. At present we are not able to make a practical application of fuller details than are given in the text above.

The fibres for the leg, however, probably pass in part down the lateral tract of the same side, establishing thus a connection between the leg and both hemispheres of the brain. This is probably true also of the path to the bilateral muscles, such as the intercostals, and to other muscles of the trunk, and likewise to those muscles that pass from the trunk to the arm, muscles that are often used with their fellows on the opposite side. None of these muscles are ever completely and permanently paralyzed by a lesion on one side of the brain. We do not know whether this relation is subserved in any degree by fibres of the direct pyramidal tract.

The fibres of the anterior, motor, nerve roots, all of which can probably be stimulated through the pyramidal tracts, are far more numerous than the fibres of these tracts. The latter apparently end in a complex mechanism, constituted by the motor nerve cells and fibrillary network. This mechanism is such that the stimulation of certain pyramidal fibres excites to action a much greater number of nerve cells, so connected and arranged as to produce, through the related nerve roots, a complex movement in which many muscles may take part. The nerve cells thus connected may not all be at precisely the same level, and only some of those at a given level may be thus associated. The simpler the movements, and the fewer their possible variations, the fewer pyramidal fibres may suffice for the production of the movements. Thus it is conceivable that for such movements as those of the intercostal muscles, which are simple and constant, very few pyramidal fibres may suffice, whereas a much larger number must be necessary for the highly variable movements of the hand.

Representation of Movements in the Spinal Cord.—It is important to inquire how far we can identify these various motor mechanisms of the anterior gray matter. There are several sources of information on this subject. We have already seen that the arrangement of fibres in the anterior roots is such as to associate certain movements with certain nerve roots, and it is a reasonable assumption that this corresponds, to some extent at least, with the arrangement in the gray matter. The sources of our information on this point are the limited facts of anatomy, rare cases of restricted disease of the nerve roots in man, and especially the experiments of Ferrier and Yeo on monkeys. We have already considered the indications that these experiments afford, and their suggestiveness, and we have seen that their value is relative rather than absolute. As regards the gray matter, we must remember, also, that there may not be a strict horizontal correspondence between the nerve roots and the nerve cells, because it is probable that some root fibres enter the antero-lateral white columns, and are connected with nerve cells at a higher or lower level than that at which they leave the cord. Another source of information is the degeneration of nerve cells that follows slowly on an amputation of a limb, and the condition of the spinal cord in congenital absence of part of a limb. The last and most important source of information is supplied by cases of limited disease of the anterior cornua, in which the position of the lesion and the distribution of the resulting palsy can be compared. Destruction of nerve cells causes degeneration of the motor fibres proceeding from them, and wasting of the muscles to which those motor fibres proceed. This alone affords us certain knowledge. Cases of clear significance are, however, rare, and it will be long before our knowledge can be complete. Meanwhile, we may learn something of the central association of muscles by observing what muscles are most frequently paralyzed together by such disease. This subject has been carefully studied by E. Remak* and many valuable isolated observations have been published. It is only the associations which are frequent that can be allowed significance, because it is not uncommon to have more than one focus of disease in the gray matter. The following summary presents the conclusions that seem probable from the evidence at present available.†

The various facts for the most part fully confirm the conclusion of Remak, corroborated by the experiments of Ferrier and Yeo, that most movements and muscles are represented in vertical tracts, and the whole anterior gray matter, at any one nerve segment, contains cells that are concerned with different movements. An extensive lesion of small vertical extent may thus weaken many movements, but abolish none. The special representation of the muscular function, *i. e.*, of definite movements, is no doubt related to special groups of nerve cells, but a single group may be concerned in more than one associated movement. Different groups are probably intimately connected, because we know that the contraction of any muscle is accompanied by a slighter but proportioned contraction of its antagonists. Anatomical connections in both spinal cord and brain doubtless underlie this association.

Cervical Enlargement.—We are chiefly concerned with the lower brachial part of this region, that below the fourth cervical. The upper part contains centres for the muscles of the neck, especially for the sterno-mastoid and upper part of the trapezius. The diaphragm is probably represented in the gray matter at the level of its nerve roots, the fourth cervical. In the brachial region of the cervical enlargement, the muscles of the shoulder are represented chiefly in the upper part; the intrinsic muscles of the hand in the lower part. The flexors of the elbow are represented above its extensors; and the supinators and extensors of the wrist above the flexors of the wrist.

Deltoid, Scapular Muscles, Pectoralis, and *Serratus.*—Fifth and sixth segments, for the most part the sixth, and probably the outer group of nerve cells. The centres for the two parts of the pectoralis are separate, and are associated—the clavicular with that of the serratus magnus, the costal with that of the latissimus dorsi (see p. 49). These associated muscles are represented near together, but not at the same spot; the association is often present in disease of the anterior cornu, but one muscle may be affected without that which is commonly associated with it.

The *Flexors of the elbow* and *supinators* probably correspond nearly in level with the deltoid. The whole of this series of muscles may be affected alone by disease of the nerve roots (see p. 95) or of the gray matter, and then we have the "upper arm type" of palsy, described first by Erb.

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* *Archiv f. Psychiatrie*, vol. ix.

† A useful summary of the evidence up to the middle of 1884, was given by Dr. Allen Starr, in the *American Journal of Neurology*, Aug. and Nov., 1884. The subject is a tempting one for theory, but it is very important to keep to the solid ground of facts, however limited it may be in extent.

Extensor of the elbow (*triceps*): probably the middle of the brachial region, chiefly the seventh segment, extending down to the eighth, and perhaps up to the sixth, possibly chiefly in the outer group of nerve cells.

The *Extensors of the wrist* are represented above those of the elbow; probably chiefly at the sixth or sixth and seventh segments, and not from the postero-external, but from the other groups. *Flexors of the wrist*: below the extensors, nearly on the same level as the extensors of the elbow, at the seventh and eighth segments, and probably also from one of the outer groups of cells. The *pronators* have nearly the same representation as the flexors of the wrist.

Long Extensors of the fingers: upper part of the brachial region, about the level of the sixth, or between it and the seventh segment, probably from one of the anterior groups of cells; not from the postero-lateral group. *Long Flexors of the fingers*: below the extensors; probably seventh or eighth segments.

Intrinsic muscles of the hand: lowest part of the brachial region, and the thenar muscles a little higher than the interossei; the latter being represented in the first dorsal segment.* The intrinsic muscles are probably related less to the outer groups of cells than to the inner and anterior groups. We have seen that this is probably true also of the long extensors of the fingers. The two groups of muscles often act together, and their central connection was illustrated by a case of concussion lesion of the cord under my care, in which the two outer interossei were paralyzed and wasted, and also the part of the long extensor supplying these two fingers, but no other muscles.

Lumbar Enlargement.—Our knowledge of the representation of muscles in the lumbar enlargement is very slight, and we have scarcely any definite facts concerning their relation to the cell groups. The postero-external group preponderates over the others in size, even more than in the cervical cord, and doubtless also in importance. The few pathological observations of changes in the several groups are not altogether consistent. The chief facts available relate to the probable segmental level and association of the centres, and are as follows:—

Cremaster, second lumbar segment. *Psoas*, second, *iliacus*, third lumbar; the two muscles, one in function, are probably related to a group of cells extending through both segments. *Adductors*, fourth lumbar segment. *Gluteal muscles* (extensors of hip), fourth and fifth lumbar segments.

Extensors of Knee.—Third and fourth lumbar segments; probably from the same group as the flexors of the hip; the two sets of muscles are often affected together, and sometimes the adductors suffer with them. The *Sartorius* is probably related to the third segment, but not to the same cell group as the extensors of the knee (to which, indeed, it does not belong; see p. 56). It usually escapes in atrophic palsy of the extensors. *Flexors of knee*, fifth lumbar and first dorsal segments. Their centre is thus below and certainly distinct from that of the extensors.

The muscles of the lower leg, moving the foot and toes, are related to the fourth and fifth lumbar and the first sacral segments. The *calf muscles*, the *tibialis anticus*, and the *peroneus longus* have certainly separate centres and are often affected separately. The tibialis anticus may suffer alone, or may alone escape when the other muscles in front of the leg are paralyzed. The muscles of the calf may be affected when those in front of the leg are not. It

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* This is the indication of the majority of cases. Nevertheless, Sahli has recorded a case of atrophy of all the intrinsic muscles of the hand, in which the lesion did not extend below the seventh cervical: the eighth cervical and first dorsal segments were perfectly normal. I have seen a case of atrophy limited to the deltoid and intrinsic muscles of the hand, due to an acute process, probably hemorrhage, but in this case there may have been two lesions.

is probable that the peroneus longus is the highest in central representation, and is related to the fourth lumbar segment.

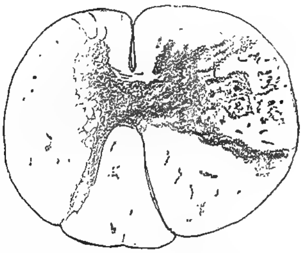
The *intrinsic muscles of the foot*, especially the interossei, are the lowest in central relations, being connected with the second sacral segment. The position of their centres, in the lowest part of the lumbar enlargement, thus corresponds to that of the analogous muscles of the hand, in the lowest part of the cervical enlargement.

Sensory Conduction.—Our knowledge of the sensory path in the cord is far less definite than that of the motor path, in spite of the fact that it has been the subject of a large number of experimental investigations. The subject is a very difficult one for experimental solution, on account of the difficulty of ascertaining the condition of sensation in animals. The indications afforded by disease are equivocal for another reason. We have seen that, in the sensory nerves, a lesion which permanently interrupts motor conduction may scarcely affect sensory conduction, and the same thing is apparently true of the spinal cord. If loss of feeling results from an acute lesion, it often quickly passes away, although motor palsy remains complete and absolute, and this when all the elements of the cord seem equally damaged by the morbid process. In cases of chronic disease, such as compression, which also acts equally on all the structures, the same escape of sensory conduction is often observed. The result, in acute cases, has been thought to indicate that the sensation may find new paths, but this explanation can hardly apply to cases in which all the elements are equally compressed. It is more probable that the nerve fibres suffer partial damage, without interruption of the axis cylinder, so that their conducting power is lowered, and not lost, and suffices for sensation but not for motion. We have seen reason, on more than one ground, to believe that this is the case in partial lesions of the peripheral nerves (pp. 70 and 71). Hence we may find disease in those elements of the cord, to which other facts point as the sensory path, and yet there may have been no corresponding loss of sensation. In an area damaged by sclerosis or chronic myelitis, it is common to find many nerve fibres retaining their form, although evidently changed in nature, since their white substance stains more readily than in health ; it is common also to find axis cylinders surrounded by a sheath of myelin much narrower than normal, and this in parts in which there is a considerable increase of connective tissue. Such fibres may still possess some conducting power. Hence we cannot infer, because the supposed sensory tracts are visibly diseased in a case in which there was no loss of sensibility, that these tracts have not the function assigned to them, unless we can feel sure that all the axis cylinders are destroyed. For this reason, and because morbid processes are often wide in distribution, the evidence that can be obtained by comparing the position of lesions with the sensory symptoms they produce, is limited and uncertain, Another source of information is the effect of partial transverse lesions of the cord, intense in degree, but limited in area, especially traumatic lesions affecting one-half of the cord or part of one-half. Cases of this kind are of the highest value, and it will probably be by these that the question will be ultimately decided. But the cases are few, at present, in which the extent of lesion and the range of symptoms have been accurately ascertained.

It would take too much space even to enumerate the various conclusions which have been reached by the many investigators who have endeavored to solve the problem of sensory conduction. It may suffice, for practical purposes, to point out the evidence that seems most important, and the conclusions that seem, on the whole, most probable. The earlier experiences of Brown-Séquard established one important fact, which has been fully confirmed by pathology, that the chief part of the sensory path crosses the middle line soon after it enters the cord, and passes up to the brain on the other side. These experiments also suggested that one upward path is in the intermediate gray substance. This possibility cannot be said to be entirely disproved, but later investigations have given it no confirmation, and it is on the whole improbable. The experiments by others which seem to deserve the greatest weight are those of Schiff and Woroschiloff. The latter employed, as a test of upward conduction, the effect of sensory stimulation on the vessels. These experiments, taken together, point to two regions of the white substance as containing the sensory path; the posterior columns and the lateral columns. In the lateral columns the path cannot be contained in the pyramidal tract, and it is not likely that the cerebellar tract conducts sensation from the limbs. Hence it is to the anterior part of the lateral column and the posterior column that experiment points. It cannot be an accidental coincidence that it is in these two regions that secondary degeneration proves the existence of fibres which conduct upward, and extend through the spinal cord to the brain. These are in the posterior median columns, and in what I have termed the "antero-lateral ascending tract" (p. 134). The experiments of Schiff suggest that the lateral column is the path of sensibility to pain, and those of Woroschiloff point to the same conclusion, since he refers to it the conduction of impulses that have a reflex influence on the vasomotor nerves, and such an influence is probably only exerted by the impulses that excite a sensation of pain. Schiff concluded that the conduction of tactile sensibility is in the posterior columns, and, moreover, in the posterior column of the same side, there being no decussation in the cord. The latter is certainly erroneous, so far as man is concerned. That the path of sensibility to touch, as well as that of sensibility to pain, crosses the middle line is proved by the effects of unilateral lesions of the cord. To these we may now turn, and ask how far they support the suggestions of experiment.

Unilateral lesions of the spinal cord, which cause loss of sensibility of the skin, do so on the side opposite to the lesion. This is true of all forms of sensibility. They thus prove, beyond doubt, the decussation of the spinal cord of the path from the cutaneous nerves of pain (*i. e.*, common sensibility), of touch, and of temperature. An analysis of the cases in which both effect and lesion have been exactly ascertained—which will be considered more fully when these lesions are described—suggests certain other conclusions regarding the position of these paths. They suggest, first, that the paths for sensations of pain and of touch are not near together. Sensibility to pain has been lost in almost all recorded cases, but that to touch in only two-thirds. They suggest, secondly, that the two paths for tactile sensibility are nearer together than are the two paths for common sensibility. In no case of chiefly unilateral lesion has sensibility to pain been lost on both sides, whereas in two recorded cases sensibility to pain was lost on the side opposite to the lesion, while that to touch was lost on both sides.* Both these conclusions harmonize with the suggestion of experiment, that painful sensations are conducted in the lateral column, those of touch in the posterior column. These conclusions, moreover, are supported by the facts of the only two cases that I have been able to find which afford direct localizing indications. One is a case, recorded by myself,† in which the spinal cord in the upper cervical region was damaged by a small spiculum of bone being driven against it by a bullet, which, entering the mouth, lodged in the body of the third cervical vertebra. The extent of the lesion of the cord is shown in Fig. 73. The chief injury is clearly to the lateral column and gray matter, the posterior column being merely swollen, apparently by œdema. The affected part was the seat of hemorrhage and hemorrhagic infiltration. The effect was entire loss of sensibility to pain on the opposite side without any impairment of tactile sensibility. To this the other case, recorded by Müller,‡ is almost a complement. A stab wound divided the whole of one-half of the cord, including the posterior column, and also the posterior column of the other side almost up to the nerve roots. The whole of both postero-median columns were thus divided. The effect was loss of sensibility to pain on the side opposite to the lesion, and of that to touch on both sides.

Fig. 73.



Section of spinal cord between the second and third cervical nerves, contused by gunshot injury.

From all the evidence, direct and indirect, it seems to be almost certain that the antero-lateral ascending tract constitutes the path for sensibility to pain, and to this conclusion no important facts are opposed. But it is otherwise with the other conclusion, also strongly suggested by the evidence we have reviewed, that the posterior median column contains the path for sensibility to touch. Regarding this column there is another class of facts, suggesting a different function for its fibres, and these facts must receive

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*It is possible that tactile sensibility may have been lost, in other cases, on the second side, *i. e.*, on the side of the lesion, and may have escaped notice in consequence of the exaltation of sensibility, which is commonly present, and which is presumably due to the nerves of common sensibility.

† *Clinical Society's Trans.*, vol. xi, 1878, p. 24.

‡ *Beitrage zur Path. Anat. und Phys. der Ruckenm.*, Leipzig, 1871. Abstracts, which supplement each other, and, together, give a full account of the case, will be found in Köbner's article on unilateral lesions, *Deut. Arch. f. klin. Med.*, *1877*, Bd. xix, p. 190, and in *Virchow's Jahresbericht*, *1871*, Bd. i, p. 152.

careful consideration before we can draw any conclusion regarding its relation to tactile sensibility. The column is often found diseased, and intensely diseased in some parts, when there is no loss of any form of cutaneous sensibility. Moreover, certain facts seem to indicate another function for the fibres of the median columns. We have seen that the way in which their ascending degeneration spreads out toward the periphery of the cord, just above a lesion, and reaches the posterior root zone, suggests that posterior root fibres pass directly to this column without decussating. There is one form of sensibility the path for which seems not to decussate in the cord—the sensibility of the muscles. The facts, old and recent, of unilateral lesions of the cord (considered more fully in the next section) supply conclusive evidence that the sensibility of the muscles is lost on the side of the lesion, as was first pointed out by Brown-Séquard. These non-decussating fibres of the posterior median columns may constitute the path for this sensibility. In harmony with this view is the fact that these columns are diseased almost constantly in locomotor ataxy (*q. v.*). But, even if we admit this theory as probable, we are not justified in assuming that this is the sole function of these columns. Fibres pass to them also in other ways, from the posterior commissure in the middle line, and from the neck of the posterior cornu (see Fig. 71), and, as the figure shows, some of these fibres may readily come from the posterior commissure, and both these sets of fibres may have crossed in the middle line. Is it possible that these fibres conduct tactile sensibility, and that they are mingled with those that conduct muscular sensibility? This theory, perhaps better than any other, harmonizes ascertained facts, but it can only be regarded as a suggestion for future observations. If the path is in the posterior columns, and not in their median portion, it must have a zigzag course, passing in and out of the gray matter scores of times in its upward progress, since no fibres of the external column have a continuous upward course of more than a few centimetres. Such an assumption must be a last resort of theory. The case figured on p. 143 seems to exclude the possibility that the gray matter can contain the tactile path.

The path for sensations of temperature is still unknown. The fact that this sense is frequently impaired with sensibility to pain suggests that the two paths may be near together in the lateral columns. ~~More than~~ this cannot be said.

Flechsig thinks that the direct cerebellar tract conducts sensation from the muscles of the lower part of the trunk. It may be diseased when there is no cutaneous anæsthesia, and is chiefly formed at the spinal level of those muscles. Hence the theory has some probability, although the function of the remarkable posterior vesicular tract, from which the fibres chiefly come, is not known. This view would assign a similar function to the direct cerebellar tract, and to part at least of the posterior median column. The post.-pyramidal nucleus, in which the latter fibres end, has also an extensive connection with the cerebellum. It may be that the impressions from the muscle nerves, conducted by these fibres, influence cerebellar coördination, but do not directly affect consciousness as a distinct sensation, although they may indirectly influence our conceptions.

But we have to face another difficulty in our localization of the sensory path. The same impression that is felt, may excite a reflex action. For this, afferent root fibres must end in the gray matter of the cord. Are the two functions subserved by the same or by different fibres? A similar question presents itself with regard to the muscle nerves. If the ascending degeneration in the postero-median column, when the cauda equina is diseased, is admitted, as provisionally it must be, as proof that these root fibres pass up without interruption, the muscle reflex action must be subserved by other fibres. For it, however, comparatively few fibres may suffice. With regard to the sensory fibres from the skin, the evidence at present suggests that all undergo interruption in the spinal cord, since the paths have not yet been found degenerated when a lesion has been confined to the nerve roots. If so, the same fibres probably subserve sensation and reflex action.

We know very little of the serial representation of cutaneous sensibility in the posterior roots. It is very probable, however, that there is a progressive representation of the skin, without relation to the muscles. We know that this obtains in the simpler arrangement of the nerves of the trunk; ascending disease causes an affection of sensation which extends around the trunk at higher and higher levels. In the limbs we have indications of the same relation: the soles and palms may be affected alone, and anæsthesia may extend on the limb to a certain level, irrespective of nerve distribution. This is often seen in the legs. I have seen anæsthesia in the arms, due to disease of the spinal cord, up to midway between shoulder and elbow, and there cease at the same level around each arm. At the same time, the strictly progressive representation may obtain only within the cord, and be modified in the nerve roots. Thus, affection of the first dorsal roots causes anæsthesia only in the ulnar region of the palm. Disease of the cord itself may affect sensation on the tips of the fingers only. It is possible that the afferent nerves from the muscles differ from those from the skin, and correspond in origin to the motor nerves to the same muscles.

Reflex Centres.—The gray matter of the spinal cord constitutes a series of reflex centres, some of which must be of considerable vertical extent and considerable complexity, and are subserved probably not only by the gray matter but by the short fibres that in the antero-lateral column (mixed zone and limiting layer) and the postero-external columns (posterior ground fibres) connect the gray matter at different levels. We have already seen (p. 34) that we must distinguish two forms of reflex action—the cutaneous reflex action and the muscle reflex action, the latter producing the "myotatic irritability" which is assumed to determine so-called "tendon-reflex contractions." The first form of reflex action is not, however, limited to impressions on the cutaneous nerves. It may be produced by stimulation of the nerves supplying the deeper structures, including those of the tendons and muscles. The attempt to obtain the knee jerk may cause a true reflex action as well as a local contraction, the two being separated by an appreciable interval of time. In the skin it is apparently subserved by all the sensory nerves. It may be present, although the nerves for either pain or touch are degenerated.

The central process involved in reflex action must take place between the posterior nerve roots and the anterior roots. The motor ganglion cells must take part in it, and form part of the reflex centre, and the rest of the centre is probably constituted by part of the fibrillary plexus, and perhaps also by sensory nerve cells. The whole part may be spoken of as the "reflex arc." Lines of different resistance doubtless exist in the centre, and determine the form of the reflex action. The stronger (within limits) the afferent stimulus, the wider does the process spread in the centre, and the more extensive is the movement produced. Thus a slight touch on the sole may cause only a movement of the foot, while a stronger impression may cause a movement of the whole limb. This is true only "within limits," because a very strong stimulation may prevent any reflex movement, by inhibiting the centre which a slighter stimulus discharges.

The motor ganglion cells from which the anterior roots proceed, must form part of the centre for each form of reflex action, but the afferent impression for the two must be conveyed by different nerves, and that portion of the centres which intervenes between the posterior nerve roots and the motor ganglion cells, may be distinct. Beyond this, we can say nothing of the relation of the two centres. The muscle reflex action must be the less complex. It is a simple effect of the stimulation of certain fibres on certain nerve cells, strictly limited to the cells related to the muscle from which the afferent impulse comes. The cutaneous reflex process is far more considerable in degree, wider in range, and more complex in character. In some of the lower animals it may attain the elaborateness of a purposed movement.

These reflex centres are subject to control. That for cutaneous reflex action must be controlled by a cerebral centre, since, when all cerebral influence is cut off, as by a transverse lesion in a higher part of the cord, the action of the lower spinal centres becomes excessive. We have no precise knowledge of the seat of the controlling centre or of the path by which the control is exerted. The mechanism must, however, be very complex, since many cerebral lesions, which cause hemiplegia, cause also a diminution of cutaneous reflex action on the paralyzed side. This effect seems to indicate that the cerebral centre which controls reflex action, is itself habitually controlled by a higher centre, perhaps in the cortex. When this is destroyed, or the path from it is interrupted, the controlling centre passes into a condition of increased activity, and the reflex action is lessened. This is a theory only, but it seems impossible otherwise to explain the facts.

The muscle reflex centres seem also to be under the control of other centres, because, if there is disease of the cord higher up, they also pass into a condition of increased activity. But the phenomena of their disturbance presents three important points of difference from those of the cutaneous reflex action. (1) We know with certainty the path, disease of which determines their excess: it is the pyramidal tracts. (2) Disease of the brain never causes a permanent diminution in the action of the spinal centres, as it so frequently does in the case of the centres for cutaneous reflex action. On the contrary, disease of the pyramidal fibres in the brain causes the same excess of this form of reflex action as does disease of the pyramidal fibres in the cord. (3) The excess does not, as a rule, quickly follow an interruption of the path from the brain, as does the excess of superficial reflex action. It is manifested at the end of a week or ten days, and gradually increases. Two explanations of the lateness of the manifestation of the excess are conceivable. The lesion of the pyramidal fibres causes also their degeneration below the lesion, and this must involve their termination in the gray matter. The excess of action of the centres may be the result of the degeneration of this terminal ramification, which may be the controlling structure that normally restrains the activity of the centres. In this case, the time before the excess is manifested may be that needed for the degenerative changes to descend the fibres to the level of the centres concerned. Secondly, the mere arrest of the central influences passing down the pyramidal fibres may cause the excess of the muscle reflex centres, and the interval which elapses before the excess is manifested may be due to the fact that there is little tendency to increased activity, that but slight habitual control is exercised or needed, and that the capacity for over-action is gradually developed and gradually increases. On the whole, it seems to be highly probable that the normal restraint is due, in some way, to the influence of the terminal portion of the pyramidal fibres within the gray matter, and that the excess of this reflex action follows any impairment of the integrity of these structures. This may not be, however, the only cause of excessive reflex action. A primary over-action of the centres is conceivable, and may occur in some conditions, but it is not probable that such over-action is the cause of the permanent excess which results from disease higher up the spinal cord.

The chief local forms of superficial and muscle reflex action have been already described (see pp. 35, *et seq.*). Their centres are found in the corresponding segments of the cord here enumerated. The superficial reflex centres are, however, extensive, and may be excited through a wider region than that indicated. (See also p. 152.)

Superficial Reflex Action.—Plantar, second sacral; gluteal, fourth lumbar; cremaster, second lumbar; abdominal, sixth to eleventh dorsal (epigastric, sixth dorsal); scapular, fifth cervical to first dorsal.

Muscle Reflex Action.—Calf muscles (foot clonus), fifth lumbar and first sacral; knee jerk, third and fourth lumbar; flexor digitorum, triceps, seventh cervical; biceps, supinator longus, sixth cervical.

Coördination of Movement.—The coördination of movement is certainly in part determined by the spinal cord. It is a complex function, probably depending on both motor and sensory arrangements, but chiefly on the impressions from the sensory muscle nerves. The consideration of its mechanism may conveniently be postponed until the discussion of its defect.

Influence of Nutrition.—The nutrition of the *muscles* is under the control of the anterior gray matter, and probably of the motor nerve cells. The influence is exerted through the motor nerves, and indeed consists in the normal conducting integrity of the fibres (see p. 44).

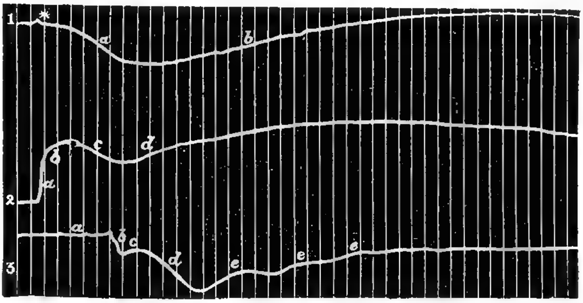
The nutrition of the *bones and joints* is also under the control of the cord, but we do not know by what part that control is exerted. The growth of the bones is hindered when the anterior cornu are diseased, but no change seems then to occur in the osseous structure. Such a change, and an alteration in the joints, may occur in locomotor ataxy, in which there is disease of the posterior nerve roots.

The nutrition of the *skin* seems to depend on nerves that have their course in the posterior roots, but we do not know whether there are special trophic nerves, or the position of the centres by which the influence is exerted. The clearest fact is that irritation of the nerve structures has far more effect than simple loss of function. It is when the cord or nerve roots are the seat of irritative inflammation that the most serious trophic changes occur.

Visceral Control.—Although the viscera are under the immediate control of the sympathetic system of nerves, they are related to centres in the spinal cord, and the relation is the most direct and important in the case of the organs over which the will has an influence, the *rectum* and the *bladder*. The centres for these are in the lumbar enlargement, but we do not know their exact position. They are probably complex reflex centres. We can best understand their action by studying them when voluntary influence is lost.

The centre for the sphincter ani is the more simple, but the system of action of each may be the same. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter arrangement to prevent their continuous evacuation. Fæces or air in the rectum, and urine in the bladder, may excite the lumbar centre, and cause two effects —contraction in the wall and relaxation of the sphincter. This process can be controlled by the will to a considerable extent, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is interrupted above the lumbar centres, the will can no longer control the reflex processes; as soon as fæces irritate the rectum, they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder, a reflex contraction of the detrusor, and relaxation of the sphincter, cause its escape. The affection of the voluntary path for the sphincters is not always proportioned to that for the legs. If the damage to the cord involves also the sensory tract, the patient is unconscious of the action of the bladder or bowel. If the sensory tract is unaffected, the patient is aware of the process, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition, evacuation occurs as soon as fæces or urine enter the bowel or rectum. The urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is not such continuous passage of fæces into the rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic contraction. I have verified this by introducing an India-rubber cylinder, instead of the finger, and registering the pressure on the cylinder by connecting it with a recording apparatus. The relaxation is then found to be preceded by a very slight brief contraction, and to be followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane above the sphincter. Fig. 74 shows some of the tracings obtained.

Fig. 74.



Tracings of the Action of the Sphincter ani. The vertical lines represent seconds of time.

1. Effect on contraction of sphincter of the injection into the rectum of a small quantity of air at *. *a*, Fall in pressure due to the inhibition of the contraction; *b*, rise due to the slowly returning contraction. 2. Effect of the introduction of the instrument. *a*, Sudden rise of lever at moment of introduction, due to the exposure of the instrument to the pressure of the sphincter (the top of this line represents the degree of previous contraction); *b*, initial rise due to increased contraction; *c*, fall from partial inhibition; *d*, subsequent contraction, rising to a greater degree than the initial contraction, and subsequently falling slightly. 3. Effect of cough. *a*, Pressure of tonic contraction of sphincter (the slight irregularities are due to pulse waves); *b*, fall in pressure, due to the movement of the instrument by the cough; *c*, initial contraction; *d*, relaxation of inhibited sphincter, *e*, *e*, *e*, rhythmical variations in subsequent rise. (*From the Proc. Royal Society, 1877.*)

The action of the bladder mechanism can be best understood by assuming that the motor centre really consists of two parts, one (MS, Fig. 75) maintaining the contraction of the sphincter, the other (MD) exciting the contraction of the detrusor fibres, and that these two parts are antagonistic; when one acts the other is inhibited. Thus, in normal rest, the sphincter centre is active, the detrusor at rest. Action is produced by an afferent impulse from the bladder and a voluntary impulse from the brain. Then the detrusor centre acts, and the sphincter ceases to act. If voluntary power is impaired, the afferent impulse from the bladder may be insufficient, and then there is retention, or in other cases the motor centre may yield too readily to the afferent impulse, and there is reflex incontinence.

Although no other part of the *alimentary canal* is under voluntary influence, all parts are probably related to the spinal cord, by means of the connection between the sympathetic system and the nerve roots. Constipation is extremely common in disease of the spinal cord, and is often greater than can be accounted for by the mere loss of power of the abdominal muscles. The pain in the back that is so common in disease of the stomach, has been regarded as a reflected pain, due to this connection.

FIG. 75.

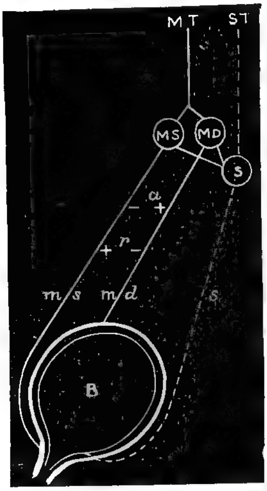


Diagram showing probable plan of the Centre for Micturition.

MT, Motor tract, ST, sensory tract in the spinal cord; MS, centre, and *ms*, motor nerve for sphincter; MD, centre, and *md*, motor nerve for detrusor; *s*, afferent nerve from mucous membrane to S, sensory portion of centre; B, bladder. At *r*, the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a*, the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

The *uterine* functions are probably independent of the cord in greater degree than those of any other organ under the influence of the sympathetic. The function of menstruation goes on independently of cord disease and may be regular, although there is absolute arrest of all conduction, sensory and motor. Pregnancy and labor may proceed in normal course, in spite of such disease of the cord as causes complete motor paraplegia.

Sexual Functions.—The activity of the sexual organs depends on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as on reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the cutaneous reflexes, and it shares the condition of these rather than of the muscle reflex processes.

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 146) is unimpaired, the reflex sexual processes are not in excess, and may even be diminished; but if the path from this controlling centre is also interrupted, the reflex sexual processes are in excess like the other superficial reflexes, and priapism results. If the reflex centre, or connecting paths, are partially diseased, the sexual process may be impaired and imperfect, but not abolished.

Vasomotor Centres.—The sympathetic nerves to the vessels are influenced from the spinal cord. We have no definite knowledge of the position of the centres or paths from and through which this influence is exerted, but some facts of disease suggest that the centres may be situated in the intermediate gray matter. The action of the vasomotor nerves may be deranged by disease of the cord. It is probable that such a relation exists between all parts of the vasomotor system of nerves and the cord, and that the relation obtains between each segment of the cord and the vascular nerves to the corresponding part of the trunk and limbs. This derangement in disease may take a part in the production of the changes in nutrition, but it is probably not the sole mechanism by which these changes are produced.

There is a special connection between the sympathetic and the cervical region of the spinal cord. This part contains the path to the visceral nerves that control the sugar-forming functions of the liver. The lower part of the cervical enlargement contains also an important centre which controls the sympathetic nerves for the vessels of the head and face. The path of central influence over the radiating fibres of the iris passes also from the region of the third ventricle, down the cervical cord, by the lowest cervical or first dorsal nerve, to the sympathetic, and then ascends the cervical trunk, and passes along the fibres which accompany the internal carotid artery to the ciliary ganglion. Fibres also pass from the cervical cord to the sympathetic through which the action of the heart is accelerated.

Mutual Relation of the Functions of the Spinal Cord.—It may be well to present, in the form of a table, the relation of the several functions of the cord, motor, sensory, and reflex, as far as our knowledge of these relations extends. Many points are still uncertain; these have been, for the most part, omitted. The table does not need any detailed explanation. It is merely a comparative collection of the facts that have been stated in the preceding pages. We shall find the value of this collocation of functions when we consider the effects of transverse lesions of the cord at different levels.

TABLE SHOWING THE APPROXIMATE RELATION TO THE SPINAL NERVES OF THE VARIOUS MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE SPINAL CORD.

MOTOR.	(NERVES.)	MOTOR.		SENSORY.		REFLEX.
	C 1	Small rotators of head	1	Scalp	1	
St.-mastoid, Upper neck muscles, Upper part of Trapezius	**2**		2	Neck and upper part of chest	2	
	3	Lev. ang. scapulæ	3		3	
	4	Diaphragm; Shoulder muscles	4	Shoulder	4	
	5	Serratus Flex. of elbow Supinators	5		5	Scapular
Lower neck muscles, Middle part of Trapezius	**6**	Ext. wrist and fingers	6	Arm	6	
	7	Ext. elbow Flex. wrist and fingers Pronators	7		7	
	8	Muscles of hand	8	Hand (ulnar n. lowest)	8	
	D 1	Intercostals	1	Front of thorax	1	
Lower part of Trapezius and Dorsal muscles	**2**		2		2	
	3		3		3	
	4		4		4	Epigastric
	5		5		5	
	6		6	Ensiform area	6	
	7	Abdominal muscles	7		7	
	8		8	Abdomen (Umbilicus 10th)	8	Abdominal
	9		9		9	
	10		10		10	
	11		11		11	
	12		12	Buttock, upper part	12	
	L 1		1	Groin and scrotum front	1	Cremasteric
Lumbar muscles	**2**	Cremaster	2	Thigh: outer side, front, inner side	2	*Knee jerk*
	3	Flexors of hip Extensors of knee	3		3	
Peroneus, l. Flex. of ankle, Ext. of ankle	**4**	Adductors of hip Ext. and abduct. of hip	4		4	Gluteal
	5	Flexors of knee	5	Leg, inner side	5	*Foot clonus*
	S 1	Intrinsic muscles of foot	1	Buttock, lower part Back of thigh Leg & foot except inner part	1	Plantar
	2		2		2	
	3	Perineal and anal muscles	3	Perineum and anus	3	
	4		4		4	
	5		5	Skin from coccyx to anus	5	
	Co.		Co		Co	

SYMPTOMS OF DISEASE OF THE SPINAL CORD: INDICATIONS OF THE SEAT OF THE LESION: ANATOMICAL DIAGNOSIS.

The symptoms of disease of the spinal cord consist in derangement of its various functions; the loss of some, the exaltation and perversion of others. We have already considered these functions, and the parts of the cord by which they are subserved. We may now briefly consider the character of their derangement and its significance in regard to the position of the disease. The combination of symptoms indicates the seat of the lesion; we infer its nature from their mode of development, and other considerations, which will be subsequently discussed. It is always important to keep these two elements of the diagnosis distinct in the mind.

A common feature of the symptoms of disease of the spinal cord is their bilateral character. This depends on two causes. First, the structures in each half of the spinal cord are in close proximity, and any random process, such as inflammation or tumor, readily affects both halves. Secondly, morbid processes that commence in the nerve elements, and affect them according to their function ("system diseases") usually involve the corresponding structures of the two sides. Hence the characteristic type of motor palsy is paraplegia. The vertical extent of the bilateral symptoms depends on the position of the disease. Here, however, we must distinguish two classes of symptoms. Those which depend on the interruption of the conducting path, to or from the brain, involve the whole region below the disease. Those which depend on damage to the centres in the cord, are present only at the level of the lesion.

But disease of the cord does not always cause bilateral symptoms. A random process, of small extent, may damage one side only. It may affect one or several structures on one side. Such a process never affects all parts of one side without damaging, in some degree, the other side, except in very rare forms of traumatic lesion. Again, "system diseases," in quite exceptional cases, are limited to one side, and, much more frequently, they may affect one side earlier and more than the other.

Motor Symptoms.—*Loss of motor power* results from interruption of the motor path in any part of its intra-spinal course,—pyramidal tracts, anterior gray matter, anterior nerve roots. We have seen that the path may be divided into two segments, upper and lower (see p. 130). The spinal cord contains a considerable portion of the nerve fibres of the upper segment, and their lower termination in the gray matter, but only the commencement of the lower segment, the motor ganglion cells, and root fibres proceeding from them. The chief part of the lower segment is outside the spine, in the nerve trunks. So far as the loss of power is concerned, the effect is the same, in whatever part of the motor path the interruption is situated, but the other symptoms that accompany the loss of power differ very much according as the interruption is in the upper or lower segment. The lower segment influences muscular nutrition, and forms part of the path of reflex action. Hence, as we shall presently see, the muscles waste, and reflex action is lost, when this is diseased; but when the upper segment is damaged, there is no muscular wasting, and reflex action, instead of being lost, is commonly increased. The symptoms produced by disease of either segment are essentially the same, whatever part of the segment is diseased, whether the cell at the upper extremity, the fibre, or the ramification at the lower extremity of the fibre. If, for instance, the intra-muscular nerve ending is paralyzed by curara, the symptoms produced are the same as if the fibre in the nerve is divided or the cell in the cord is destroyed. Disease of the termination of the upper segment, in the gray matter of the cord, must, of necessity, produce the same effect as disease of the pyramidal fibre itself, or the cell in the cerebral cortex from which the fibre springs. We shall afterward see that this consideration has very important applications.

If the fibre of either segment is divided, it degenerates below the division, since its nutrition depends on the influence of the cell from which it has sprung. It is probable that the nutritional stability of the fibre, or rather of its essential element, the axis cylinder, becomes less, and more easily deranged, the greater the distance from the parent cell, and that it is least in the terminal ramification of each segment. This may be the reason why curara acts chiefly on the intra-muscular nerves. If this is true of the upper segment, many facts of pathology become clearer to us.

A lesion of the pyramidal tract causes loss of power in all parts below the level of the disease, that is to say, of all parts the fibres for which are interrupted. A lesion of the gray matter, or of the anterior roots, causes paralysis only of those parts which are, functionally, on the level of the lesion. The two mechanisms often coincide. A transverse lesion in the cervical enlargement, for instance, may cause paralysis of the arms from the damage to the gray matter and nerve roots, and paralysis of the legs from damage to the pyramidal fibres. The disease involves, in the former case, the commencement of the second segment of the motor path for the arms; in the latter, the middle of the upper segment. The associations of the paralysis differ accordingly. The parts affected by a lesion at any level will be readily ascertained by an examination of the table of functions on p. 152.

It is important to note that the affection of motor power is often incomplete. It may then involve one set of muscles more than another. The flexors or the extensors may be chiefly paralyzed. In disease of the dorsal cord, it is very common for the flexor muscles of the hip and knee to suffer more than the extensors. Why this should be we do not know, but the fact is important, because considerable weakness of the flexors of the knee is readily overlooked. In examining motor power, each set of muscles should be separately tested.

Motor over-action; Spasm is frequent in disease of the spinal cord and its membranes. In the latter case it seems to be the result of direct irritation of the motor structures. In some acute lesions of the cord itself, spasm seems to have a similar origin. In chronic disease the mechanism is certainly different, and apparently depends on over-action of the reflex centres, due, not to irritation, but to deficient control. The spasm may preponderate in either the flexors or extensors, so that the legs, when rigid, may be drawn up or straight out. The flexor spasm seems to be due to an over-action of the centres for cutaneous reflex action, the extensor spasm chiefly to that of the centres for muscle-reflex action, although it may also be excited by a cutaneous impression. The latter will be better understood when the disturbance of this form of reflex action has been described.

Muscular contractions, shortening of muscles, occur in three different ways. (1) As a result of long-continued irritation of motor fibres, chiefly of those of the nerve roots. This form has been already described and illustrated in the account of diseases of the nerves (p. 96, Fig. 50). (2) From long-continued spasm, shortening of muscles may occur. Usually the contraction is facilitated by weakness of the opponents or by posture. It is especially common in the flexors of hip and knee, in cases in which flexor spasm predominates, and in the extensors of the ankle when there is extensor spasm. The last is aided by the extended position of the foot in the recumbent posture. (3) Whenever one group of muscles is much more paralyzed than their opponents, the latter become contracted, although spasm is absent. Such contraction is common in atrophic spinal paralysis, and gives rise to serious deformities. The active contraction of muscles in all these forms may ultimately result in actual tissue changes in them, so that they cannot be passively extended except by the application of great force. This condition must be carefully distinguished from active contracture.* In the latter, gentle extension, kept up for a few minutes, restores the muscle to its normal length. All forms, and especially the first two, begin as an active contracture, and it is only after this has continued for some time that the tissue changes occur, which prevent extension. In the third form, however, the tissue changes develop much more quickly than in the second.

Incoördination of movement, although a motor symptom in character, is probably sensory in origin. It occurs chiefly in disease of the posterior columns and posterior nerve fibres. This is its chief significance. It has also been met with in rare cases of unilateral lesion of the cord. Its probable mechanism will be presently mentioned, and will be discussed more fully in the account of locomotor ataxy.

Sensory Symptoms.—Loss of sensation is a very common effect of disease of the spinal cord. It may be total, and involve all forms of sensibility, or partial and affect only some forms. The statements made on pp. 29–34 regarding impairment of sensation generally, and the method of ascertaining it, apply to that which results from cord disease, and need not be here repeated. It has been also pointed out (p. 141) that loss of sensation occurs less readily than loss of motor power, so that it is common for complete muscular paralysis to exist with intact sensibility. The latter may be

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* The word "contracture" is applied to persistent shortening of a muscle, depending on active contraction of the fibres, and not on tissue changes.

impaired by disease of any part of the sensory path,—posterior roots, probably also the posterior cornua and commissure, or the conducting tracts in the cord. A division of the sensory path into upper and lower segments is conceivable, analogous to that of the motor path, although we have not the same clear ground for the distinction. Disease of the posterior nerve roots causes loss of reflex action as well as loss of sensation, just as disease of the anterior roots interferes with the reflex action as well as with motor power. Interruption of the sensory conducting tracts higher up leaves reflex action unaffected. A focal lesion, such as transverse myelitis, may damage both the conducting tracts and the nerve roots, or only one of these. Disease outside the cord, compressing it, often has the same double effect. Hence it is important to test sensation at the level of the lesion, as well as in the parts below. Areas of anæsthesia may thus be found on the trunk when there is none in the legs. Considerable disease of the nerve roots outside the cord may impair all forms of sensibility, although slight damage may arrest the conduction of tactile impressions, and not those of pain, which are probably more energetic. Disease of the roots within the cord often causes only partial loss, probably because the fibres which conduct different impressions have a different course. Disease higher up the cord still more frequently causes partial loss; either sensibility to pain or to touch may be impaired. That to temperature is rarely affected without that to pain. We do not yet know precisely the significance of the special form of loss. It is highly probable, however, that loss of sensibility to pain is produced by disease of the antero-lateral ascending tract (p. 142). Future observations must determine whether loss of tactile sensibility can be regarded as evidence of disease of any part of the posterior columns.

It is probable, however, that disease of the posterior median column and cerebellar tract impairs the conduction of impressions from the muscles, although it is doubtful to what extent the impressions thus conducted influence consciousness, and how far their arrest entails a distinct sensory loss. They may pass up to the cerebellum and influence the coördinating function of its middle lobe, and even influence indirectly the cerebral processes which subserve conceptions of movement and posture, without giving rise to any conscious sensation. At the same time it is certain that sensations from the muscles may influence consciousness directly, as the pain of cramp sufficiently shows. The sensory muscle nerves are apparently influenced by pressure and extension; and impressions from them, determined by the adaptation of the muscles to their elongation or shortening on passive or active movement, are the chief elements in the sense of posture. The methods by which this sense can be tested have been already described (p. 33). Its loss is more common in disease of the nerves or nerve roots (as in tabes), than in disease of the conducting tracts, but is often very marked in cases of unilateral lesion of the cord (see p. 165) and then exists only on the side of the lesion. The sense of posture seems to be impaired more readily, and by a slighter lesion, than the sensitiveness of the nerves to external pressure or extreme contraction of the fibres, whether that of cramp or produced by electricity. The impairment of the function of these nerves seems capable of causing incoördination of movement (see p. 166). Perhaps this is due to the interruption of the fibres that go to the cerebellum.

Increased sensitiveness, hyperæsthesia and hyperalgesia, are also common in disease of the spinal cord, and usually depend on irritation of the conducting fibres in some part of the path, but whether the irritation produces the effect by intensifying the impression as it passes, or by exalting the irritability of the related part of the cerebral centre, we do not know. The former hypothesis is tenable, since the phenomena of stimulation of nerves shows that their axis cylinders have some power of transforming other forms of energy into nerve force, *i. e.*, of evolving nerve force, and therefore of increasing the strength of that which passes along them.

The increased sensation may be felt as actual pain, or as "thrilling" or "shock-like sensations." It is probable that the latter are due to increased sensitiveness of the nerves of tactile sensibility. When there is an extreme increase, pain may be produced by a touch, but it is more probable that the touch stimulates the over-sensitive nerves of common sensibility, than that actual pain is produced through the tactile nerves. Other forms of altered sensibility will be described in the account of the disease in which they occur.

Pain, referred to the spine, occasionally present in organic disease of the cord, is more frequent in disease originating in the meninges or bones. But the frequency with which spinal pain is present in abdominal, especially gastric, disease, and in neuralgic affections, lessens its diagnostic value when it exists alone. Moreover, in cases of organic disease, pain is far less frequent when the disease begins in the cord, than when it commences in the protecting structures. In meningitis, acute or chronic, spinal pain is frequent, and in organic disease of the bones of the vertebral column it is an almost constant symptom, and is combined with local tenderness. The same combination of local pain and tenderness frequently occurs in cases of neuralgic pain, "rachialgia,"—a condition that is often loosely termed "spinal irritation," especially when it succeeds, as it often does, concussion of the spine. When pain about the spine occurs in organic disease of the cord, it is more often referred to the neighborhood of the spine, to the loins or the sacrum, than to the spinal column itself. Such pain is occasionally an early symptom of organic disease, but its origin and precise significance are not known.

A still more important group of pains are those which are referred to the parts to which the sensory nerves are distributed, and have hence been termed "excentric" or "radiating pains." They are due to the irritation of the posterior nerve roots in their passage through the intervertebral foramina, through the membranes, or through the posterior columns of the cord. Other similar pains, usually less acute, may be produced by irritation of the sensory conducting tract higher up the cord. These are often dull pains, closely resembling rheumatism, and frequently mistaken for rheumatism by the patients themselves and their medical attendants. The mistake is the more easily made, because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains, in acute cases, may be accompanied by febrile symptoms, and in chronic cases may be influenced by weather, being much more troublesome in damp and cold than in fine and dry weather. In all cases, persistent rheumatic pains in the limbs should suggest the possibility of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. In other cases, especially in locomotor ataxy, the pains are sharp and darting. The position in which the various radiating pains are felt—legs, trunk, or arms—depends (when the nerve roots are irritated) upon the seat of the disease—at the lumbar, dorsal, or cervical region of the cord. Occasionally the irritation is felt, not as a sharp pain, but as a painful sense of tightness, as if a band were tied tightly around the limb or trunk—the "girdle pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve endings) causes the girdle pain. When the nerve roots are irritated by disease of the vertebræ, caries or cancer, the pain is sometimes severe, and, in cancer, is especially increased by movement.

Spontaneous sensations, other than pain, are very common in disease of the spinal cord, and are often of considerable diagnostic importance, but suggestive rather of the presence of a morbid state than of its nature. They may present many varieties of character, but the familiar sensation produced by pressure on a nerve trunk, when the part is said to be "asleep," is the most common and the most significant. It may occur in functional disturbance of the cord as well as in organic disease. It no doubt depends on over action of the sensory structures, but it has no special significance as to the locality of disease. It may be excited by contact when it is not spontaneous. The still vaguer feeling called "numbness" is also common, and may occur, in slight degree, in diseases of which all other symptoms are purely motor. All spontaneous sensations are felt most in the extremities of the limbs, on the palms and soles, fingers and toes.

Reflex Action.—*Loss of reflex action* indicates an interruption of the reflex arc concerned. This interruption may be anywhere between the peripheral endings of the motor and sensory nerves, and thus is not necessarily within the spinal cord, or even within the spinal canal. The position of the interruption must be determined by the associated symptoms; if it is in the centripetal portion of the reflex arc, there is impairment of sensation, since the interruption will equally arrest conduction to the brain. If it is in the centrifugal portion of the arc, there is a corresponding interruption in the path of the voluntary impulse, and loss of motor power. Moreover, disease of the motor centre or motor nerves causes also degeneration of the nerves, and wasting of the muscles. Disease of the reflex centre, or motor nerves, usually causes loss of each form of reflex action, but partial disease of the centripetal path may impair one and not the other, may abolish the muscle-reflex action (myotatic irritability), and not the superficial reflex action, since the sensory nerves for the two are distinct, coming in the one case from the muscles, in the other from the skin. It is also possible that partial disease of the central structures within the cord may impair one and not the other, but we have at present no evidence of the difference in position of the lesion that would cause the difference in effect. It is important to remember that loss of all reflex action may occur as a transient symptom, immediately after the onset of an acute lesion of the cord, from irritative inhibition, and that loss of cutaneous reflex action may occur as a persistent symptom in cases of brain disease.

Excess of reflex action is also very common in disease of the spinal cord. It implies, of necessity, the integrity of the reflex arc concerned. Each form of reflex action may be increased. In some acute diseases, the increase may be due to an irritation of the centres, but in chronic disease it is apparently the result of a loss of control, and indicates disease between the centre concerned and the brain. We have already considered (p. 146) the probable mechanism, and have seen that the excess of the muscle-reflex action is related to disease of the pyramidal fibres, and especially to the degeneration or altered functional condition of the terminal part of these fibres, within the gray matter, adjacent to the centres concerned.* This is the significance of considerable excess. Degeneration of the terminal portions of the fibres is commonly secondary to degeneration of the fibres themselves. It is possible, however, that the termination of the pyramidal fibres may be affected without degeneration of the fibres themselves in the lateral column. (This subject will be further considered in the account of primary spastic paraplegia.) The increase of reflex action is chiefly manifested in the legs, in which reflex action is normally more active and important than in the arms. The increase in the cutaneous reflex action is shown by its occurrence on a slighter stimulation of the skin, and by a greater degree and extent of the muscular contractions produced. In these, the flexors usually predominate, so that the leg is drawn up in a strong movement. As the action of the sexual organs depends, in part, on superficial reflex processes, priapism is common when the reflex excess is great. The increase of the muscle-reflex action is shown by an increase of myotatic irritability, and therefore of the so-called "tendon reflex contractions,"—the increased knee jerk, rectus clonus, foot clonus, etc. These have been already described (pp. 35 *et*

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* It is interesting to note the analogy between the effects of degeneration of the termination of the two segments of the motor path. The over-action, increased irritability, of the muscle-reflex centres, which results from degeneration of the termination of the upper segment, presents some similarity to the increased irritability of the muscular fibres which results from degeneration of the termination of the lower segment. The analogy must not, however, be overestimated, since the altered mode of response to electricity of the muscular fibres, and wasting of the muscles, seem to show an abnormal state of nutrition, of which we have no indication in the reflex centres of the cord—unless it be in the slow and moderate muscular wasting, mentioned in the next section, as occurring after other *irritating* lesions of the pyramidal fibres—a wasting which indicates a slow change in the nutrition of the ganglion cells.

seq.).* The excess of this myotatic irritability is at first moderate in degree, and slowly increases. A high degree of over-action is never suddenly attained, as it may be in the centres for superficial reflex action. It would seem as though, in the centres liberated from control, a sort of functional hypertrophy occurs; their nutrition, and capacity for action, responds to the increased demand upon them.

Reflex Spasm.—We have seen (p. 40) that the muscular state on which myotatic irritability depends, assumed to be due to a muscle-reflex action, is probably identical with physiological *tone.* Whenever there is a considerable increase in the irritability, there is a tendency to tonic extensor spasm, which is probably an excessive and morbid degree of the normal tone. At first there is merely slight stiffness of the legs, especially felt on waking from sleep; afterward the legs, when extended, become distinctly stiff, although they may be quite supple when flexed. If gradually extended, when near full extension the spasm suddenly comes on and completes the movement, as the blade of a pocket knife moves under the influence of the spring. Hence this has been termed "clasp-knife rigidity." Ultimately the extensor spasm may be so great that, when it occurs, both legs are almost as rigid as an iron bar, and are so firmly connected with the pelvis, that if one is lifted from the bed the other is moved also. Paroxysms may be brought on by any impression on the sensory nerves, a prick on the skin, or an attempt to obtain the clonus. The extreme spasm prevents any clonus being obtained, and it may be necessary to flex the limb before an attempt to produce the clonus is successful. As violent spasm is passing off, however, the tension of the muscles may alone set up a clonus, so that the paroxysm of tonic spasm passes into clonic spasm, a phenomenon that was termed by Brown-Séquard "spinal epilepsy," on account of the superficial resemblance to the sequence of spasm in an epileptic fit. This clonic spasm may often be arrested by any strong painful impression on the sensory nerves, a pinch of the skin, or painful flexion of the big toe. There is always much weakness of the legs, when there is much spasm, and there may be complete paralysis. Hence the condition has been termed "spastic paraplegia." It is probable that the central mechanisms concerned in the production of this spasm are those concerned in the reflex act of standing. The spasm often enables a patient to stand, when his voluntary power is quite insufficient for the act. The extensor spasm may be varied by attacks of flexor spasm. The latter are especially apt to come on during sleep.

Vasomotor and Trophic Disturbance.—*Changes in Nutrition.*—Considerable wasting of the *muscles* indicates disease of the motor nerve cells or of the nerve fibres proceeding from these cells. If the disease is acute, there is rapid degeneration of the nerves, with the reaction of degeneration —loss of faradaic irritability of the nerves, both within the muscles and outside them, with preservation of the voltaic irritability of the muscular fibres themselves (see p. 66). The wasting in these cases is always great, and there is always loss of reflex action. Occasionally some fibres of the nerve and muscle suffer and others do not, causing the middle or mixed form of reaction (see p. 45). In other cases of disease of the spinal cord, however, there is a slighter degree of wasting of the muscles; there is no loss of irritability in the nerves; the irritability may be slightly increased or lessened, but is changed in the same way to each form of electricity. In this condition there is no loss of reflex action. The change is apparently the result of a peculiar alteration in the nutrition of the nerve cells. It is usually the effect of an *irritative* degeneration of the pyramidal fibres.

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* In addition to the phenomena there described, Morris Lewis has pointed out that a similar contraction in the muscles of mastication can be obtained by striking the chin when the mouth is open, and that this contraction may be excessive when there is a general increase of these phenomena. Lewis called it the "chin reflex" (Philadelphia *Med. and Surg. Reporter*, May, 1886). It has also been described by Beevor (*Brain*, Jan., 1886). De Watteville (*Ib.*) proposes for it the name of "jaw jerk."

The changes in the nutrition of the skin, if slight and chronic, resemble those produced by disease of the nerves (see p. 72), and suggest disease of the posterior roots. Sometimes, however, these changes are more acute than is ever seen in disease of the nerves. Very slight pressure, continued for a few hours, causes vesication, and even sloughing of the skin. Sometimes bullæ form where there has been no pressure. Occasionally, there is inflammation of the joints. Such intense changes are only met with when the morbid process in the cord is irritative in its character. The points on which sloughing occurs with greatest readiness are the heel, over the malleoli, the trochanters, and the sacrum. The sacral position of the bedsores in paraplegia constitutes a difference from those which occur in hemiplegia, which form over the gluteal region on the paralyzed side.

Vasomotor disturbance is common in slight degree, irrespective of the seat of the disease, but, like the trophic changes, which probably depend, in some degree, upon it, it is always most intense in irritative lesions of the cord. The slighter and common degree consists merely in increased warmth of the limb in the early stages, with increased redness, while, at a later period, the limbs are colder, paler, and often livid at the extremes. In rare cases intense flushing of the skin has been observed. This has been seen chiefly in cases of disease of the intermediate gray matter. In the same class of cases copious and long-continued perspiration sometimes occurs. When the disease is in the lower cervical region, the flushing and perspiration involve the face and head.

Disease of the cervical cord or nerves is sometimes attended by a change in the pupil, usually one-sided. There may be contraction, from paralysis of the radiating fibres (the sympathetic supply of which is ultimately derived from the cord), or there may be persistent dilatation from irritation of the fibres. The double myosis, so common in some degenerative diseases of the cord, is probably not due to the latter, but is the result of simultaneous degeneration of part of the nucleus of the third nerve.

The frequency of the heart's action is often permanently increased in locomotor ataxy, but we do not know by what mechanism. In disease of the cervical cord (perhaps also in that of the cervical nerve roots) the heart may be slowed to forty, thirty, or twenty beats per minute, usually only for a time. The effect may perhaps be due to a loss of the influence of the nerves which, when stimulated, quicken the heart, or it may be produced by an indirect upward influence on the centre for the vagus.

Visceral and Other Disturbances.—Various disturbances in the *alimentary canal* occur in disease of the spinal cord, and are no doubt due to the fact that the sympathetic nerves immediately controlling the canal are under the influence of the spinal cord, and may be excited to abnormal action by its irritation, or may underact when its influence is lessened. Thus difficulty of deglutition is observed in some cases of acute disease of the upper cervical cord. Vomiting occurs also in disease of the same part, especially in fracture of the cervical vertebræ, and, in paroxysmal form, attends some cases of chronic disease. Constipation is extremely common in disease of the cord above the lumbar enlargement.

Paralysis of the Sphincters.—Incontinence of urine and fæces may be due to disease of the centres that control the action of the sphincter ani and the muscular mechanism of the bladder respectively, or it may be due merely to disease of the path (probably the pyramidal tract) through which voluntary control is exerted. In the latter case the sphincter ani acts in an automatic reflex manner, as already explained. If the finger is introduced within it, the initial relaxation is felt to be succeeded by a firm tonic contraction. This proves that the anal centre in the lumbar enlargement, and the nerves proceeding from it, are intact. If this centre is destroyed, or the nerves interrupted, no tonic contraction can be felt; there is complete and persistent relaxation. The sphincter of the bladder is not accessible to such direct examination, and the mechanism seems to be more complex, but we may distinguish conditions corresponding to those of the rectum. If the lumbar centre is destroyed the sphincter is permanently relaxed; urine dribbles out of the bladder as fast as it enters it; there is simple incontinence. If there is an interruption of the voluntary path, the sphincter may act automatically; when a certain amount of urine has collected in the bladder it excites the muscular fibres in the wall to contract; the sphincter relaxes, and the urine escapes; there is intermittent incontinence. In this condition the mechanism is excited to action by any pressure on the bladder; a movement, or the act of coughing, will make the urine come away. But the muscle of the wall of the bladder is indirectly under the influence of the will; when the sphincter is voluntarily relaxed the detrusor fibres in the wall contract. Loss of voluntary power seems to lead to weakness in the wall of the bladder, so that the bladder is never perfectly emptied, and ultimately the detrusor cannot contract to expel the urine, even when the sphincter is relaxed. Hence retention of urine occurs. When a certain degree of distention of the bladder is attained, the pressure becomes sufficient to force the urine out, whenever the sphincter relaxes, and such relaxation is continually occurring under the high pressure that is continually renewed in consequence of the flow from the kidneys. Hence incontinence results—"overflow incontinence." It is important, if the urine comes away involuntarily, to ascertain which form of incontinence exists; since overflow incontinence, and the retention it indicates, have much graver consequences than simple incontinence. The permanent distention of the bladder constitutes a permanent hindrance to the flow of urine from the kidneys, and serious kidney disease may result. This is a not uncommon cause of death in spinal disease. Moreover, whenever the bladder is habitually emptied imperfectly, decomposition is apt to occur in the residual urine. The decomposition is often aided by the lessened acidity, or even actual alkalinity, of the urine. This change in the composition of the urine excites inflammation of the bladder; pus is formed by the mucous membrane, and this, in its turn, increases the decomposition of the urine. The damage to the kidneys, by the hindrance to the flow of urine from them, may be increased by inflammation ascending the ureters from the bladder.

Pyrexia.—The general body heat is sometimes changed in disease of the cord apart from the influence of the kind of lesion. Disease of the upper cervical cord, like that of the medulla, sometimes causes hyperpyrexia, and this may also result from acute central lesions that occupy a considerable vertical extent, such as a central hemorrhage.

Convulsions.—Lastly, convulsive attacks have been known to attend the onset of acute lesions in various parts of the cord, in adults as well as in children. They are most frequent in disease of the cervical cord, but I have known a convulsion to occur at the onset of myelitis in the lower dorsal region. The convulsions are general. Their mechanism is unknown, but in connection with them the readiness with which hemisection of the cord renders a guinea-pig epileptic may be borne in mind.

Combination of Symptoms.—Certain symptoms are frequently combined, and their combination has a definite localizing significance. Loss of motor power occurs in two different associations, according to the position of its cause in the motor path. It may be combined with muscular wasting, loss of electric irritability, and loss of all reflex action. In this case the disease is in the anterior cornua or nerve roots, the lower segment of the motor path. On the other hand, there may be no wasting, no change in irritability, and, instead of a loss of reflex action, the myotatic irritability is increased. The interruption of the motor path is then in the upper segment, usually in the pyramidal tracts. It may be a focal lesion of the cord, or a primary degeneration of the tracts. The distinction is that, in the latter case, there is nothing more than the muscular weakness and increased myotatic irritability. In the former case there are, or have been, indications that the lesion has extended beyond the purely motor elements.

The symptoms caused by disease of the several elements of the cord have now been described, but it is necessary to consider further the combination of symptoms that results from a total transverse lesion of the cord, and from a unilateral lesion.

A *total transverse lesion*, however limited in vertical extent, separates from the brain all parts below the lesion, and hence, so far as will and perception are concerned, it produces the same effect as if the whole of the cord below the lesion were destroyed. A section across the cord in the middle of the cervical enlargement, for instance, abolishes motion and sensation in all parts below the distribution of the cervical plexus and the phrenic nerve. Hence the extent of the motor and sensory paralysis indicates only the upward limit of the lesion. This is also indicated by the position of the girdle pain, and radiating pains, or zone of hyperæsthesia, which are due to the irritation of the sensory nerves in the lowest part of the upper segment. It is desirable to know the symptoms that occur when a transverse lesion is at different heights in the spinal cord. They may readily be ascertained by an examination of the table given at p. 152. There is, however, some uncertainty as to the effect on sensation in the limbs, of disease at different parts of the lumbar and cervical enlargements, since, as we have seen, the exact representation of sensation in the cord is still uncertain.

The upper limit of the lesion is shown by the upward extent of the motor and sensory loss, according to the first three columns. The lowest nerves supply the anus and perineum. Those that supply the skin and muscles of the leg and foot arise from the fourth lumbar to the third sacral segments, and are damaged by a lesion involving the lower part of the lumbar enlargement. We must, however, remember that the skin of the inner part of the leg is not supplied from the sacral nerves, and so may escape when the outer part of the leg and back of the thigh have lost sensation. In the middle of the lumbar enlargement the nerves arise which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip muscles supplied by the sacral plexus, the glutei, quadratus, and gemelli, and for the skin of the lower part of the gluteal region. These parts then will be paralyzed by disease in the middle of the lumbar enlargement, while the muscles and skin in front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, etc.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the second and third lumbar nerves arise, and then the flexors of the hip become paralyzed. In proportion as the disease is higher in the dorsal region, we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment—first, of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the tenth dorsal nerves, and the ensiform area to the sixth and seventh. When the disease reaches the lowest part of the cervical enlargement (the first dorsal nerves), we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the fifth, sixth, seventh cervical), the shoulder muscles and the serratus magnus become paralyzed, and there is general loss of power and sensation. (For details see table, p. 152, and also p. 139.) Above the level of the sixth pair, the trapezius and sterno-mastoid become somewhat weakened, for the fibres of the spinal accessory which supply them undoubtedly arise in part from this region of the cord. At the fourth and fifth cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localization might cease, for total transverse lesions at this spot necessarily cause death. For a little time the sterno-mastoids and scaleni can still get some air into the chest, rarely in sufficient amount to maintain life for more than a few days. But limited lesions may occur higher up, and then we have complete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the fifth nerve.

The extent downward of the lesion, its vertical extent, is thus not indicated by the impairment of the conducting functions, by the motor or sensory paralysis; to ascertain it we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralyzed region—to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are injured. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes, and the relation of each to the cord, are shown in the third column of the table; the myotatic contractions are printed in italics in the position which corresponds to the centres and nerve roots which are essential for their production. Excess of superficial reflex action indicates withdrawal of the cerebral controlling influence of the reflex centres, and marked excess of the muscle reflexes suggests the existence of a descending degeneration in the lateral columns, since it implies impaired function of the lowest part of the pyramidal tracts.

Unilateral lesions interrupt the motor path to the muscles on the same side as the lesion, causing one-sided palsy, termed "spinal hemiplegia" when the disease is so high as to affect both arm and leg, "hemi-paraplegia" when it is lower, and affects one leg only. There is often some loss of power on the opposite side, which may be due either to slighter damage to the other side of the cord (since few lesions are strictly unilateral) or to damage to non-decussating pyramidal fibres. Conversely, the paralysis of the leg may be incomplete when that of the arm is complete, owing to the escape of the fibres for the leg which cross lower down the cord. Sensation is affected on the opposite side, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs somewhat above the entrance of the nerves. The upper level may vary for different forms of sensibility, in consequence probably of the level of crossing (in relation to entrance) being different for the several paths. A lesion in one side of the lumbar enlargement often affects sensation on the same side as motion, because it damages the sensory path before it has crossed. In all cases of crossed motor and sensory paralysis, the sensibility of the muscles differs from the other forms of sensibility, and if it is affected on one side, this is the side of the motor palsy and not of the cutaneous anæsthesia.*

The crossed affection of cutaneous sensibility may involve all forms of sensation, or only some of them. Sensibility to pain is almost invariably impaired. The temperature sense is usually affected with that for pain; in only two of twenty recorded cases (in which the affection of sensibility was carefully noted) was the sense of temperature normal, and that of pain impaired, and in neither of these cases was the sensibility to pain actually lost. On the other hand, in one-third of the cases tactile sensibility was unaffected and in about one-tenth of the cases it has been impaired on both sides.

Cutaneous sensibility is sometimes impaired in a zone at the level of the lesion, and on the same side, in consequence of the damage to the nerve roots entering the cord, and above this there may be a narrow band of hyperæsthesia from irritation of the roots at the upper part of the lesion. On the side of the lesion, below the anæsthetic zone, there is, in most cases, a remarkable hyperæsthesia, corresponding in distribution with the anæsthesia on the opposite side. Painful impressions are felt most acutely, and even a touch on the skin, or warm or cold bodies, produce pain. The cause of this hyperæsthesia is obscure. Both it and the opposite loss of sensibility may last for twenty years, and it cannot be ascribed therefore to any irritation by the morbid process. It is perhaps due to an altered action of the cerebral centres, on the opposite side of the brain. There is much evidence of an intimate connection between the sensory centres on the two sides, and it is conceivable that the altered functional state of the centre on the side of the lesion, to which impressions cease to come, may induce in the opposite hemisphere a condition expressed as hyperæsthesia. The condition may be thought of in relation to the peculiar phenomena of transfer in hysterical hemianæsthesia.

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* This was first pointed out by Brown-Séquard in his important study of these cases. Scepticism has been expressed as to the fact, based on a denial that the test commonly employed, recognition of posture, is significant. Experiments on animals, even monkeys, are inconclusive, but the evidence from cases in man is very strong, not only of the occurrence of the loss, but of the significance of the test. In some cases, moreover, the muscles were insensitive to pressure. An instance recently reported is the following: A stab in the dorsal region caused loss of cutaneous sensibility in the right leg and motor palsy in the left. On this left side, on which cutaneous sensibility was normal, the sense of posture was absolutely lost, while it was present on the right side, on which cutaneous sensibility was impaired. The patient often thought the left leg was flexed when it was extended. Ultimately this leg presented distinct ataxy. (Gilbert, *Arch. de Neurologie, 1882*, p 275.)

Reflex action is increased in all its forms on the side of the lesion, but the increase only occurs after some days. At first it is lessened or even abolished, no doubt from the inhibitory influence of the irritation of the morbid process.

The side below the lesion is at first, for some days or even weeks, warmer than the other, the difference being usually about a degree Fahrenheit. In the course of time this difference passes away and the side may even be colder than the other.

These symptoms may be shown in the form of a table:—

Zone of cutaneous hyperæsthesia. Zone of cutaneous anæsthesia.	LESION.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened, then increased. Temperature raised.		Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

The vertical distribution of the symptoms varies according to the vertical position of the lesion. Their relations are best marked when the lesion is in the dorsal region of the spinal cord. When it is in the cervical or lumbar enlargement the upper limit is less distinct because of the complex representation of skin and muscle in the nerve roots.

GENERAL PATHOLOGY AND ETIOLOGY: INDICATIONS OF THE NATURE OF THE LESION: PATHOLOGICAL DIAGNOSIS.

The kinds of disease to which the spinal cord is liable are not specifically numerous, but those which are common present several varieties. The processes are congestion and anæmia, hemorrhage, inflammation, degeneration and growths. Hemorrhage and growths are, on the whole, rare. Congestion and anæmia play an uncertain part in the production of symptoms; their influence has been certainly overrated by some writers, and perhaps under-estimated by others. The most frequent lesions are inflammation and degeneration. These two processes, distinct in their typical forms, blend in their intermediate varieties. Inflammation varies much in character and course. Its effects are, in some cases, produced with great rapidity, in other cases with extreme slowness. It probably commences in most instances outside the nerve elements, which are secondarily involved. Perhaps some acute processes in the nerve elements themselves are most accurately regarded as a parenchymatous inflammation. Degeneration consists of two processes, a wasting of the nerve structures, cells and fibres, and an overgrowth of the connective-tissue elements. The latter leads to the condition termed sclerosis. The name has been given to it, probably, because the process is one that, in other organs, increases their consistence. In the cord it has not often this effect. The newly-formed connective tissue is rarely of greater firmness, and is often less firm, than the nerve elements it replaces. In degeneration we must distinguish two types. In one the process is limited to structures that have the same function; the degeneration probably begins in the nerve elements, and the overgrowth of connective tissue is consecutive. The so-called "secondary degenerations" are of this character; but similar changes are often primary. In the other type the degenerative changes are not distributed according to function. They are apparently random in incidence, and probably begin in the connective tissue outside the nerve elements, which suffer secondarily. Insular sclerosis is an example of this type. It is this form which presents the chief gradation to chronic inflammation.

Softening of the spinal cord is very common. Whenever the nerve fibres are broken up into disconnected globules of myelin, separated by serum in a sort of emulsion, the consistence of the part is necessarily lessened. Inflammation always causes such a breaking up of the nerve fibres, and hence inflammation always causes softening as its first effect. Ultimately, connective-tissue elements are formed, which increase the consistence, it may be up to, and even beyond, the normal degree. In a very chronic inflammation the formation of new tissue may proceed *pari passu* with the destructive process, and there may be, at no time, much diminution of consistence. It is in these cases that it is difficult to draw the line between inflammation and degeneration. Does such softening of the cord occur apart from inflammation? The question is not easy to answer. In the brain, necrotic softening is very common as a consequence of arterial obstruction—is indeed the common form of softening. But in the spinal cord we have no evidence of the occurrence of necrotic softening. If embolism occurs, it is excessively rare. Arterial thrombosis, due to atheroma of the walls of the vessels, such as is so common in the brain, probably does not occur in the cord. The arteries are smaller than those in which atheroma is met with in the brain, and the lowness of the blood pressure within them involves the absence of the chief cause of atheroma. It is possible that spontaneous thrombosis may sometimes set up the changes that are now regarded as those of primary inflammation, but we have no direct evidence of this. The question will be further considered in the account of inflammation.

Besides the morbid processes to which the cord itself is liable, it suffers also in consequence of disease outside it. It may be compressed by growths, and by products of inflammation, within the spinal canal. Compression not only causes acute degeneration of the nerve elements, but usually excites actual inflammation. This "compression myelitis" may attain a degree and an acuteness out of all proportion to the causal compression. The membranes may be the seat of hemorrhage which compresses the cord, or inflammation which irritates it, even when the organ is not invaded in appreciable degree.

The series of recognized lesions of the spinal cord, obvious or microscopic, does not by any means exhaust the list of its morbid states. Changes may occur in the nutrition of its elements interfering with their function, which are, and are likely to remain, altogether beyond our means of detection. Such morbid states are often called "functional diseases," but when prolonged they are probably more accurately conceived as nutritional diseases (see p. 25). Derangements of function that we can regard as purely such are few and rare. It is thus convenient to think of the pathological conditions of the cord under the four types which we considered (p. 26) as presented by diseases of the nervous system generally. The types are not always sharply separated, but are not therefore less useful. (1) *Organic disease,* conspicuous to naked-eye examination, such as a hemorrhage or tumor. (2) *Structural disease,* presenting only change in tint or consistence to the naked eye, but distinct tissue changes on microscopical examination. (3) *Nutritional disease,* causing no change recognizable even by the microscope. (4) *Functional disease,* purely such.

In connection with the general pathology of diseases of the spinal cord, it is important to note certain general facts of their causation: (1) An inherited tendency to disease of the nerve elements, manifested by such affections as epilepsy and insanity. This case is chiefly influential in producing the structural and nutritional diseases, beginning in the nerve elements, and especially, among structural diseases, the "system degenerations" as they are called. (2) Injury.—A severe concussion of the cord may cause (*a*) instant grave damage, usually hemorrhage. Or (*b*) it may cause no immediate effect, but symptoms may come on at the end of a few days and progress slowly or rapidly. Such symptoms are usually due to inflammation, simple, or secondary to minute spots of injury. Lastly (*c*), the concussion sometimes seems to pervert the process of nutrition in the nerve elements. Slow symptoms of impaired function may result, and these may progress until, after months or years, there is actual structural disease. (3) Exposure to cold.—Wet cold is especially effective. The most frequent effect of a severe exposure is acute inflammation, but habitual exposure may produce chronic inflammation or degenerative disease. The exposure that is effective may be general, or chiefly of the feet and legs, occasionally of the back. (4) Syphilis.—The demonstrated ways in which syphilis interferes with the spinal cord are (*a*) by a syphilitic growth, compressing or invading the cord, and (*b*) by chronic syphilitic meningitis, damaging the cord and the nerve roots. But (*c*) acute and chronic inflammations of the cord are often met with in syphilitic subjects, and have been thought to be, in many instances, of syphilitic origin. It is doubtful whether they present any syphilitic characters, *i. e.*, any histological features by which they differ from non-syphilitic inflammations. The evidence of dependence on syphilis is stronger in the case of chronic and subacute disseminated inflammation than in the case of acute myelitis. Lastly (*d*), certain degenerative diseases of the cord are very commonly preceded by syphilis; one of them, locomotor ataxy, so frequently that a causal relation between the two can scarcely be doubted. But these degenerative diseases are certainly not syphilitic in nature; they differ in no respect from the similar morbid processes that occur in individuals who have not had syphilis.

The outline just given of the general pathology and etiology of diseases of the spinal cord will enable us to consider the last element in diagnosis, the nature of the lesion. The seat of the disease is indicated by the combination of symptoms; its nature can only be determined by considering, separately and together, several other points: (1) The way in which the symptoms came on. (2) The causes that can be traced, taken in conjunction with the known effects of those causes. (3) The seat of the disease, taken in conjunction with the known liability of certain structures to certain lesions. The most important of these elements is the mode of onset, and the other indications should only be used in strict subordination to this.

The time occupied by the onset of the disease is thus the first element in the pathological diagnosis. By "time of onset" is meant the period that elapses between the actual commencement of the symptoms and their attainment of a considerable degree of intensity. We may divide the chief modes of onset into five classes, and classify the most common diseases in relation to them, in the following table:—

Disease.	Onset.	Disease.
	Sudden (few minutes)	Vascular lesions.
	Acute (few hours or days)	
Pressure and growths	*Subacute* (one to six weeks)	Inflammation.
	Subchronic (six weeks to six months)	
	Chronic (more than six months)	Degeneration.

A lesion of sudden occurrence, the symptoms developing in the course of a few minutes, is almost always vascular, commonly hemorrhage, sometimes perhaps vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a few weeks. Subacute and chronic inflammation occupies from a few weeks to a few months. Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by tumors which invade or compress, and by simple pressure (traumatic causes included) are never sudden or very acute, and rarely very chronic, the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more morbid processes may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and, on the other hand, in degenerated tissues, sudden vascular lesions occasionally occur. Pressure often produces local myelitis, which may be acute or subacute in its development. Cancer of the vertebræ, for instance, often causes rapid myelitis. The whole course of the disease must be ascertained before an inference is drawn, and the possibility of a double process must always be kept in view.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset that has been ascertained. This indication involves a knowledge of the various diseases and their seat. The most important consideration is that a wide range of symptoms of uniform character indicates the affection of a definite system of structure, and in most instances a disease commencing in the nerve elements, and if the onset be chronic we may feel sure that it is a degeneration. On the other hand, the involvement of many functions suggests a random process, such as inflammation or pressure. But this indication is always to be subordinated to the mode of onset. Thus the limitation to a single structure does not exclude inflammation: this may affect, for instance, the anterior gray matter only, and cause corresponding symptoms.

The symptoms may indicate a morbid process limited to one-half of the cord, but this does not materially modify the diagnostic method. Almost any process may, in rare cases, be thus limited. System degenerations and acute inflammations are least frequently unilateral, and they never reach a considerable degree of intensity on one side without some affection of the other side. On the other hand, tumors and foci of chronic myelitis are often one-sided, and still more often affect one-half of the cord first and then the other.

Indication of disease outside the cord, irritation of certain nerve roots, causing severe local pain, often precedes the symptoms of compression and is an important aid to diagnosis. It shows the existence of a morbid process outside the cord before the cord is involved. But we cannot use even this indication except in dependence on the mode of onset. A disease, as a growth outside the cord, may, as we have seen, not only compress the cord, and cause slow loss of power; it may excite inflammation and cause rapid palsy.

The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the disease to certain possible forms of lesion; the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause and the knowledge of the lesions that cause produces may help us to carry the diagnosis still further. The most important general causes of disease of the cord, and the processes to which they chiefly give rise, have been already mentioned. The causal element in diagnosis is chiefly an application of those facts.

It would be easy to formulate more detailed diagnostic indications. Those given will suffice to indicate the general principles of diagnosis; to do more than this would be to hinder, rather than to help. The only sure ground for diagnosis is a thorough knowledge of the various diseases and their symptoms; without this elaborate formulæ are useless; with this they are unnecessary.

The distinction of functional and nutritional disease from organic lesions may conveniently be postponed until the symptoms of the former are specially described.

SPECIAL DISEASES OF THE SPINAL CORD.

DISEASES OF THE VERTEBRAL COLUMN.

Diseases of the bones of the spine fall for the most part within the province of surgery. But there are few of these diseases that do not, among their most frequent effects, interfere with the functions of the spinal cord. Hence an account of the diseases of the cord would be incomplete without some mention of the morbid states that begin in its bony case.

INJURIES OF THE SPINE.

Injuries to the spinal column may consist of punctured wounds (which need not be considered here), concussion (the effects of which on the cord will be considered later), and fracture or dislocation of the vertebræ. A brief mention of the two last conditions may be given, the reader being referred, for fuller information, to surgical treatises.

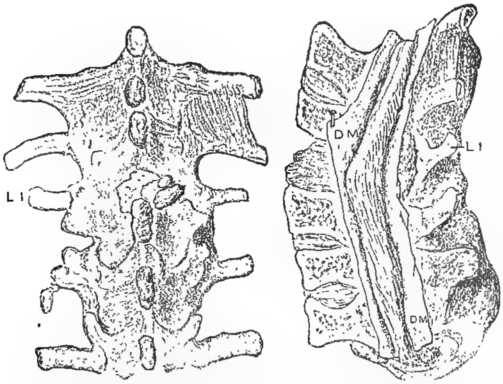
Dislocation.—Simple dislocation occurs in the cervical region, most frequently at the fifth and sixth vertebræ. It may take place gradually or suddenly: gradual displacement is always secondary to disease of the bones; sudden displacement may occur in disease or from injury. The damage to the cord is always greatest in traumatic displacement of normal bones, because the force needed to produce the dislocation is much greater, the displacement is more considerable, and the obliteration of the canal is more complete. Displacement may occur in any direction: it usually involves both vertebral articulations, but, in rare cases, it is oblique, involving one articulation only. The common causes are violent blows or falls on the head, rarely sudden rotation of the head when a weight is carried upon it. The symptoms are a lateral or forward or backward displacement of the head, so that the chin is in contact with the shoulder or the chest, or the occiput with the nape of the neck. The irregularity of the vertebral spines is usually readily detected. The cord is damaged in most cases, and the symptoms are those of a total transverse lesion in the situation of the luxation. It may be merely compressed, especially in cases of disease, in which the displacement has occurred with little force. The symptoms of paralysis have been known to pass away, in such a case, on the reduction of the dislocation. More commonly the cord is also bruised, with extravasation of blood, and secondary myelitis occurs later. In such cases, if the patients live, there may be anæsthesia or hyperæsthesia below the lesion, with total paralysis of the limbs, and excess of reflex action. In rare instances the cord has been completely divided. In still rarer instances of slight displacement it has not been injured.

Rupture of the transverse ligament, which retains the odontoid process, may permit the latter to compress the cord, and thus to cause instant death. This often results from sudden suspension by the head, as in criminal executions. One of the curiosities of surgical literature is a case related by Petit in which a man, playing with a neighbor's child, lifted it up by the head, and caused instant death by rupturing the transverse ligament. The father of the child, entering at the moment, stabbed the man with a knife, the blade of which passed in between the first and second cervical vertebræ, divided the spinal cord, and the man also fell dead. Rupture of the ligament has also resulted from raising a heavy weight with the head. The treatment of dislocation is too purely surgical to be described here.

Fracture.—All organic diseases of the bones, weakening them, predispose to fracture. Apart from disease, the accident is most common in adults, the greater elasticity of the vertebral column in children giving to them a comparative immunity. Its great cause is a blow or fall on the spine, or sudden forcible flexion. In extremely rare cases a fracture, usually slight, has resulted from a severe muscular exertion. This is an important fact, showing that muscular exertion may injure the spine. Symptoms of such injury, in slight degree, are not uncommon.

Fracture may occur at any part of the spine, but is most frequent at the fifth or sixth cervical, and at the last dorsal or first lumbar vertebræ. In the dorsal and lumbar regions, the bodies are fractured in two-thirds of the cases, but in the cervical regions, the arches suffer alone in one-half. Usually there is a displacement of the vertebral column at the seat of fracture. Rarely the bodies may be crushed without displacement. The displacement of the bone involves a narrowing of the canal, and, usually, compression of the cord. This may also result when the arches only are driven in. But the cord may be seriously damaged when there is no permanent narrowing of the canal, as in Figs. 76 and 77. The dura mater is rarely torn except by a splinter. Blood is almost always extravasated outside the dura mater, often in considerable quantity, from the rupture of the large veins in this situation. There are usually only small extravasations in the pia mater. The cord is, in most cases, bruised and compressed by the lower fragment (Fig. 78). Sometimes it is flattened, and it may even be divided, all nerve substance being squeezed out of the pia matral sheath at the spot. In the case shown in Figs. 76 and 77, the cord appeared to have been split longitudinally at the spot. Blood is extravasated into the bruised part, sometimes in minute spots, sometimes in larger hemorrhages, which may occupy longitudinal cavities. These changes are usually limited to the spot compressed. Secondary myelitis is set up in the damaged part, and this sometimes extends beyond the contused area. In cases of some duration the usual ascending and descending secondary degenerations are also found. Fig. 67, p. 131, represents sections of the cord from the case of fracture figured above. Occasionally the injury leads to secondary caries of the bone, with all its consequences.

Fig. 76.



Fracture of the first lumbar vertebra. D M. Dura mater. There was no permanent narrowing of the canal, but, nevertheless, the spinal cord was greatly damaged at the spot; see next figure. (*From a drawing by Mr. V. Horsley.*)

Three classes of symptoms result. The first consists of the local indication of the injury. Secondly, there may be certain nervous symptoms, not distinctly due to the damage to the cord. One of these is general shock, which may be so great as to entail transient loss of consciousness. Vomiting occasionally occurs. There is great pain in the position of the fracture, rendered very intense by pressure, and often radiating along the nerves which come from this part, the roots of which are compressed. Tetanus occasionally ensues, and has been ascribed also to the irritation of the nerves. In rare cases epileptiform convulsions have followed fracture, usually at an interval of some days.

Fig. 77.

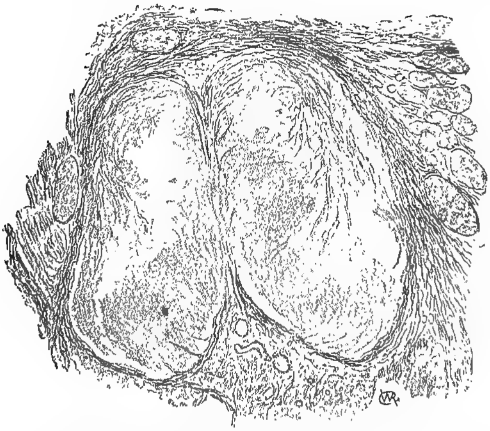


Fig. 78.

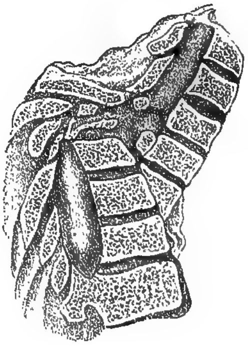


Fig. 77.—Spinal cord damaged by the fracture shown in Fig. 76. The elements of the cord itself are changed beyond the possibility of identification. The ascending degeneration is shown in Fig. 67.
Fig. 78.—Fracture of the body of the fifth dorsal vertebra and of its processes. (*After Gurlt.*)

The third group of symptoms are those which result from the damage to the cord, and consist in paralysis of the parts below the injury. Its character depends on the amount of damage. If this is considerable, there is both motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Reflex action is lost at the level of the lesion, and the examination of the trunk reflexes often gives important information regarding the extent of the damage, when this is in the dorsal region. Below reflex action is commonly in excess, unless the centres are impaired by descending myelitis. Spasmodic twitchings sometimes occur in the limbs immediately after the injury, accompanied by priapism. The pains are severe in the arms when the fracture is opposite the cervical enlargement and in the legs when at the lumbar enlargement, or below the latter, so as to damage the nerve roots. In these cases there may be rapid wasting of the muscles, with loss of electric irritability. There is usually at first incontinence, afterward retention of urine, but the former is persistent if the lumbar centres are damaged. Cystitis, bedsores, etc., may supervene. Ultimately, if the damage is above the lumbar enlargement and the patient lives, there may be increased myotatic irritability of the limbs, increasing to spasm, so that spastic paraplegia results.

Special symptoms result when the injury is in certain parts of the spine. Fracture of the *first two cervical vertebræ* causes instant death, unless the displacement is very slight, and even then there is imminent danger of further displacement, with the most serious consequences, on any incautious voluntary movement. With slight displacement persons have been known to live for weeks, and then die from secondary myelitis; they have even recovered altogether. Now and then there is no compression of the cord, although there is distinct displacement, even sufficient to be recognized in the pharynx (Leyden). In such a case death has resulted at a later period from caries. The characteristic symptoms are local pain, increased by all movements (which are thus rendered impossible), displacement, and spinal symptoms. The latter may be slight—merely difficulty in breathing or swallowing—or considerable, and involving the trunk and limbs. Sometimes there is hyperpyrexia. Not more than one case in fifty recovers.

Middle Cervical Vertebræ.—The third, fourth, and fifth vertebræ are most frequently fractured. When the injury to the cord is considerable, death usually occurs very rapidly, because the roots of the phrenic nerve are involved. In some cases there is little immediate displacement, and the symptoms are slight until further displacement occurs in some movement. Thus a man who had met with an injury of this kind went to be shaved; during the process, his head was turned on one side by the barber, with the unexpected result of causing displacement of the fracture, and immediate death. When the fracture is at the *cervico-dorsal* region, opposite the lower part of the cervical enlargement, the arms frequently escape at first, the early paralysis being confined to the legs and muscles of the trunk. Respiration is diaphragmatic only. After a few days the arms become involved, but their paralysis is often partial, affecting, for instance, only certain muscles, as the extensors of the hand, and it is often accompanied by local spasmodic movements. Movements and pressure cause pain, and there is local muscular rigidity. The head may be in normal or in abnormal position. Vasomotor disturbance in the face and general hyperpyrexia have been observed.

In fracture of the *dorsal vertebræ* (2–11) the arms escape, the legs are paralyzed, and the trunk muscles up to the height of the lesion. The pain in the trunk may be very severe. There is hyperæsthesia or anæsthesia in the parts below. The reflex action in the legs is excessive; that in the trunk is abolished at the level of the lesion. The *last dorsal* and *first lumbar* vertebræ are often fractured, and there results paralysis of the legs, complete or irregular, severe pains, tingling, etc., sometimes followed by hyperæsthesia or loss of sensibility, and by rapid disturbance of nutrition, in both the muscles and the skin. In fracture of the *lower lumbar vertebræ* the symptoms are often slight; below the extremity of the cord the nerves occupy a smaller space in the canal, and so may escape compression by a moderate displacement. The fractured vertebræ unite very slowly. A false joint is occasionally formed. If the patient recovers from the immediate effects he often dies from secondary myelitis and its consequences.

CARIES OF THE SPINE.

Caries of the bones of the spine is a frequent cause of paraplegia. It is often termed "Pott's disease," from the English surgeon, Percival Pott, who first described it (in 1779) as a cause of paralysis.

CAUSES.—Males are said to be rather more liable than females, but the difference in sexual incidence is not great. It is more common in childhood (after three) and next in early adult life, but it may occur at any age, and is perhaps more common in the second half of life than any other scrofulous lesion. I have known it commence at fifty, and it has been met with as late as seventy. It is distinctly a manifestation of the tubercular and scrofulous diathesis, and evidence of such inheritance is to be traced in most cases. Occasionally the sufferer himself presents such indications, lung disease, etc., or caries of other bones. It occasionally develops simultaneously with other signs of acute tuberculosis. Caries has been thought to result from syphilis, but this cause is certainly rare.

Injuries seem frequently to excite the bone mischief in the spine, as they certainly do analogous bone disease elsewhere, in those who are predisposed, possibly, sometimes, in healthy persons. Falls, blows on the back, and severe strains, are the most frequent traumatic antecedents. The last may act by straining the ligaments, and setting up inflammation which spreads to the bones directly or through the intervertebral cartilages. There is usually an interval, sometimes of many months, between the injury and the definite symptoms of bone disease. Adjacent inflammation, secondarily affecting the bone, is a rare cause. Caries of the cervical spine has thus been secondary to a primary retropharyngeal abscess. The disease sometimes follows septicæmia in a way that suggests a secondary abscess in the bone. This was the case in a surgeon about sixty years of age, in whom symptoms of blood poisoning followed an injury to the ankle. These were succeeded by paraplegia of rapid onset, and this by the development of angular curvature in the lower dorsal region. In a large number of cases no exciting cause can be traced.

Pathological Anatomy.—The changes in the bones need not detain us, since they belong to surgical pathology, except in so far as they lead to damage to the cord. There is inflammation of the bodies of the vertebræ, and of the intervertebral substance, at first with enlargement, afterward with breaking down of the substance of the bone, which gives way under the pressure to which it is exposed, so that deformity of the spine results. Pus often accumulates outside the spine, and may burrow in various directions, according to the seat of the disease.

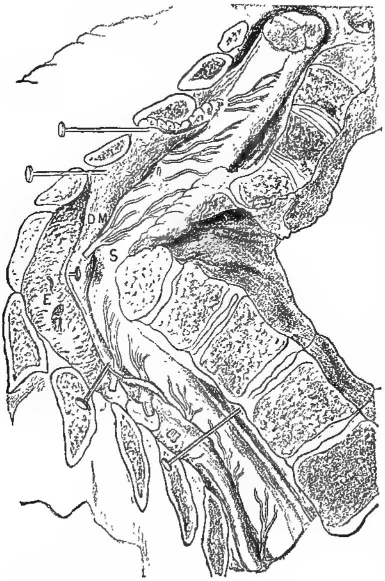
The inflammation extends to the loose cellular and adipose tissue between the bone and the dura mater, and often to the dura mater itself, the outer layer of which becomes irregularly thickened in the neighborhood of the disease (see Fig. 80). The inner surface of the dura mater is often normal when the outer surface is much changed. Inflammatory products, cheesy material, pus, etc., accumulate between the bone and the dura mater, and often in such quantity as to compress the cord. Inflammation is frequently set up in the cord, but usually only when it is also compressed.

The displacement that results from the collapse of the bodies varies much according to the extent and character of the disease. Occasionally there is simply a lateral displacement—one spine is situated a little to one side of that above it. More commonly the collapse of the bodies leads to "angular curvature," the spinal column is bent forward at an acute angle, and one or two vertebral spines are much more prominent than the others. There is often, however, a less abrupt bend; the curvature may extend over four or five vertebræ.

The *nerves*, as they pass through the membranes and intervertebral foramina, are irritated by the inflammation and often compressed by the thickening of the dura mater which sheaths them. Those passing by the seat of compression may also suffer from the narrowing of the canal. They may be found red and swollen, or shrunken and gray. They may be damaged when the cord is normal, or but little affected when this is compressed.

The damage to the *spinal cord* depends on the secondary consequences of the caries, and both its occurrence and character are variable and uncertain. The mechanism of the damage is twofold—compression and inflammation. The relation between the two is considered more fully in a subsequent chapter ("Compression of the Spinal Cord"). There is never compression without inflammation, but it is probable that, in many chronic cases, the inflammatory changes are secondary to the damage to the nerve elements. On the other hand there may be inflammation when there is no compression. The mechanism of compression varies. The most frequent is the collection of inflammatory products outside the dura mater, and the thickening of this membrane. Less commonly the cord is compressed by the displacement of the bone, or by fragments of bone that are pushed into the canal. Often both these causes are influential, as in the case shown in Fig. 79. Probably an inflammatory swelling or abscess of the bone is sometimes the mechanism of compression, since the signs of pressure have disappeared when an abscess has formed outside the spine, or even when deformity has come on. The inflammatory changes are most intense at the point of greatest compression, but extend, lessening in degree, through a few inches of the cord above and below the lesion. They are described in the chapter on "Compression."

Fig. 79.



Caries of the spine, mid-dorsal region. The spinal cord is much narrowed and discolored at S from depression between the displaced bone and a mass of inflammatory products, E, outside the dura mater, D M, the inner surface of which is normal. (*After Leyden.*)

Symptoms.—Caries of the spine causes symptoms of three classes: (1) those of the bone disease; (2) the effects of damage to the nerve roots; (3) those due to the changes in the cord itself. Only the symptoms due to the damage to nerves and cord come within the special province of this book, but the bone symptoms are of much importance, as on them the diagnosis of the nature of the nervous symptoms often depends.

These vertebral symptoms are various. The first is pain in the spine, chiefly felt at the affected spot, often increased by movement, and especially by pressure on the bone. The local tenderness is a very important sign. It is elicited not only by direct pressure on the spines, but by lateral pressure; if they are grasped and pressed to one side considerable pain is usually produced. The increase of pain by movement is greatest when the disease is in the more mobile parts of the vertebral column, especially when it is in the cervical region. Movement of the head occasions pain, and there is an instinctive fixation of the head, which is sometimes inclined to right or left, less commonly backward. It may thus produce the aspect of torticollis, which differs from that due to muscular contraction in that the sterno-mastoid is tense on the side toward which the head is turned, the muscle being simply stretched by the deviation of the head.

The deformity of the spine is a later symptom than the tenderness, and usually comes on gradually. Its characters have been already described. It is often absent when the disease is in the cervical region. In this part there is usually another symptom, thickening of the tissues about the spine. This is rare in other parts. In any region an abscess may form in the neighborhood of the disease. Those which come backward, or descend to the groin by the psoas muscle, can be recognized externally. Those that form in front of the diseased vertebræ may give rise to symptoms that are puzzling, if the existence of spinal caries is not known. Thus a retropharyngeal abscess may cause difficulty of deglutition, and one in the dorsal region may cause symptoms of œsophageal obstruction, etc.

Increased pain and tenderness in the vertebral column often precede the symptoms of damage to its contents. The symptoms due to interference with the *nerve roots* are very variable, and are often absent. They are both sensory and motor. There is pain along the course and in the distribution of the nerves, and hyperæsthesia of corresponding extent, sometimes with spots of anæsthesia. There is also muscular weakness, and sometimes muscular wasting. These symptoms are more fully described in the chapter on "Slow Compression." Here it may suffice to say that they are conspicuous only in a minority of the cases, and chiefly occur when there is pachymeningitis. The motor symptoms are most marked when the disease is in the cervical region, and the nerve roots for the muscles of the arms are damaged, since slight and limited impairment is more readily recognized in the arms than in the trunk. If the disease is in the highest part of this region, the pains may be referred to the occiput. Herpes zoster has been occasionally met with along the course of the irritated nerves. Spasmodic contraction in the muscles supplied by the affected roots is extremely rare in cases of caries. Reflex action is abolished in the affected parts, and the change in the superficial reflexes of the trunk sometimes gives important diagnostic information. Disease of the lower cervical roots may cause symptoms of deranged action of the sympathetic on that side of the head, occasionally shown in the pupil, more often in the vessels. I have seen persistent sweating on one-half of the forehead from this cause.

The symptoms of interference with the function of the spinal cord itself are chiefly impairment of its conducting power, causing paralysis in the parts below the lesion. As the disease is most frequently in some part of the dorsal region, paralysis of the legs is the common effect. But the symptoms vary in their characters, according not only to the position of the disease, but also to the immediate process of damage to the cord. The signs of caries may have existed for years before paralysis comes on. Angular curvature may even come on in early childhood, and paralysis not till adult life. More commonly the paralysis comes on one or two years after the curvature; sometimes at the end of a few months. On the other hand, the two classes of symptoms may manifest themselves at the same time, and the symptoms of cord disease may even develop, and reach a considerable degree, before the local symptoms are sufficiently marked for bone disease to be suspected. In one patient, after absolute paralysis of the legs had existed for six months, an experienced surgeon doubted the existence of bone disease, but a few months later the signs of this were indubitable.

The cord symptoms vary much in their mode of onset. Usually there is no exciting cause, but sometimes a strain of the back or exposure to cold seems to excite a change in the condition of the bones, in consequence of which the cord suffers. It is easy to conceive that, when the conditions are favorable, a very slight strain may be effective. In one case, the first symptoms followed instantly three violent sneezes. When the cord symptoms have commenced, they may develop quickly or slowly. As instances of chronic onset may be mentioned cases in which the symptoms reached a considerable degree of intensity at the end of nine months, four months, and two months, after their commencement. But the onset is sometimes much more rapid; in one case there was complete paraplegia at the end of three weeks. Sometimes it is still more acute, and in such cases, myelitis rather than pressure is probably the effective agent. Thus, in one patient, a child of three, slight weakness existed for three weeks, and then the power of standing was lost in a single night. In another child, aged eight, who had presented, for two years, indications of disease of the cervical vertebræ, the power of moving the legs was lost in the course of twenty-four hours; during the second day the left arm became paralyzed, and, at the end of a week, the right arm. Very rarely the onset is instantaneous, probably from sudden displacement, since hemorrhage has not been proved to occur. A child with angular curvature was walking across the room, when she fell, and, on being lifted up, the legs were powerless.

Both legs usually suffer together; rarely one is paralyzed before the other; very rarely one leg suffers much and the other little or not at all. Thus in one case angular curvature developed in childhood; at sixteen there was an attack of weakness in the legs, which passed away at the end of three weeks. At 17½ the patient sprained his back; pain in it followed, and six weeks later the right leg gradually became weak, and a year afterward presented intense spastic paralysis, the left leg being very little affected. He ultimately recovered. An instance in which one leg became affected before the other is a case already alluded to. A woman forty-five years of age suffered from pain in the spine, and one day, when walking, sneezed three times, and immediately felt "pins and needles" in the right knee, and presently in the foot. The leg became almost powerless during the next three days. A fortnight afterward, she felt similar tingling in the left knee, and, at the end of another week, in the foot, and during the next three weeks this also lost power, so that at the end of six weeks from the onset both legs were motionless. The diagnosis was verified on her death, six months later.

The characters of the paralysis in relation to the seat of the disease are described in the chapter on "Compression." In the most frequent cases the dorsal cord is damaged and the condition of the legs is that of spastic paraplegia. If it is situated in the cervical region there is muscular atrophy in the arms, sometimes combined with palsy without atrophy, according as the disease is at the level of the cervical enlargement, or above this. There is the same spastic paralysis in the legs as when the disease is in the dorsal region. When, as is commonly the case, the arms suffer from damage to the nerve roots, they suffer before the legs, but if the disease is so high up that the arms suffer from the damage to the cord itself, the legs may be paralyzed before the arms, as in the case of the child mentioned above. In this case the muscles of the shoulder were wasted, the disease being near the level of their nerves, while the forearm muscles were not wasted. In this case, as in others of similar seat, the diaphragm was paralyzed. The muscular part of the spinal accessory may be involved, and the power of supporting the head may be almost lost. Very rarely, from disease of the highest cervical vertebræ, the other nerves of the medulla are implicated.

Sensory symptoms due to the disease of the cord are less common than motor palsy. As a case already mentioned shows, the onset of paraplegia may be heralded by subjective sensations in the legs. Dull aching pain in them is not uncommon. Often there is no anæsthesia; in other cases there are various degrees of loss of sensation. Touch or pain may be lost alone, or there may be absolute loss of sensibility up to the level of the lesion. Reflex action in all forms is excessive (unless the disease involves the lumbar enlargement), and great excess of the superficial reflex action is a common and important feature. The legs are often cold and sometimes perspire continually. The sphincters are often affected, sometimes early, but they may escape even when there is a complete motor palsy of the legs.

Complications.—Among common complications are bedsores, cystitis, various secondary effects of the bone disease and general tuberculosis. In very severe cases peculiar secondary mischief has occurred in the spinal cord, and has run an independent course, giving rise to very anomalous symptoms.* Thus a descending myelitis may invade the lumbar enlargement in its entirety and abolish its central and reflex functions, causing rapid wasting of the muscles and acute trophic changes in the skin. Inflammation may *ascend* the pyramidal tracts and thus paralyze the arms. Ascending degeneration of the posterior median columns may spread to the postero-external columns and cause symptoms of ataxy. It is probable, also, that a similar affection of these columns may descend the cord, since ataxy may come on as the power returns, when the disease is in the dorsal region. Lastly, myelitis may occur in disseminated foci in various parts of the cord, and even in the medulla, giving rise to scattered symptoms of anomalous character.

Course.—The bone disease may heal, anchylosis occurring, or it may persist with continued formation of pus, or it may become quiescent and from time to time become active. The cord mischief is influenced by the state of the bone disease, although its progress may be to some extent independent. Thus inflammation in the cord may subside in spite of the progress of that in the bone. Pressure on the cord may be relieved, although the bone disease continues, and even sometimes as a result of the increased breaking down of bone and exit of pus by another channel. Hence there is no strict correspondence between the course of the bone mischief and that of the cord disease. In some cases the paralysis, motor and sensory, remains absolute. More often the sensory loss passes away, while motor paralysis remains, usually as spastic paraplegia, with muscular contractions due to posture. Life may be prolonged in that condition for years, but often bedsores form, or cystitis leads to kidney disease, and thus the patient dies, or the lessened respiratory power renders an attack of bronchitis fatal. In many cases, again, the paralysis gradually passes away even when the compression continues, and the cord is found considerably narrowed if the patient dies from some other cause. In children recovery occurs far more readily than in adults. Even in adults, however, recovery may occur from palsy that has lasted for some years with all the signs of descending degeneration in the cord. Usually rest on the back or mechanical supports are necessary to secure recovery, but it occasionally occurs without these measures. A youth acquired angular curvature at sixteen; at twenty paraplegia came on slowly, and progressed, with some variations, during the next seven years. There was then absolute motor palsy in the legs, and sensation was lessened. He continued to follow his occupation, a tailor, but took cod-liver oil and iron, and he gradually regained useful power, so as to be able to walk about. Such a case, however, is exceptional.

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* See Charcot, "Leçons sur les Mal. de Syst, Nerv.," tom. ii.

Relapses are apt to occur in cases that improve, although they are certainly not nearly so frequent as might be expected from the nature of the disease. In the majority of cases recovery is permanent. In a minority the paralysis returns when some exciting cause renews activity in the bone disease. The tendency to relapse and possibility of repeated recovery are very strikingly shown by the following case. In a girl of fifteen, paraplegia developed during nine months, slowly at first, more rapidly toward the end of that time. She came under my care six months later, having been unable to walk for eight months, to stand for seven, and to move her legs for six. Bone disease had not been previously suspected, but there was slight tenderness and enough lateral irregularity of the mid-dorsal spines to show the nature of the case. Rest in bed and tonics were soon followed by improvement; in six weeks she could stand, and even walk with help, and in four months was discharged, able to walk well. As she gained power, angular curvature came on, prominence of the seventh and eighth dorsal spines. Five months after her discharge, she fell and struck her back. The curvature increased, and her legs gradually became weak again. Seven months after the fall she was readmitted, unable to stand, although the paralysis was not absolute. There was foot clonus on each side. Sensibility was lessened below the ensiform cartilage. Rest on the back was again followed by slow improvement. In three months she could just walk. She was then suspended, and encased in plaster-of-Paris. At the end of another month she could walk about the room, and no clonus could be obtained. She was soon afterward discharged, and her progress continued, so that, at the end of nine months, she could walk five miles, and there was no trace of clonus, although there was still some excess of the knee jerk. She soon afterward married, and bore a child, which died two years and a half after her discharge. She caught cold at the funeral, and a fortnight later again began to lose power; in six weeks the legs were almost motionless, with marked foot clonus; sensation to touch was lost up to the umbilicus, that of pain being preserved. Neither rest nor encasement caused any improvement. After some months sulphide of calcium was given, and in a few days power began to return; in a month she could take a few steps, and in four months she could walk about the ward without difficulty. She made another good recovery. Some years later, however, paralysis again came on, and this attack proved permanent.

DIAGNOSIS.—When clear indications of caries precede the paralysis, the nature of the case can hardly be mistaken. The obvious inference, that the affection of the cord is secondary to that of the bone, is scarcely ever wrong. When the two develop together, mistakes in diagnosis are often made, but are usually due to the want of *repeated* examinations of the spinal column. It is when the root or cord symptoms precede distinct evidence of bone disease that the chief real difficulty in diagnosis occurs; the affection is apt to be mistaken for a primary disease of the cord or its membranes, a transverse myelitis when the dorsal region is affected, a progressive muscular atrophy, or primary pachymeningitis, when the disease is in the cervical region. A correct diagnosis can only be made in these cases by recognizing the significance of the slight bone symptoms that are always present, the deep tenderness and often slight irregularity. Even slight irregularity derives significance from tenderness, limited in extent, and corresponding in position to the deviation. The irregularity may be absent at first, and then its development is doubly significant. So, too, an increase in the amount of displacement may give significance to an irregularity that is no greater than is occasionally met with in normal spines.

The early excess of the cutaneous reflex action (from the sole, for instance), while not diagnostic, adds weight to the other symptoms of bone disease. It may be very marked, even while the patient is able to walk about. When root pains are present of definite character, they give additional significance to the other symptoms, since it is rare for a primary myelitis to cause more than a sense of constriction. Spots of anæsthesia along the course of the nerves are of still greater diagnostic value. All these symptoms derive additional weight from their coincidence in level with the deep spinal tenderness. The anæsthesia, with hyperæsthesia and bone tenderness, sometimes serves to distinguish the pains from those of intercostal neuralgia, with which they may be confounded at an early stage of the disease.

When damage to the cervical roots causes muscular wasting in the arms, the case may be mistaken for one of progressive muscular atrophy. The irregular distribution of the wasting differs from that common in the latter disease, and in this there are none of the sensory symptoms which are usually so pronounced in caries. The same symptoms may, however, be produced by a primary pachymeningitis, and the further distinction between the two rests on the presence of the signs of bone disease, on the absence of the causes of meningitis (of which syphilis is the most common), and on the youthful age of the patient; cervical symptoms are more likely to be due to caries if the patient is young than if he has reached adult life, and conversely they are more likely to be due to a primary pachymeningitis in adult life than in youth.

In the first half of life the recognition of bone disease is practically tantamount to the recognition of caries. In the second half, however, the relative infrequency of caries, the greater frequency of growths in the bone, and the occurrence of eroding aneurisms introduce a fresh diagnostic problem. The absence of any other indication of a tumor or an aneurism is the first distinction, and the second is the fact that in both these diseases the root pains commonly reach a degree of severity scarcely ever attained in caries, and are especially increased by movement, often to an intensity which is literally frightful.

When there are merely tenderness of the spine and slight weakness of the legs, the question may arise whether there is organic disease, or merely the condition termed "spinal irritation." In the latter, tenderness is usually found over a considerable area of the spinal column, with more than one point of special intensity, and there are no root pains or spots of anæsthesia. The latter sometimes afford important help in the diagnosis of an obscure case. In some instances, however, it is necessary to wait and watch the progress of the symptoms before a definite opinion can be formed. The same general principles must guide the diagnosis between caries and so-called hysterical paraplegia with spinal tenderness. There is more danger that caries of the spine in a young woman may be passed as hysterical paraplegia, than of the opposite error. Especially when the subjects of caries present distinct symptoms of hysteria, there is risk, as experience shows, that unequivocal symptoms of caries may be overlooked.

PROGNOSIS.—Our ignorance of the precise character of the morbid process which is damaging the cord renders the prognosis, in every case of caries, a matter of much uncertainty. Nevertheless there is no disease of the cord in which symptoms of equal gravity so often pass away. The cases are few, therefore, in which hope is unjustified, but they are equally few in which we are warranted in a confident expectation of recovery. The indications that may guide the prognosis in an individual case are slight. In childhood the prospects of recovery are certainly better than in adult life, and they are least in declining years. Damage to the cord between the enlargements is less serious than when these are affected, because the strong tendency to trophic changes constitutes a grave danger when the lumbar enlargement is diseased, and the diminished breathing power an equally serious danger in disease of the cervical enlargement.

But how perilous a condition may be recovered from is shown by the fact that the child mentioned on p. 180 with paralysis of all four limbs, the diaphragm, and weakening of the intercostals, made a good recovery. Still more striking is a case narrated by Dr. Buzzard, in which disease in the region of the third cervical vertebra caused almost complete palsy of arms, legs, intercostals, and diaphragm, respiration being carried on by the accessory muscles of the neck. Yet the child recovered in spite of the occurrence of an attack of pneumonia when the paralysis was at its height. Neither rapidity nor slowness of onset affords any guide to prognosis, nor does the relative order of paralysis and curvature, nor the degree of palsy. Even pronounced spastic paraplegia may pass away entirely. The prognosis is perhaps a little better when there is no loss of sensation, since this proves that the damage to the cord is moderate in degree, but even complete anæsthesia does not preclude recovery, as the cases mentioned show. The danger to life is dependent in considerable degree on the evidence that the scrofulous or tubercular tendency is active elsewhere, and also on the possibility of securing proper treatment.

Treatment.—The first and chief element in the treatment of paralysis is that of the bone disease which causes it, and for full details of this the reader is referred to treatises on surgery. If the bone disease heals, the spinal cord, in most cases, will recover. The two most potent therapeutic agents are the customary measures,—persistent recumbency, and tonics, especially cod-liver oil and iron. In the cases that have done best under my own observation, these, and these alone, have been the effective agents. Rest should be maintained for months. The posture that answers best is upon the back. It is true that the spine is then the lowest part, and spinal congestion is favored, but experience is here at variance with theory, and the greater ease with which immobility is secured and maintained seems to more than counterbalance the disadvantages of this posture. If no improvement occurs after some months' rest, suspension may be tried. It is said to be sometimes attended by instant improvement, especially in children. I have not myself seen any instance of this result, which can only occur in the cases, not very common, in which displaced bone compresses the cord. It may, indeed, be well to see the effect of suspension before rest is commenced. The plaster jacket is an inefficient substitute for rest, and an unnecessary concomitant—to be adopted only when rest cannot be secured. It may be tried if rest alone has failed. In caries of the mobile cervical spine, the fixation of the head by the support of a "jury mast" is necessary.

The influence of cod-liver oil and iron is as marked in this as in other scrofulous diseases. They constitute an indispensable adjunct to rest, and may be effective even alone, as is shown by the case mentioned on p. 182. But rest should always be secured if possible.

Counter-irritation, opposite the seat of caries, has often appeared to do good, perhaps acting especially on the process of myelitis which attends compression. The actual cautery, in mild form, is that which has been most frequently useful. Charcot and others have published instances of speedy improvement after its use. It is not always effective, nor can indications for its use be laid down. I have not, myself, seen any distinct improvement after the use of the cautery, although I have employed it in a considerable number of cases. But the testimony of others is so strong as to justify its adoption if the symptoms do not speedily yield to other treatment. Sulphide of calcium, which is said to influence scrofulous processes, deserves trial. Although I have not seen benefit from its use in any other case than that mentioned on p. 183, the rapidity of improvement in that case was very striking. Benefit can only be expected from its use when inflammatory products constitute the compressing agent. Extreme care in general management, to avoid bedsores, bladder trouble, and bronchitis are of great importance. In many cases there is a tendency to improvement after a time, especially when the bone disease is stationary, or the cord has been inflamed, and in these the mere maintenance of life may result in recovery of strength. When power has returned, but the use of the limbs is restrained by the muscular contractions which come on during the paralysis, the ability to stand and walk may, as I have seen, be quickly restored by tenotomy, and more slowly by the use of extending splints. Electricity is useful only to maintain the nutrition of the muscles when these are wasting. The combined teaching of the pathology of caries and of the recent experience of antiseptic surgery justifies the anticipation that the trephine may sometimes be successful when other agents fail, since the cause of compression is generally outside the dura mater, and the power of recovery of the cord, when compression is removed, is very great.

TUMORS AND OTHER DISEASES OF THE SPINE.

GROWTHS IN THE SPINAL COLUMN.

The bones of the spine are sometimes the seat of primary or secondary growths, and, less commonly, are invaded by tumors springing from the structures in front of the spine. Cancer (scirrhus and encephaloid) and sarcoma are the most common forms of tumor that begin in or invade the spine. Myxoma has been also met with. Cancer is often secondary to a primary growth elsewhere, in the breast, stomach, etc. A primary growth usually begins in the bodies of the vertebræ, and spreads from one to another. The bodies may collapse, because the soft tissue which replaces the bone yields under the weight it has to bear. .Thus angular curvature may occur. From the bodies the growth may extend into the lateral processes, enlarging them, and narrowing the intervertebral foramina through which the nerves pass. It may extend into the arches, and even into the spines, and may grow into the muscles and beside and behind the vertebral column. The nerve roots suffer (1) by pressure, (2) by simple inflammation, (3) sometimes by cancerous infiltration. They may thus be found reddened, swollen and soft, or gray and atrophied, or enlarged and hardened. The growth, cancer especially, is apt to spread in the adipose tissue between the bodies and the dura mater. It may even entirely surround the dura mater and cord. The cord may suffer compression, but this is less frequent than in caries. On the other hand, it is also inflamed, sometimes acutely, and considerable inflammation without compression is far more frequent than in caries. Myelitis occurs without any perforation of the dura mater. Why this should be is not known. The inflammation thus excited sometimes spreads widely in the cord. The growth never invades the cord itself. In rare cases the cord is compressed by displaced bone.

ETIOLOGY.—The general causes are the same as those of similar growths elsewhere. Males suffer, however, more frequently than females, and the disease is most common between forty and fifty years of age. An injury has been supposed sometimes to excite a growth in this as in other situations.

SYMPTOMS.—There may be direct symptoms of the presence of the growth—pain in the spine, sometimes severe, sometimes absent; local tenderness, and occasional interference with movement apart from pain. A palpable tumor is never an early symptom, but ultimately a deep-seated, hard swelling may be felt on one side of the spine, most readily and earliest when the disease is in the cervical region, where it is occasionally perceptible in the posterior triangle of the neck. The angular curvature which sometimes occurs may not differ from that of caries in its external characters, or it may be accompanied by the indications of a tumor, never by those of an abscess.

The nerves suffer almost invariably in spinal growths and give rise to the most distressing symptoms, which are also the earliest and most frequent—radiating pain, felt along the course of the nerves that emerge from the diseased portion of the spine, and due to their irritation by pressure or inflammation.

Such pains are far more prominent symptoms in this disease than in caries. At first slight, they gradually increase to extreme intensity. It was this character that led Cruveilhier to call the disease *paraplegia dolorosa*, a name it has since commonly borne. The pains are paroxysmal; in the intervals the patient is at first free from pain, but subsequently pain is constant, with paroxysms of greater suffering from time to time. It is usually sharp, lancinating pain, and its special characteristic is the degree in which it is increased by movement. The distribution in the arms, trunk, legs, depends on the seat of the disease. It is extremely rare for these pains to be absent, but they are now and then a late, instead of an early, symptom.* Cutaneous hyperæsthesia usually accompanies them, and spots of anæsthesia often develop after a time in the hyperæsthetic area. Corresponding damage to the motor roots may cause painful muscular contracture (Fig. 50, p. 96), paralysis, and wasting. Paroxysms of spasm often attend the pains, especially in the abdominal muscles when the disease is in the dorsal region, and are apparently reflex in nature.

The damage to the cord causes symptoms similar to those in caries, and described more fully in the chapter on "Compression." The chief difference from caries is the frequency of a rapid onset of the paraplegic symptoms, due to the invasion of the cord by acute inflammation. All power in the legs is often lost in twelve or twenty-four hours from the onset, and this when no curvature has taken place. Displacement of bone has also been known to cause rapid paralysis.† On the other hand, in very slowly growing tumors, the onset of the palsy may be gradual. I have known it to occupy several years in reaching a considerable degree. Probably in these cases the mechanism is a pure compression. Thus, compared with caries, the onset is more often rapid, and occasionally much more deliberate. The characters of the resulting paralysis are, as a rule, similar to those in caries, but are more frequently modified by the spread of inflammation. Hence the central and reflex functions of the lumbar enlargement, at first normal or excessive, may be afterward lost, although the bone disease is some distance above it. In a man with a growth in the mid-dorsal region, paraplegia came on rapidly, evidently from myelitis, and was followed by foot clonus, etc. But a few weeks later the clonus suddenly ceased, the muscles became toneless, with loss of faradaic irritability, and the skin began to slough. The inflammation had spread down into the lumbar enlargement. Sensation is lost rather more frequently than in caries. Other symptoms are the same as in compression from any cause. The course of the disease is, from its nature, usually progressive. Occasionally, however, some improvement occurs. An inflamed cord may partially recover if life is prolonged for a sufficient length of time. Much more rarely the pains may lessen, although the growth spreads—perhaps from destruction of the irritated nerves.

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* The pains succeeded curvature in a case described by L. Humphry (*Lancet, 1884*, i, p. 15).

† Humphry, *loc. sit.*

The *duration* of the disease varies according to the nature of the growth. In cancer it may almost always be measured by months. In slowly growing tumors the symptoms may last for years. Death may be due to bedsores, etc., to cystitis and kidney disease, to growths elsewhere, or the patient may be simply worn out by the prolonged agony.

DIAGNOSIS.—The recognition of the disease is only a matter of certainty when signs of a tumor are present, but the probability almost amounts to certainty when such symptoms as those described follow a primary growth elsewhere. It must be remembered, however, that similar root symptoms are sometimes due to a growth in front of the vertebral column, commencing, for instance, in the glands, and irritating the nerves as they emerge from the intervertebral foramina.

From intercostal neuralgia, the influence of movement on the pain is usually a sufficient distinction, even when cord symptoms are absent. The commonly bilateral character of the pain is a further difference. The chief difficulty in diagnosis is the distinction from caries when other evidence of growth is absent. In the first half of life caries would alone be thought of in such a case, but in the second half the two diseases are about equally frequent. One distinction, suggestive, not absolute, is the intensity of the pain in tumor, taken in conjunction with its great increase when the patient moves. It is true that the root pains of caries are said to be sometimes most severe, but such severity is not frequent enough to destroy the significance of intensity. I have not, for instance, seen a single case of caries (of a large number) in which the pain was comparable to that in most of the cases of growth that have come under my observation. Therefore, while absence of pain is of slighter diagnostic value (in favor of caries), severe intensity, and agonizing increase by movement, are strongly in favor of vertebral growth.

In each disease there may be angular curvature, but this, in growths, is usually soon succeeded by other signs of tumor. In caries these signs are absent, and an abscess often develops. A history or indication of tubercle is strong evidence in favor of caries. These points will, I believe, avail for the distinction in most cases. In a few it is necessary to wait and watch before an opinion can be formed. The distinction from tumors of the spinal cord and membranes is considered in a later chapter.

PROGNOSIS.—The prognosis scarcely requires formulating. The chief differences are in the time that life is likely to last. The pains usually persist, in spite of the progress of the disease, although there is a bare possibility of their subsidence. The chance of any return of power in the paralyzed part is small, although not quite absent if the palsy develops in a manner to suggest a secondary myelitis, and the progress of the growth itself is slow.

TREATMENT.—This must necessarily be confined to the relief of pain, and to the avoidance of bedsores and other results of the cord disease. Morphia is alone effective for the relief of the pain, but unhappily the dose has to be quickly increased, and the power of the drug is lessened by custom. It becomes a race between dose and pain, in which, if life lasts long, the pain not uncommonly gets in front of the narcotic. It remains to be seen whether, in this as in so many other diseases, cocain may prove an effective substitute for morphia.

VERTEBRAL EXOSTOSES.

Exostoses sometimes grow from the bodies of the vertebræ into the spinal canal, and may compress the cord or nerves. They are, however, exceedingly rare. The symptoms may be those of slow compression of the cord, or of irritation, expressed chiefly by pain. They usually resemble those of a tumor of the cord or membranes rather than of the bones, but the pain is occasionally increased by movement. Their chief characteristic is extreme chronicity, but in this they are rivaled occasionally by some cord tumors. In a case (seen with Mr. W. Adams, of Camden Town) the patient suffered frequent intense paroxysms of pain in the right groin, which had occurred for two years, with occasional intervals of freedom. There was some weakness of the legs, but no considerable paralysis. Ten years before he had had some loss of sensibility in each thigh, which had passed away. An intra-spinal tumor was diagnosed; the post-mortem examination (by Mr. Coode Adams) revealed exostoses from the bodies of the ninth and tenth dorsal vertebræ, slightly compressing the cord. Although extreme chronicity may raise a suspicion of exostosis, it is doubtful whether a confident diagnosis is ever justified except in the cases in which there are similar exostoses elsewhere.

A patient with multiple exostoses, under the care of my colleague, Dr. Barlow, presented paraplegia of gradual onset, which was supposed during life, and found after death, to be due to a similar exostosis within the spinal canal. It had sprung from one of the lumbar vertebræ and had compressed the nerves of the cauda equina.

SYPHILITIC DISEASE.

Syphilitic caries of the bodies of the vertebræ is a rare variety, the symptoms of which do not differ from those of the scrofulous form. It has been observed in the cervical region, secondary to deep syphilitic ulceration of the pharynx. Nodes of the vertebræ, within the canal, are occasionally presumed to exist and to compress the cord, but I do not know of any pathological observation confirming the assumption, and it is probable that most of the supposed instances have been cases of syphilitic gummata in the meninges. Deep-seated thickening of the tissues about the cervical vertebræ sometimes occurs in syphilitic subjects. It may develop on one side or both, and is apparently due to a syphilitic cellulitis. The swelling may be felt either on each side and behind the upper cervical spine, or deep in the posterior triangle of the neck. Movements of the neck may be interfered with, and irritation of the nerves may cause neuralgia-like pain, sometimes felt down the arm. The spinal cord does not usually suffer. All the symptoms slowly pass away if iodide is given.

EROSION BY ANEURISM.

Bones, like other structures, may atrophy and waste before the pressure of an aneurism, and the bodies of the dorsal, or rarely, of the lumbar vertebræ may be thus eroded by aneurisms of the aorta. The pressure and absorption take place from the left side. Two or three vertebræ usually suffer, and the bodies more than the intervertebral cartilages. The periosteum becomes thickened and may resist the pressure and to some extent protect the cord. Sometimes, however, the cord becomes compressed, or the periosteum may come to form part of the wall of the aneurism, and may give way before the blood pressure, so that rupture occurs into the spinal canal.

The symptoms vary much. Pain along the nerve roots is usually severe, but this may attend aneurisms that merely compress the nerves after their emergence, and do not damage the bone. The process of erosion is usually attended by severe pain in the spine. When the cord is reached, compression causes the usual paraplegic symptoms of slow or rapid onset. Rupture into the canal is attended by sudden complete paraplegia and death, either immediate or in the course of a few hours from ascending paralysis, due to the hemorrhage around the spinal cord.

The diagnosis is scarcely possible unless other indications of aneurism are detected, since the symptoms closely resemble those of a growth in the bone. The nature of the disease may, however, be suspected if such symptoms as have been described are succeeded by sudden paraplegia followed quickly by ascending paralysis.

HYDATID DISEASE.

Hydatid cysts sometimes develop in the loose adipose tissue between the dura mater and the bone, and it is believed that they sometimes form in the substance of the bones themselves. About a dozen cases have been collected by Leyden.* As the cyst grows, the bone of the arches becomes atrophied by pressure, and the cyst develops outside the canal, so that there may be a double cyst, outside and inside, connected by a narrower part. Occasionally the cyst develops also in front of the spine, in the thorax or abdomen. The internal cyst necessarily compresses the spinal cord, which often also becomes inflamed. The usual paraplegic symptoms develop, both motion and sensation being often lost. Radiating pains along the nerve roots are frequent. The symptoms, in themselves, resemble too closely those caused by other diseases of the spinal column to permit a diagnosis to be made unless similar disease elsewhere suggests the nature of the spinal lesion, or unless the cyst can be felt in the back, when a puncture may prove its nature. All the cases hitherto recorded have terminated fatally, but it has been conjectured that, if puncture can be effected, a cure may sometimes be possible.

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* *Klinik der Rückenm. Krankh.*, Band i.

DISEASES OF THE ARTICULATIONS.

Lateral Curvature of the Spine scarcely ever affects the functions of the cord. Even when slight compression has occurred, the slowness of its development has apparently prevented interference with function. In very rare cases, some weakness of the legs has been present, possibly, but not certainly, the result of the curvature. Occasionally the intervertebral foramina have been narrowed, and the pressure on the nerves has caused radiating neuralgic pains.

Arthritis Deformans.—In chronic rheumatoid arthritis, the intervertebral articulations may be attacked, and the cartilages may undergo atrophy. Enlargement of the ends of the bones may develop, and, in thin persons, the swelling may even be felt. The symptoms are local pain, tenderness, and limitation of movement, which may go on to absolute fixation, especially in the cervical region, if anchylosis occurs. The cord is scarcely ever compressed, but the narrowing of the foramina may damage the nerve roots, and cause radiating pains, and sometimes set up a descending neuritis. The nerve trunks in the limbs may then become tender as well as painful, muscular atrophy may occur, and, when the disease is in the cervical region, there may be symptoms of disturbance of the sympathetic. Spasmodic torticollis is said to have been caused by this disease in the cervical region. The atlo-occipital articulation has been affected alone, or with the adjacent vertebræ, and peculiar interference with the movement of the head has resulted. Sometimes the foramen magnum is narrowed, or the odontoid process projects into the canal. Symptoms of damage to the nerves of the medulla have been noted (Solbrig), but often the disease has been found out at the post-mortem examination without being suspected during life. The treatment of vertebral arthritis is the same as that of the disease elsewhere, nerve symptoms alone needing special treatment, such as is described in the section on diseases of the nerves.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

The general arrangement of the membranes of the spinal cord has been already mentioned (p. 122). The pia mater closely invests the cord, while the arachnoid forms a loose sheath around it, and the two membranes are connected by trabeculæ and membranous expansions of fine connective tissue, which occupy the "subarachnoid space." Each surface of the dura mater is covered by a layer of epithelium. The outer sheaths of the nerves are continuous with the dura mater; the connective tissue, within the outer sheath, is continuous with both the pia mater and arachnoid. The blood vessels of the cord ramify, as we have seen, in the pia mater, and the lymphatic canals of the two are continuous. Most of the cerebro-spinal fluid within the vertebral canal is contained in the subarachnoid space, but there is a little fluid between the dura mater and the arachnoid. Both dura mater and pia mater are supplied with nerves, but those of the pia mater are the more abundant.

The morbid processes that involve the membranes are chiefly three—growths, hemorrhage, and inflammation. The consideration of tumors of the membranes may be conveniently postponed until the consideration of growths in the spinal cord, with which they have much in common. Inflammation of the membranes and hemorrhage into the membranes will be here described.

SPINAL MENINGITIS: INFLAMMATION OF THE MEMBRANES OF THE SPINAL CORD.

Inflammation of the membranes may be acute or chronic, and may begin, and affect chiefly, the dura mater (*pachymeningitis*) or the pia mater (*leptomeningitis* of German and French writers). The arachnoid usually suffers with the pia mater, but is sometimes the seat of inflammation that affects the pia mater but little, a form that has been termed *arachnitis*. Acute inflammation, beginning in one membrane, usually spreads to the others. Chronic inflammation may remain limited to one membrane, dura mater or pia mater. Inflammation may affect the outer or inner surface of the dura mater. All forms of acute inflammation, wherever they begin, cause similar symptoms. It is only when the inflammation is chronic that the symptoms may differ sufficiently to allow of a precise diagnosis of the membrane affected.

Spinal meningitis may be conveniently divided into two forms: (1) that which begins outside the dura mater, and commonly arises by extension from some adjacent focus of inflammation,—*external meningitis;* (2) that which begins within the dura matral sheath, and is often primary,—*internal meningitis*. External meningitis may spread through the dura mater, and the two forms then coexist. Internal inflammation rarely spreads to the outer surface of the dura mater.

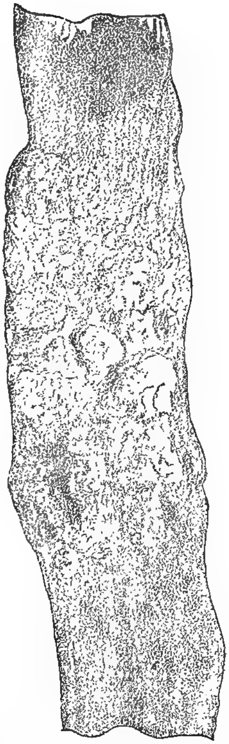
EXTERNAL MENINGITIS.

External meningitis is thus an inflammation of the dura mater. It has been termed *external pachymeningitis*, *peripachymeningitis* and *perimeningitis*. The inflammation involves not only the outer surface of the dura mater, but also the connective tissue which intervenes between the membrane and the bones, and in which are the venous plexuses and a quantity of adipose tissue. In most cases this connective tissue is inflamed before the membrane itself. The morbid process usually remains limited to the outer surface of the dura mater, passing through the membrane only when the inflammation is acute and intense.

Causes.—It is doubtful whether external meningitis is ever primary. In most cases, perhaps in all, it arises from extension from adjacent disease. The most common cause to which it is secondary is caries of the spine. It may also result from deep sacral bedsores, and suppuration outside the spinal column, such as retropharyngeal abscess, or collections of pus in the muscles near the spine, in the back or abdomen. At the same time it is possible that an abscess found outside the spine has occasionally been secondary to the suppuration within the spinal canal, rather than the cause of this. External meningitis has been thought, in rare cases, to be due to extension from inflammation of the loose connective tissue beneath an inflamed pleura, to an ascending neuritis from a limb, and to syphilis.

Pathological Anatomy.—The inflammation is sometimes simple, but far more frequently purulent, or semi-purulent. In the former case the outer surface of the dura mater may be merely reddened and opaque, with a little lymph on the surface. When the inflammation is purulent the surface may be covered by a layer of pus. Such inflammation is commonly the result of acute septic suppuration in the vicinity, and then it may pass through the dura mater, and purulent internal meningitis coexists. In the more common semi-purulent form, such as is common in caries of the spine, a layer of inflammatory products covers the outer surface of the dura mater, soft, semi-caseous, or firm caseous material, sometimes with liquid pus here and there, or contained in cavities within the firmer substance. This layer may be of considerable thickness, sometimes half an inch thick (see Fig. 79, p. 178). It may surround the dura mater, or may be chiefly situated on one side. Sometimes there is an irregular nodular thickening of the outer layer of the dura mater or even a sort of fungoid growth from it. An example of this is shown in Fig. 80. The bulk of these inflammatory products may cause serious compression of the cord. In most cases the inner surface of the dura mater and the pia-arachnoid are normal even when the cord itself is the seat of pressure myelitis. Occasionally the inner surface of the dura mater is also inflamed, and adhesions may form between it and the pia mater. In all forms of external inflammation, the fat outside the dura mater

Fig. 80.



External pachymeningitis; nodular thickening of outer layer of dura mater in caries of the spine.

quickly becomes absorbed. The membrane may ultimately become adherent to the bone. The vertical extent of the disease varies much. When secondary to caries, the inflammation is usually limited to the neighborhood of the bone disease. In other cases the mischief spreads more widely, but when it begins in the dorsal or lumbar regions, it commonly ceases at the cervical region, in consequence of the closer attachment of the dura mater to the bone. In most forms of external meningitis the nerve roots suffer as they pass through the membrane. They may be inflamed and swollen, or compressed and wasted.

SYMPTOMS.—In different cases of external meningitis the symptoms vary much, and they are usually complicated with those due to the cause of inflammation. Those that are produced by the local inflammation which is secondary to caries have been already described in the account of that disease. In other acute cases, the manifestations of the process are nearly the same as those of internal meningitis, presently to be described in detail. The chief are pain in the back, increased by movement, accompanied by stiffness of the muscles of the spine, and by cutaneous hyperæsthesia. Another important symptom is the "excentric" pain, radiating into the parts supplied by the nerves which pass through the inflamed membrane, and due to irritation of their roots. The pains accompany the hyperæsthesia, and are usually associated with spasm; they may be followed by anæsthesia, and sometimes by paralysis of the muscles supplied by the nerves. In addition there may be indications of interference with the functions of the cord itself, due to its compression or inflammation. There are then pains in the limbs, often so great as to prevent walking, paraplegic weakness, and increased reflex action when the disease is above the lumbar enlargement. The skin of the limbs may be hyperæsthetic or anæsthetic, and trophic lesions may occur.

These symptoms differ in character according to the acuteness of the inflammation, and in distribution according to its seat. It is in the most acute form that the cord suffers and corresponding symptoms are developed. In such cases there is usually much pyrexia. In chronic cases, although the pain may be considerable, other signs of irritation, stiffness and muscular spasm, may be inconspicuous, and the symptoms resemble those due to bone disease.

DIAGNOSIS.—The most important indications of the disease are the vertebral pain, and the radiating pains, with other symptoms of irritation of the nerve roots, combined with the signs of pressure on the cord itself. These symptoms have much in common with those of internal meningitis, and it is doubtful whether a diagnosis of external inflammation is justified, unless there is evidence of a morbid process to which the meningitis may be secondary.

PROGNOSIS.—The acute affection is a grave one, most recorded cases having ended in death. But it must be remembered that the disease is usually secondary to maladies which are in themselves most serious, and that the exact seat of the inflammation has usually been made out only after death. It is possible, therefore, that some cases which have recovered, and in which the exact form of meningitis was uncertain, may have been instances of this variety, and that the fatality of the disease may not be so great as published facts suggest. The chronic form, which results from caries, is only serious in the compression it exerts on the cord.

TREATMENT.—The first and most important element is the treatment of the original cause of the disease, the caries, etc., to which the meningitis is secondary. Any accessible collection of pus should be removed. Rest, counter-irritation to the spine, especially the actual cautery, sedatives to relieve the pain, and tonics in chronic cases, are the most important measures. The general treatment for internal meningitis is also suitable to the external form.

INTERNAL MENINGITIS.

Internal meningitis, inflammation beginning within the dura-matral sheath, may be either acute or chronic. The acute form usually commences in the pia mater and arachnoid, the chronic forms may begin in these membranes, or in the inner surface of the dura mater. Little is known practically of any separate affection of the arachnoid.

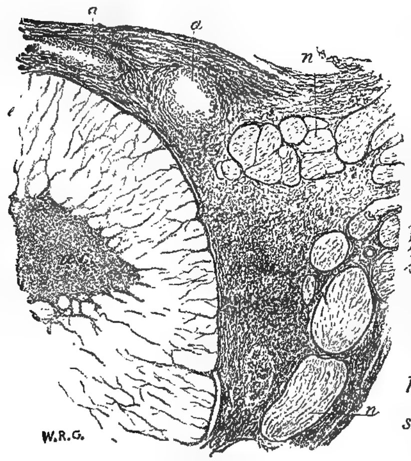
ACUTE INTERNAL MENINGITIS.

Acute internal meningitis, since it begins usually in the pia mater and arachnoid, has been termed "acute leptomeningitis," but it only remains limited to those membranes when of slight degree. In other cases the inner surface of the dura mater is also inflamed. Frequently the inflammation extends rapidly to the cord, and to such cases the term "meningo-myelitis" is often applied. The inflammation may be either simple or purulent, or it may be secondary to tubercle. It also occurs in conjunction with cerebral meningitis in epidemic form. Acute simple meningitis is a rare disease.

CAUSES.—Little is known of the predisposing causes of acute meningitis, except that the disease is more common in males than in females and in early adult life than at other periods, and that its occurrence is favored by depressing influences of various kinds, especially by over-exertion. Among traceable exciting causes, the most important is exposure to cold, such as is involved in sitting on damp grass, remaining in wet clothes, exposure to cold winds during perspiration. It may result, also, from various traumatic influences, from fracture to simple concussion, and from surgical procedures, such as puncture of the sac of a spina bifida. Insolation, prolonged exposure of the back to the heat of the sun, has been the apparent cause in some instances. Suppression of the menses has been regarded as a cause; and perhaps the predisposition to suffer is greater at the menstrual period, but the suppression is more probably a coincident effect of the cause of the meningitis. It has been observed, in rare cases, to come on in the course of acute febrile diseases, scarlet and typhoid fever, acute rheumatism, pneumonia, and in the course of septicæmia. It may also occur by extension, first from the cerebral membranes in cases in which the intra-cranial inflammation is of local origin, and, secondly, from adjacent disease, outside the spinal membranes. External meningitis is first produced, and therefore all the causes of external meningitis may also be occasional causes of internal meningitis. In septicæmia the inflammation is always purulent. When due to other causes it may be simple, but in most conditions, an acute inflammation may go on to the formation of pus. In some instances no cause can be traced. Even purulent meningitis occasionally develops from some thus obscure cause, especially in children.

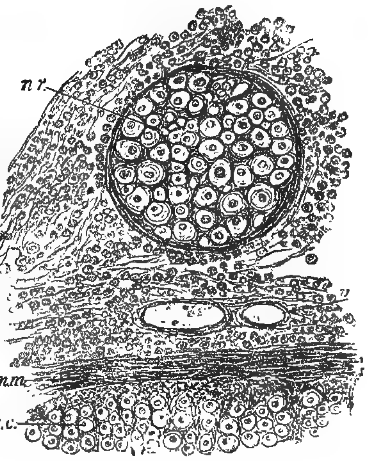
PATHOLOGICAL ANATOMY.—Internal meningitis is usually of wide extent, since the inflammation spreads readily in the loose tissue of the arachnoid. Probably also the movement of cerebro-spinal fluid aids in the extension, by conveying from one part to another irritant material. In the earliest stage, of which few observations exist, the only change is congestion of the pia mater, which is reddened from vascularity and dotted with ecchymoses.

FIG. 81.



Purulent meningitis; portion of spinal cord and membranes; the space between the pia mater and dura mater is occupied by inflammatory products, pus, etc., in which the nerve roots, *n n*, are embedded. *a a.* Cavities which had apparently been filled with liquid pus. From a case of septic origin, secondary to caries of the jaw.

FIG. 82.



From the same; meshes of arachnoid infiltrated with pus corpuscles. A nerve root, *n. r.*, although surrounded by pus, is perfectly normal, and so also are the pia mater, *p. m.*, and the peripheral layer of the spinal cord, *s. c.*

The inner surface of the dura mater, and the substance of the spinal cord, may be similarly congested. When the inflammation is further advanced, in the stage in which the condition most often comes under observation, the pia mater and arachnoid are opaque and thickened, and an "exudation" of inflammatory products, grayish yellow in tint, may cover the pia mater and occupy the meshes of the arachnoid. The exudation may be semi-purulent in aspect, and in the suppurative form the structures are bathed in pus. The inner surface of the dura mater usually presents similar changes, and the inflammatory products may fill up the whole space between the dura and pia mater, thus connecting the two membranes and surrounding the nerve roots (Fig. 81). The microscope shows abundant leucocytal elements, and the larger round and spindle cells that are common in all inflammatory products. The former corpuscles, which resemble, and are probably identical with, pus corpuscles, may be abundant, even when the exudation has not a distinctly purulent aspect, and when it has they constitute almost the whole of the material, lying among the fibres of the arachnoid (Fig. 82). The vessels are dilated, and their sheaths distended with cells. The spinal fluid is increased in quantity, and turbid from flocculi, or even purulent in aspect. The nerve roots are covered with exudation, and are often swollen and reddened from invasion by the inflammation. But they do not always suffer, even in purulent meningitis, as Fig. 82 shows, in which a nerve root is almost normal in aspect, and its sheath is unaffected, although it is surrounded by pus cells. The spinal cord is often invaded by the inflammation; it is reddened, pale, and softened, and the microscope shows the tissue changes common in other forms of myelitis. The change is always most marked in the periphery of the cord, and may occur in wedge-shaped areas, having the apex directed inward, and coalescing at the periphery. The continuity of the vessels, and lymphatic spaces, of the pia mater and cord renders the invasion of the latter intelligible. In some cases of purulent meningitis the pia mater itself is little affected,even when the arachnoid is filled with pus, and in these cases the spinal cord may be normal, as in the example shown in Fig. 82. If recovery takes place, the inflammatory products may undergo cicatricial changes, the membranes remaining opaque and adherent, and there may be permanent excess of the arachnoid fluid. The changes in the cord may lead to sclerosis, widely spread, or limited to certain spots, and from these secondary degenerations, ascending and descending, may develop. The area affected. varies in different cases. Occasionally the membranes are involved in their whole extent. Usually the affection is greater on the posterior than on the anterior surface; probably on account of the influence of the recumbent posture.

In tubercular inflammation the amount of exudation is usually less, and it may be absent. If present, it is commonly gray and gelatinous in appearance, and in it are scattered the grayish or whitish tubercular granulations. Similar granulations may usually be found in abundance upon the inner surface of the dura mater. Such gray granulations are often found upon the spinal membranes in cases of tuberculosis when there is no inflammation, and even when the cerebral membranes are intensely inflamed. The arachnoid, especially that covering the cauda equina, may appear as if dusted over with gray particles, so abundant are the granulations.

In many cases, the signs of spinal meningitis are associated with those of inflammation of the membranes of the brain, chiefly of those about the base and posterior part of the brain, rarely of the vertex. The continuity of the spinal and cerebral inflammation may be obvious or indistinct. In the latter case, the connecting inflammation has apparently been much slighter than that in the base of the brain or around the cord. In cases of slight cerebral meningitis, in which the patient has been recumbent, signs of spinal meningitis, opacity of the arachnoid and its distention with slightly turbid fluid, may exist only in the cervical region, ceasing opposite the upper dorsal vertebræ, and thus extending as far as gravitation (the head being slightly raised on a pillow) favored the descent of inflammatory products. In the violent purulent cerebro-spinal meningitis which is occasionally produced by a septic influence, pus around the cord is often continuous with that which bathes the base of the brain. In a case of this kind, in which the meningitis was secondary to acute double purulent otitis, Leyden found abundant active micrococci, very similar to those associated with erysipelas.

Symptoms.—Slight pain in the back, and malaise, may precede the acute onset of the symptoms. This is usually marked by a rigor, by pyrexia, and by severe pain in the back. The pain varies in position, according to the locality of the inflammation. It is often felt along the whole extent of the spine. Pain also radiates along the distribution of the nerves, round the trunk or to the extremities. This is paroxysmal and very intense; sharp, darting, burning, or constricting. The pain in the back is usually constant, with exacerbations. It is often increased by movement, usually also by pressure and by the application of a hot sponge to the skin. It is no doubt due to the irritation of the inflamed meninges, while the radiating pain is produced by the inflammation of the sensory nerve roots. Muscular spasm usually comes on at the same time as the pain. It shows itself first in rigidity of the muscles of the back, most marked, if the inflammation is local, in the neighborhood of the inflamed part. It may merely cause retraction of the head, or stiffness of the back, or may be so general and severe as to cause opisthotonos, resembling that of tetanus. The spasm usually involves also other trunk muscles, especially those of the abdomen, which become hard and cramped. The limbs also become rigid, and painful cramp-like spasms occur in them, especially on attempts to move. The spasm is probably in part due to the irritation of the motor nerve roots, and is in part reflex from the irritation of the sensory fibres distributed to the pia mater. There is usually great hyperæsthesia of the skin to all forms of sensation, and also increased sensitiveness of the muscles, especially in the lower extremities. Pressure on the muscles of the arms may cause no uneasiness, while a similar pressure on those of the legs occasions great pain. Reflex action is usually increased at the beginning. There are constipation and often retention of urine, in spite of irritable attempts to pass water, the result, apparently, of reflex spasm of the bladder and sphincter. The vascular dilatation which follows a scratch on the skin is prolonged (meningeal streak). Dyspnœa may result from the spasm of the thoracic muscles, and may be almost suffocating in its severity. The pulse may be frequent or retarded. The temperature is raised, sometimes, however, to only a slight degree. Cerebral symptoms, headache, delirium, coma, occur when inflammation has extended within the skull. The "Cheyne-Stokes breathing" may be present, from implication of the medulla.

As the disease progresses, the symptoms of irritation give place to those of paralysis, which may be most marked where the rigidity was greatest. The limbs become relaxed, and feeble or powerless. Sensibility is lessened or lost. Reflex action disappears, and death may occur from asthenia, or from paralysis of the respiratory muscles. Toward the end there is sometimes considerable rise of temperature. In some cases the symptoms become less progressive and the disease passes into a less acute stage; the pains persist, the loss of power continues and may even slowly increase. Death may occur, after weeks of suffering, from the effects of bedsores or from secondary kidney disease, due to the retention of urine, and facilitated by trophic disturbance. On the other hand, in slight cases, the signs of irritation may lessen and pass away, while those of structural damage, paralysis and anæsthesia may remain, and to these may be added muscular atrophy and contractures, from the secondary consequences of the lesions of the nerve roots. Such persistent symptoms vary much in extent and degree, according to the position and intensity of the morbid process. The symptoms of damage to the cord may slowly increase, in consequence of the spread of chronic myelitis, or still more chronic degeneration, set up by the acute mischief. In very slight cases the symptoms of meningitis may pass entirely away.

The symptoms above described vary in their grouping, according to the position of the disease. When the membranes over the lumbar enlargement are chiefly affected, the pains, hyperæsthesia, and cramps are confined to the legs and loins. When the disease is in the dorsal region there may be similar hyperæsthesia and spasm in the legs, but the pain and cramp extend higher, and involve the trunk. If the cervical region is affected, the symptoms extend to the upper extremities, the dyspnœa may be great, and there is often difficulty in swallowing. Contraction or dilatation of one or both pupils may occur. Extension to the brain is marked by vomiting, general headache, delirium, and paralysis of cranial nerves. If such symptoms preceded those of spinal meningitis, we may conclude that the inflammation commenced within the skull, and descended to the spine. The symptoms vary somewhat according to the nature of the inflammation. In purulent meningitis, strange to say, the symptoms of irritation are sometimes very slight, apparently because there is little tendency to invade the nerve roots. In the case from which Figs. 81 and 82 were taken there were hardly any symptoms to suggest meningitis, and, although there was paraplegia, it was probably produced by the mere pressure of a large collection of pus on the spinal cord. There were no pains or spasms. The case was apparently of septic origin. The duration of the acute symptoms varies from a day or two, in severe cases, which end in death, to two or three weeks, in cases of less severity, which may end in either death or recovery. The duration of the subacute and chronic symptoms that supervene is to be measured by months, and sometimes by years.

The symptoms of tubercular spinal meningitis resemble those which have been described, but are usually less intense. There is pain in the back and loins, with stiffness of the spine and retraction of the neck, so that it may be difficult to bend the head forward. There is also variable rigidity, hyperæsthesia and tingling in the limbs, followed by lessened sensibility and paraplegic symptoms. Herpes along the course of an intercostal nerve has been noted.

Diagnosis.—The diagnosis of the disease rests on the pain in the back, the rigidity of the spine, the hyperæsthesia and spasms in the limbs, excited especially by attempts to move them, the acute onset of the symptoms and their association with pyrexia. In simple *myelitis*, on the other hand, there is little pain in the back, paralysis occurs early and is the characteristic symptom, and there is little or no spasm in the limbs in the early stage of the affection. Often, however, some meningitis occurs at the onset of acute myelitis, and then some pain in the back and slight rigidity in the limbs may precede or accompany the onset of the paralysis. In such cases the predominance of the meningeal or cord symptoms must determine the category in which the case is to be placed. *Tetanus* is attended by rigidity of the back, and by spasm, but there is no fever at the onset, trismus is an early and obtrusive symptom, and the paroxysms of muscular spasm are excited by peripheral impressions much more readily than in meningitis, in which they occur chiefly on attempts to move. *Rheumatism of the dorsal muscles* may cause pain in the back on movement, but there is not the spontaneous pain, or the spasm in the muscles, which characterize meningitis. In *gonorrhœal rheumatism* both spinal and radiating pain may exist, probably from an affection of the vertebral articulations, similar to that which exists elsewhere. The affection of other joints and the absence of rigidity are commonly sufficient for the diagnosis. The diagnosis of tubercular spinal meningitis depends on the combination with cerebral meningitis, which usually precedes the spinal symptoms, and on the gradual and insidious onset. Indications of the tubercular or scrofulous diathesis are commonly present in the state of other organs, or to be ascertained from the family history of the patient.

Prognosis.—The prognosis is grave in all cases. It is worse the more severe and acute are the symptoms, the higher the temperature, and the sooner the symptoms of irritation give place to those of paralysis. It is worse, also, when due to lesions of the spinal column or to tuberculosis, than when due to cold, and worse in the so-called "spontaneous" cases than in those which result from traumatic causes. Recovery is more probable in middle life than in early or advanced age. The previous health of the patient also influences the prognosis. It must always be remembered that even if the patient survives the period of acute inflammation, serious permanent mischief may remain.

Treatment.—Perfect rest and quiet are of the greatest importance throughout the course of the disease. All noise should be, as far as possible, excluded; the light should be subdued, and all bodily movement and mental exertion as far as possible avoided. Although it is undesirable that the spine should be the lowest part of the body, yet, in acute meningitis, it is scarcely possible for the patient to lie in any other posture. The prone position interferes with respiration, and both it and a lateral posture cannot be borne on account of the greater muscular exertion which, directly or indirectly, they entail. Dry, or, in robust patients, wet cupping, or leeching, along the spine may be employed at the onset, and be followed by the local application of ice; by means of a spinal ice bag which may be extemporized with gutta-percha tissue made to adhere by touching it with chloroform. Counter-irritation, by blisters or repeated sinapisms, is more useful when the disease is subsiding than at the onset. In cases that are due to cold, free diaphoresis often does good; a hot air or vapor bath should be employed at the onset of the treatment. A warm bath may be followed by moist packing for several hours. The relief thus given is sometimes very great. The bowels should also be freely opened.

The only internal remedy which has been held in general repute, as capable of influencing the inflammatory process, is mercury. The confidence placed in it of old is not altogether unwarranted. It may be given, guarded with opium to avoid irritation of the bowels, until there is a very gentle affection of the gums. The oleate of mercury may be rubbed in along the spine. It irritates the skin, and then may combine some counter-irritant influence. Iodide of potassium seems to have little influence over acute inflammation.

It is necessary to give sedatives for the relief of the pain and spasm. Of these morphia, given by the skin or the mouth, is alone really effective. Sometimes inhalations of chloroform are necessary to relieve the intense suffering. In slight cases relief may be afforded by belladonna, or by atropine injected beneath the skin. Chloral, or chloral and bromide, may be given if there is insomnia.

When the disease has passed into a chronic stage, iodides have been thought useful. Counter-irritation may be freely employed with advantage. Tonics, iron, quinine, and even strychnia, are beneficial. Warm baths, as those of Bath and Aix les Bains, seem sometimes of service, especially employed as hot douches to the spine. The local consequences, muscular atrophy, contracture, etc., need special local treatment by electricity, rubbing, and the like.

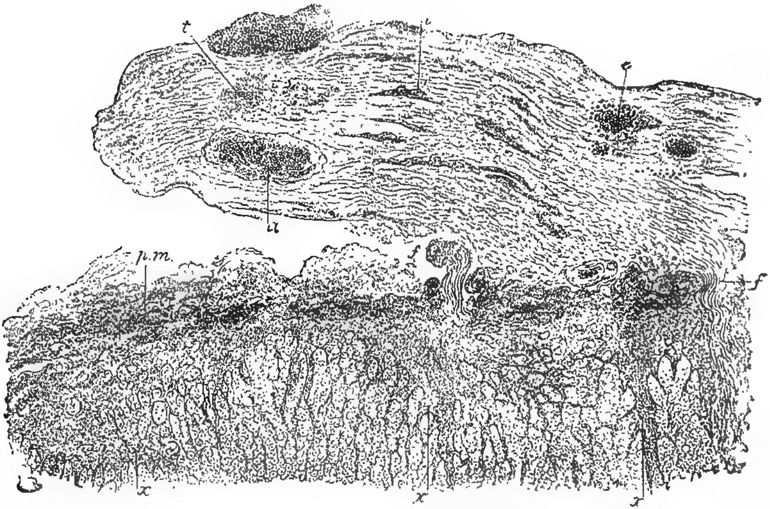
Epidemic cerebro-spinal meningitis is described in the section on diseases of the cerebral membranes.

CHRONIC INTERNAL MENINGITIS.

Chronic inflammation of the membranes of the cord, within the dura-matral sheath, is divided into two forms, according as it begins in, and chiefly affects, the dura mater, *chronic internal pachymeningitis*, or the pia mater and arachnoid, *chronic leptomeningitis*. Although these forms sometimes present distinct clinical and pathological features, they have, when of considerable degree, many characters in common; they own the same causes and need the same treatment. Hence it is most convenient to describe them together as forms of internal meningitis. The condition termed *hæmatoma of the dura mater* depends on a form of hemorrhagic inflammation.

Causes.—Chronic internal meningitis, in every form, is most frequent in adult age, and, like acute inflammation, affects men more frequently than women. It has been observed in persons with neurotic heredity, although whether any influence is to be ascribed to this relation is doubtful. Debilitating influences of various kinds may predispose to the disease, and it is sometimes the result of constitutional syphilis and of chronic alcoholism. The syphilitic form often reaches a high degree of intensity. Among exciting causes the most important is prolonged and habitual exposure to cold. It is doubtful whether simple mechanical congestion ever produces the disease. Traumatic lesions, concussion, etc., are occasional causes, and the affection has been ascribed to prolonged over-exertion and to sexual excess. It may result by extension from inflammation outside the dura mater and from chronic inflammation of the substance of the cord itself. Chronic cerebral meningitis is sometimes associated with the spinal inflammation, but it is often uncertain whether the association is due to extension or whether both are the result of a common cause. Lastly, it must be noted that chronic meningitis may be a sequel to the acute form. Hemorrhagic pachymeningitis occurs especially in the insane, but has been met with as a consequence of chronic alcoholism and after injuries.

FIG. 83.



Chronic alcohol meningitis. Section of edge of anterior column and of a large nerve root; carmine preparation. *p. m.*, Pia mater irregularly thickened and transformed into amorphous-looking tissue, from which wedge-shaped branching tracts (*x x*) extend into the white substance; *f. f.*, fasciculi of nerve fibres entering the cord; *a.*, an artery in the nerve root, enlarged and with thickened walls; *t. t.*, tracts of amorphous connective tissue; *e.*, a small extravasation.

PATHOLOGICAL ANATOMY.—In slight and moderate degree there is merely opacity and thickening of the membranes affected, sometimes with distention of vessels or minute spots of extravasation. The inner surface of the dura mater may be granular. The spinal fluid is increased in quantity and is turbid. The opacity of the arachnoid may be such that the spinal cord cannot be seen through it. When the changes are greater in degree, the dura mater and pia mater may be connected together by a tract of inflammatory tissue of considerable thickness, so that it may be impossible to say in which membrane the disease commenced. The microscope shows the ordinary elements of inflammatory tissue, cells of various kinds, many lymphoid and pus-like corpuscles, and distended vessels, often encrusted by lymphoid cells. Frequently also the pia mater is transformed into a thick irregular layer of homogeneous tissue in which no distinct cell elements can be perceived, and only faint indications of a fibrous structure (Fig. 83). The walls of its vessels may be greatly thickened by similar material.

The nerve roots passing through the diseased membranes are usually inflamed, reddened, and swollen, and afterward may be compressed and atrophied, if the amount of new tissue formed about them is considerable. They suffer thus especially when the inflammation involves the dura mater, on account of the unyielding character of the fibrous sheaths which they receive from that membrane. When the pia mater is thickened, scattered tracts of connective tissue may be seen in the substance of the nerve roots, between the fibres (Fig. 83, *t*).

The spinal cord may be little affected, but is frequently damaged by the extension to it of the inflammation, causing softening, vascularity, loss of distinction between gray and white substance, breaking down of nerve elements, and infiltration by lymphoid and angular cells. It may also suffer, when the amount of newly-formed tissue is considerable, by compression alone or in combination with the inflammation which usually accompanies pressure. When the pia mater is thickened, tracts of similar tissue may extend into the cord, becoming narrower as they pass inward and sending out branching trabeculæ (Fig. 83, *x*). Between these tracts the nerve fibres may be normal or degenerated.

The membrane in which the inflammation begins may be much more affected than the other, although the latter is very rarely healthy except in the slightest cases. Inflammation beginning in the pia mater and arachnoid is often extensive in range. That which commences on the inner surface of the dura mater is frequently localized. This local inflammation of the inner surface of the dura mater often leads to the formation of a large amount of new tissue, and has been described by Charcot and Joffroy as *hypertrophic internal pachymeningitis.* It affects most commonly the cervical region, but sometimes occurs at the lower part of the cord. On opening the spinal canal a fusiform tumor is seen, the outer surface of which is the unaltered outer surface of the dura mater, and on section the enlargement is seen to depend chiefly on a great thickening of the inner part of this membrane, sometimes amounting to one-fifth of an inch; several layers of new tissue can often be distinguished. The pia mater may be normal, but is more commonly thickened, and it is often united to the tissue proceeding from the dura mater. The cord is compressed and commonly softened at the spot, and it presents signs of old inflammation. The nerve roots are also greatly damaged by compression by the newly-formed tissue. In other cases the thickening of the dura mater is slighter and more diffuse, affecting occasionally a wide extent of the membrane. Sometimes at the spot most affected, the cord may be surrounded by a ring of new tissue of cartilaginous hardness. The vertical extent of the disease varies much. It is often considerable, sometimes universal, but occasionally it is small. The membranes above the cauda equina may alone be affected—the dura and pia mater adherent, and the nerves united in a fibrous mass.

It is doubtful whether the white fibroid or cartilaginous plates found so often in the arachnoid after death have any connection with preceding inflammation. In most cases in which they are found no symptoms have existed during life. It is said, however, that when they are numerous and extensive they may give rise to symptoms closely resembling those of chronic meningitis. (Vulpian.) Fibroid plates in the dura mater have been seen in a case in which previous symptoms suggested that they resulted from chronic inflammation. (Jaccoud.)

In the rare cases in which the inner surface of the dura mater is the seat of a form of hemorrhagic inflammation similar to, and commonly associated with, that which affects the cranial membrane (internal hemorrhagic pachymeningitis, hæmatoma of the spinal dura mater), a reddish-brown exudation covers the surface of the membrane. It is composed of fibrin and extravasated blood ; the latter may be encysted in small cavities, or may be in various stages of transformation. The change commonly extends over a great part of the cord. It is apparently the result of hemorrhage into inflammatory tissue.

Symptoms.—The symptoms of chronic internal meningitis, like those of the acute form, are due to the irritation of the membranes, to the damage to the nerve roots, and to the affection of the cord. In the different forms, one or another of these sets of symptoms predominate, so that the varieties, while possessing many characters in common, may differ considerably in their clinical aspect. In some cases, we can infer, with accuracy, the special form of the disease, but in many others we cannot carry our diagnosis beyond the conclusion that chronic internal meningitis exists, and are unable to say, even with probability, which membrane is primarily affected.

The common symptoms are these: There is pain in the back, increased by movement, sometimes, however, amounting only to heavy, dull discomfort, and accompanied by some stiffness of the back, and, when in the cervical region, by some retraction of the head. The pain is increased by pressure on the spines, and its increase by movement may cause a fixation of mobile parts, as the neck, in some abnormal position. The pain is apparently due to the irritation of the meningeal nerves, and the rigidity is to be regarded as a reflex effect of this irritation.

Still more conspicuous, in many cases, are the radiating or excentric pains due to the irritation of the nerve roots, and referred to the region in which the nerves are distributed, back of the head, neck, arms, thorax, abdomen, loins, or legs, according to the position of the disease. These pains are often very severe, sharp, darting, burning, or rheumatoid in character, paroxysmal in occurrence, and sometimes worse at night. They may be accompanied by a painful sense of constriction, and by various unpleasant sensations, numbness, tingling, formication, in the same areas. Hyperæsthesia may also exist, so that pain, sometimes thrilling in character, is produced by touching the skin, or by slight degrees of heat or cold, and a hot or cold sponge passed along the skin of the back may reveal the chief seat of disease by the change of sensation at its level. The hyperæsthesia may be more marked to one form of sensation than to another. Cutaneous eruptions, such as result from other nerve lesions, have also been observed in association with the pains. In the same regions, muscular twitching, tremor, or even spasm, may result from the irritation of the motor root fibres. These symptoms correspond in their level to the part of the membranes chiefly diseased. When the lumbar membranes are affected, the radiating pains are felt in the legs, but when the disease is higher up, the legs may merely feel heavy, and be the seat of slight abnormal sensations.

These pains having lasted for a time, weeks or months, certain paralytic symptoms manifest themselves. The pains sometimes persist, but may cease when paralysis comes on. Some paralytic symptoms occur in the regions in which the severe pains are felt, and depend on further damage to the nerve roots. Sensation becomes lessened, or even lost, in certain areas, to touch, or pain, or both. The muscles in the same region become weak, and waste; their irritability to faradaism is lessened, that to voltaism may persist or may also become less than normal. The wasting affects the muscles irregularly, but entire groups may atrophy. Reflex action in these parts is lowered. In the trunk this loss of reflex action, and anæsthesia, may be the chief local symptoms. As the disease progresses, the cord itself suffers, by compression, or by extension to it of the inflammation. The parts below the seat of the disease then become paralyzed, the legs are weak and the seat of dull, heavy pain; reflex action in them may be increased, while their muscular nutrition continues good, unless the lumbar membranes are diseased, or the myelitis descends the cord. Power over the sphincters may be lost and bedsores may form.

When the inflammation affects chiefly the pia mater and arachnoid (*chronic leptomeningitis*) the symptoms of meningeal irritation are very conspicuous,—vertebral pain and rigidity of the back, with cutaneous hyperæsthesia, and the radiating pains may also be considerable. These symptoms are especially marked in cases of subchronic meningitis, such as are due to chronic alcoholism. The local paralytic symptoms, anæsthesia and muscular wasting, may be absent in these cases, or may only come on in slight degree after the other symptoms have lasted for a considerable time. On the other hand, weakness and pains in the legs, from affection of the cord itself, may be an early symptom.

When the inflammation begins in the dura mater (*internal pachymeningitis*) the symptoms of spinal irritation, pain and stiffness of the back, although occasionally present, are much less prominent symptoms than are those which depend on the irritation of, and damage to, the nerve roots. The radiating pains are very severe and are often the earliest symptom. At a later stage the muscular weakness, followed or accompanied by wasting, chiefly attracts attention, and the wasting may be so great that the disease is mistaken for progressive muscular atrophy. This is especially the case when the lesion is in the cervical region, the "cervical hypertrophic pachymeningitis" of Charcot and Joffroy. In this affection, pains in the back of the head, neck, shoulders, and arms, often accompanied with a painful sense of constriction, precede muscular atrophy. The wasting may involve many muscles, but usually those supplied by the radial nerve are much less affected than the others; the escape of the long extensors of the wrist and fingers, while the flexors of the wrist and fingers and the interossei atrophy, leads to a peculiar deformity; the unopposed muscles cause persistent over-extension of the wrist; the phalangeal joints are flexed and the metacarpo-phalangeal joints are extended, but from the position of the wrist they are not over-extended, and the hand thus differs from the claw-like hand (p. 52). With this muscular wasting in the arms, there is paraplegic weakness, from the pressure on the cord. In rare cases the disease affects the membranes over the lumbar enlargement or cauda equina, and then the legs are the seat of the pains, paralysis, and atrophy, already described, and the sphincters are early paralyzed.

The pachymeningitis may be confined to the dorsal region. In such a case cutaneous anæsthesia over the whole region supplied by the dorsal nerves, was associated with paraplegia from compression of the cord. In another instance areas of anæsthesia, with severe pains, existed over the legs, trunk, neck, and back of the head, without any indication of damage to the cord itself.

The symptoms of syphilitic meningitis do not differ from those just described, and may present the characters of an affection beginning in either the pia mater or dura mater. As already stated, the pain and muscular wasting often reach a high degree in these cases.

In hemorrhagic pachymeningitis the symptoms are those of a slight chronic meningitis, pain in the back or extremities, interference with movement, weakness of muscles, and anæsthesia of the skin. It has never been diagnosed during life.

DIAGNOSIS.—The diseases with which chronic internal meningitis is liable to be confounded, are different according as the symptoms of irritation, of damage to the cord, or of compression of the nerve roots, predominate.

The spinal pain and stiffness may be mistaken for simple rachialgia or "spinal irritation," but in this disease the whole spine is tender, several separate points are much more tender than the rest, and there are not the radiating pains or paralytic symptoms of meningitis.

Peripheral pains, especially about the trunk and legs and shoulders, are prominent symptoms in some cases of muscular rheumatism, or rheumatic neuralgia. In these, however, there is not the spinal pain and tenderness, and although there may be apparent increased sensitiveness in the region of the pains, it is of the deeper structures and not of the skin.

The muscular wasting may be mistaken for atrophy due to degeneration or subacute inflammation of the anterior cornua of the cord (progressive muscular atrophy, subacute spinal atrophy). The cervical "hypertrophic" form of meningitis bears special resemblance to this disease, since the wasting in the arms is often associated with paraplegic symptoms in the legs. The distinction is that the atrophy, in the meningeal form, is preceded by severe pains, and often accompanied by areas of hyperæsthesia or anæsthesia, and is random in distribution. In chronic affections of the anterior cornua both pains and anæsthesia are usually absent, and the wasting often affects the muscles in a certain order, which will be described in the account of the disease. Thus in a man who was supposed to have cornual disease on account of wasting of some muscles of both legs, irregularly distributed, a history of severe sharp pains, and the discovery of a patch of anæsthesia on one leg, and the presence of a burning pain round one side of the trunk, and the irregular distribution of the wasting, led to a diagnosis of chronic meningitis damaging the nerve roots. There was a history of syphilis, and appropriate treatment completely removed the symptoms. Chronic disseminated myelitis may cause muscular wasting, but does not cause the pain in the back, or the rigidity of the spine, which occur in meningitis. The paralysis is usually out of all proportion to the wasting and anæsthesia. It must be remembered that the two conditions of meningitis and myelitis are often combined, and the fact has to be recognized in our diagnosis.

When the meningitis is lumbar, the pains in the legs may resemble those of locomotor ataxy, but the absence of rigidity in tabes and the presence of ataxy, distinguish it from meningitis. The knee jerk may be lost in each disease, but in meningitis there is muscular weakness and wasting, often with altered electrical reaction, symptoms never present in the early stage of tabes. It may be noted, however, that there is sometimes a thickening of the pia mater over the posterior columns when these are sclerosed. This thickening is due to a process partaking of the nature of inflammation. It may account for the vertebral pain occasionally felt in that disease, but does not cause any other symptom of meningitis. The greatest resemblance to chronic meningitis is presented by some cases of tabes in which the cord disease reaches a greater degree in the dorsal than in the lumbar region. There are then pains and anæsthesia in the trunk, and the atrophy in the legs may be little marked. In meningitis, if the membranes over the lumbar enlargement are normal, the knee jerk is preserved and often increased, while it is lost in tabes. The anæsthesia is more general and uniform in tabes than in meningitis.

When chronic meningitis affects the cervical region it may be mistaken for the tonic form of torticollis, but may readily be distinguished by observing, first, that the head is not fixed by spasm, since the muscles in a state of tension are not those by which the posture of the head would be produced, and, secondly, that the fixation is distinctly due to the pain which movement occasions.

Caries of the spinal bones and chronic meningitis have many symptoms in common, and necessarily, since chronic local meningitis is one of the effects of the bone disease, and produces many of the symptoms by which the latter is recognized. The diagnosis can only be made by the detection of the signs of bone disease enumerated on p. 178.

PROGNOSIS.—In all forms and degrees of chronic meningitis, except the most trifling, the prognosis is serious. The severe degrees of the affection are attended with danger to life, and the slighter forms entail serious consequences, since many effects of the disease, especially the damage to the spinal cord itself, tend to increase by the degenerative tendency which is set up. The neuralgic pains, which result from the damage to nerve roots, are extremely obstinate. But in many cases the symptoms pass away under appropriate treatment. The prognosis is most favorable in the cases which result from concussion of the spine and from syphilis. But in all cases, even those of syphilitic origin, in which there is reason to infer considerable formation of new tissue, the prognosis must be cautious, since the cicatricial contraction of this tissue may perpetuate the damage to cord and nerves, which its pressure has produced. The prognosis is better, in other cases, in proportion to the general health of the patient, to the moderate degree of the mischief, and to the extent to which its causes are under control.

TREATMENT.—Rest is essential. In some cases it should be absolute, and in all cases movements which increase pain should be avoided. The pain is the expression of mechanical irritation, which hinders recovery. The prone couch, impracticable in acute meningitis, can often be employed in chronic cases with advantage, to lessen mechanical congestion of the spine. Warm baths and diaphoretic baths have been recommended. They are most useful in subacute cases. Next in importance to rest is counter-irritation. Repeated sinapisms or stimulating liniments may be used if mild counter-irritation only is required, but in most cases greater good will result from more energetic means, blisters, the thermic hammer, or the actual cautery. The latter is especially recommended by Joffroy for the hypertrophic pachymeningitis.

Sedatives are usually needed to relieve the pain ; injections of morphia, chloral, Indian hemp, or sedative liniments of chloroform, belladonna, etc. In employing sedatives it must be remembered that they will usually be needed, in chronic cases, for many months, and after a time, by the artificial need they create, they may apparently keep up the pain. It is therefore well, after they have been used for some time, to change the sedative. If injections of morphia have been employed for a considerable time, it is well to see the effect of an occasional injection of plain water.

Iodide of potassium and mercury are the agents that have most influence over the morbid process itself. Both are effective in syphilitic cases. In other forms of the disease mercury has far more influence than iodide. In many cases tonics, iron, quinine, and cod-liver oil are needed. The muscular wasting which results from damage to the nerve roots requires treatment by galvanism, rubbing, and by passive movement to prevent contractures. The treatment of hemorrhagic pachymeningitis is the same as that of the ordinary form, but if it be suspected, injections of ergotine should be tried. Treatment appears, however, to exert little influence upon it.

HEMORRHAGE INTO THE SPINAL MEMBRANES; SPINAL MENINGEAL HEMORRHAGE; HÆMATORACHIS.

Blood may be extravasated outside the dura mater, between it and the bones (extra-meningeal hemorrhage), or within the dura mater (intra-meningeal hemorrhage). In the latter situation the blood may be in the subdural space, between the dura mater and arachnoid (subdural hemorrhage), or it may be beneath the arachnoid, between it and the pia mater (subarachnoid hemorrhage). Extravasations outside the dura mater are more common than those within it.

Causes.—Meningeal hemorrhage may occur at all ages. It is met with in newly-born children, but in them is probably of traumatic origin. Spontaneous hemorrhage is unknown in early childhood, but occurs at all other ages, and is more common in men than in women. No relation has been traced between its occurrence and the general conditions of circulation, increased blood pressure, vascular degeneration, etc., which favor the occurrence of cerebral hemorrhage. It is doubtful whether it is predisposed to by chronic alcoholism. Of the immediate causes, injury is the most frequent, fractures of the spinal column, blows or falls on the back which do not fracture the vertebræ, and even falls on the feet or buttocks. It is occasionally found after death from severe convulsions, epilepsy, puerperal eclampsia (in which an altered state of the blood may assist), chorea, strychnine poisoning, and tetanus. In these cases the blood may be outside or within the dura mater. In many instances no symptoms were observed during life which could be referred to the hemorrhage, and it is probable that the extravasation occurred only during the last moments of life. In some cases, however, the muscular spasm, resulting from the meningeal hemorrhage, may have been mistaken for an independent symptom, and the hemorrhage, which was really the cause of the spasm, regarded as its consequence. It has probably been so in some of the cases of the so-called tetanus of newly-born children. In two-thirds of these cases extra-meningeal hemorrhage is found, and in those in which the tetanoid symptoms occur almost immediately after birth, they are probably due to the extravasation. Severe and prolonged muscular exertion has been, in a few cases, the apparent cause of spontaneous hemorrhage. It also occurs in some diseases in which there is a hemorrhagic tendency, such as purpuric states, and the hemorrhagic forms of some acute specific diseases, smallpox, yellow fever, etc., and very rarely in typhoid fever, apart from any hemorrhage elsewhere. In most of these cases its occurrence has not been suspected until the post-mortem examination revealed it, and it probably occurs generally during the last moments of life. A very rare cause is the bursting of an aortic aneurism into the spinal canal, after erosion of the bodies of the vertebræ, the blood being effused outside the dura mater. Hemorrhage within the dura mater has resulted from the rupture of an aneurism of a vertebral artery. Blood may also descend into the spinal membranes from the cranium, in cases in which a cerebral extravasation, especially about the pons, escapes into the meninges. Lastly, in cases of intense meningeal inflammation, ecchymoses, and sometimes considerable extravasations have been found, on both sides of the dura mater or in the pia mater. Thus considerable extra-meningeal hemorrhage has been met with in cerebro-spinal meningitis.

PATHOLOGICAL ANATOMY.—*Extra-meningeal hemorrhage* probably comes from the plexus of large veins which lie between the dura mater and the bone. It is usually not of large extent, and the blood accumulates chiefly on the posterior aspect, where, in the recumbent posture, gravitation favors accumulation, and where the space between the membrane and the bone is greater than in front. Sometimes the extravasation is very extensive, covering a large part of the dura mater, and in such cases it may extend through the intervertebral foramina, along the nerves. Hemorrhage is more common in the cervical region than elsewhere, but may occur in any part. The blood is usually coagulated, wholly or partially. The dura mater is reddened, and sometimes the staining extends through to the inner membranes. If the hemorrhage is considerable the spinal cord may be compressed, but the amount of blood is not often sufficient for this. When a body has been lying on the back, the veins outside the dura mater become distended with blood, which escapes when they are divided in opening the spinal canal. Care must be taken to avoid the error, on the one hand, of regarding the blood thus escaping as an ante-mortem extravasation, and, on the other, of overlooking a hemorrhage which actually exists. In all cases of acute spinal disease the body should, if possible, lie face downward until the examination has been made. In rare cases hemorrhage has been found within the substance of the dura mater.

Intra-meningeal Hemorrhage.—An extravasation into the sac of the dura mater (subdural hemorrhage) may be small in quantity, or may fill the whole cavity. When small it may pass from one part to another. In subarachnoid hemorrhage the blood comes usually from the vessels of the pia mater. It may surround the cord for an inch or two, or may fill the whole subarachnoid cavity from one end of the cord to the other. Such extensive effusions are rare, except when the blood descends into the arachnoid from the cerebral membranes. Blood effused into the spinal arachnoid has been known to ascend as high as the pons and even break through the valve of Vieussens and get into the cerebral ventricles.* When cerebral and spinal hemorrhage coexist, it must not be at once assumed that the blood has passed from one to the other, since hemorrhage may occur in each at the same time, as in one recorded case of puerperal eclampsia. (Charrier.)

In meningeal hemorrhage, cerebral as well as spinal, the spinal fluid is often blood stained, and thus spinal hemorrhage may be thought to be more considerable than it really is. The spinal cord is often discolored and compressed, and is especially damaged when the hemorrhage is beneath the arachnoid. In extensive extra-meningeal hemorrhage the subarachnoid fluid may be driven away from the compressed part, and may distend the arachnoid beyond the area of compression, the limit of which is thus marked. In cases which have lasted more than a few days there are usually signs of meningitis, set up by the irritation of the blood.

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* As in an apparently conclusive case reported by Leprestre, *Arch. Gén. de Méd.*, xxii, p. 331.

SYMPTOMS.—As already stated, slight meningeal hemorrhage is sometimes found post-mortem in death from convulsive diseases when no symptoms of it were observed during life, and in these cases it is probable that the extravasation occurs during the lethal convulsion. In the cases in which hemorrhage causes symptoms, these are, for the most part, those of meningeal irritation, and thus bear some resemblance to the symptoms of meningitis. They differ in the suddenness and violence of their onset, to which exceptions are extremely rare. These symptoms are nearly the same whether the hemorrhage is outside or inside the dura mater.

The first indication of the lesion is usually sudden and violent pain in the back, corresponding in position to the seat of the hemorrhage, but sometimes felt along a considerable extent of the back, and often severe in the loins. This pain in the back is usually accompanied by pain along the course of the nerves passing through the membrane near the extravasation, darting or burning pains, often of great intensity, making the patient shriek with agony. They are paroxysmal in character, and between the pains there may be abnormal sensations, tingling, etc., and hyperæsthesia, referred to the same parts. Muscular spasm usually coincides with the pain and involves partly the vertebral muscles, causing rigidity of the spine or actual opisthotonos, partly the muscles supplied by the nerves in which the pain is felt, partly the muscles from the cord below the seat of the hemorrhage. The convulsive movements are sometimes general. Intense pain in the back and general convulsion have been known to be the only symptoms. Occasionally there is persistent contraction of muscles, and there is usually spasmodic retention of urine. These symptoms of irritation are no doubt due in part to the irritation of the membranes (causing the vertebral pain and reflex spasm), and partly to the irritation of the nerve roots, motor and sensory. Paralytic symptoms quickly follow—weakness and lessened sensitiveness in the limbs below the lesion. Ultimately there may be complete loss of motion and sensation in the lower limbs, but such absolute paralysis is not common. Loss of power at the onset of the symptoms usually indicates simultaneous hemorrhage in the cord or the effusion of a very large amount of blood. It occurs, for instance, when an aneurism bursts into the spinal canal.

The symptoms differ in their distribution according to the seat of the disease. When hemorrhage is in the cervical region (a frequent seat), the pains are felt in the neck and arms, and the rigidity may cause absolute immobility of the neck, while dysphagia, interference with respiration, and dilatation of the pupils may be added to the other paralytic symptoms. When it is in the dorsal region, intense pain encircles the chest or abdomen. If in the lumbar region, the pain is felt in the legs, and there are early paraplegic symptoms, with loss of reflex action in the legs, and paralytic incontinence of urine and fæces.

As a rule, in spinal hemorrhage, the cerebral functions are unaffected; the unfortunate patient is conscious of all his sufferings from first to last. Occasionally, however, consciousness is lost for a short time, apparently from shock, and even delirium or coma may come on, either as indirect effect of the spinal lesion on the brain, or in consequence of a sudden increase in the intra-cranial pressure, due to the displacement upward of the spinal fluid by the effusion of blood. But in most cases in which cerebral symptoms coexist with those of spinal hemorrhage, the former have been due to simultaneous intra-cranial disease. So, in some cases of spinal hemorrhage in newly-born children, apoplectiform symptoms have been present from simultaneous hemorrhage into the membranes of the brain. In very rare cases the symptoms of spinal hemorrhage have come on insidiously, without pain, as in a case in which extensive hemorrhage outside the dura mater in the cervical and upper dorsal region, in a girl of fourteen, caused only very gradual weakness in the arms, and difficulty of breathing, from which she died at the end of a fortnight.*

The symptoms in acute cases usually reach their height in two or three hours, sometimes not for a few days. Death may occur when the symptoms are fully developed, or, after reaching their height, they may decline, to be increased, a day or two later, by secondary meningitis. This is accompanied by some pyrexia and lasts a week or ten days, and then, in favorable cases, permanent improvement commences. It is, however, very common for death to take place when the symptoms have existed a few hours only. It may be due to exhaustion, in consequence of the violence of the pain and spasm, but is more often produced by interference with respiration.

DIAGNOSIS.—In cases in which marked symptoms are produced, the diagnosis rests on the combination of sudden localized pain in the back, with the other evidence of irritation of the membranes, nerve roots and cord, above enumerated. When the hemorrhage is of traumatic origin, the fact that the symptoms rapidly followed an injury facilitates the diagnosis. In the extremely rare cases in which the symptoms come on insidiously, without pain, the diagnosis of the exact nature of the disease is scarcely possible.

In hemorrhage into the substance of the cord, vertebral pain is much more frequently absent than in meningeal hemorrhage. The symptoms of injury to the cord itself are prominent from the commencement; sudden paralysis, it may be at first partial, and rapidly increasing, without initial pain. In meningeal hemorrhage, pain and symptoms of irritation usually precede any considerable paralysis. In cases which recover, the paralytic symptoms are more persistent when the hemorrhage is into the substance of the cord than when it is into the meninges. Extravasations into the substance of the cord often break through into the membranes, and the symptoms of both lesions are then combined.

Meningitis is distinguished by the more gradual onset of the symptoms and the presence of fever from the first. It must not be forgotten that

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*R. Jackson, *Lancet, 1869*, p. 5.

meningitis may result from hemorrhage. In myelitis pain is commonly absent, and the symptoms of irritation are far less pronounced. The diagnosis from tetanus depends on the absence of trismus, and on the presence of severe spinal pains, and on the more sudden onset. Most cases of tetanus come on more or less insidiously. It is chiefly in the case of newly-born children that the diagnostic difficulty will arise. Tetanus is in them most common two or three days after birth; the tetanoid symptoms which may supervene an hour or two after birth are probably not true tetanus but tetanoid spasm, the result of meningeal hemorrhage produced by injury during birth. One case has been recorded in which the symptoms most closely simulated those of strychnine poisoning. Violent paroxysms of muscular spasm, with intense general pain, but without spinal pain, followed by death in two hours, were apparently due to an extensive hemorrhage into the sac of the dura mater.* Analysis revealed no strychnia in the stomach. In such a case the diagnosis would have to be guided as much by circumstantial evidence as by the symptoms.

PROGNOSIS.—In all severe cases the prognosis is most grave. A large number of such cases prove fatal in a few hours. If the symptoms reach their height, and cease to increase, and the indications of damage to the cord itself are moderate, the patient will probably recover, although there is still some danger until the period of secondary inflammation is over. Paralytic symptoms often persist for some time, but even these, in the end, may pass away. At the onset, the prognosis must be governed by the rapidity with which the symptoms develop and by the seat of the disease. Hemorrhage in the cervical region is more serious than in the lower parts of the cord.

TREATMENT.—The first point to secure is absolute rest. Even passive movement should be avoided as far as possible. Posture is of the first importance. It should be on the face or side, and not on the back, so that the spine may not be the lowest part. Venesection has been employed in strong individuals, with the object of arresting hemorrhage by rapidly lowering the blood pressure, and it is a justifiable proceeding if the symptoms are making rapid progress, and the diagnosis is sure. Leeches or wet cupping to the spine, or leeches to the anus, have also been recommended in cases in which venesection is unadvisable. Ice should be applied to the spine in all cases, and ergotine may be injected under the skin or a full dose of ergot given by the mouth. The bowels should be freely opened. Sedatives are usually required to relieve the pain. The stage of meningitis must be treated on the principles laid down for the management of the acute form of that disease. The residual palsy requires treatment by electricity, etc., in the same manner as the consequence of meningitis.

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* Dixon, *Lancet*, *1879*, p. 333.

DISEASES OF THE SPINAL CORD.

ANÆMIA AND HYPERÆMIA OF THE SPINAL CORD.

The condition of the vessels of the spinal cord after death, their fullness or emptiness, affords no indication whatever of their state during life. Inferences as to the ante-mortem state, drawn from the post-mortem condition, are altogether erroneous, with the rare exception of local change, in which the vascular condition of one part differs from the rest. Practically, such local variation occurs only in the local hyperæmia that attends inflammation, and the anæmia that results from pressure. Hence the occurrence of variations in the state of the vessels of the cord, and the effects that such variations may produce, are matters of inference from symptoms observed during life, symptoms that are, in themselves, open to various interpretations. Where the ground is barren of facts theory is always luxuriant. Anæmia or congestion of the cord affords a ready explanation of symptoms the cause of which is unknown, and it is scarcely surprising, therefore, that such an explanation has been often given. Some surprise may, however, reasonably be felt at the absolute confidence and precision of detail with which these states have been invoked as morbid processes, when the opinions expressed rest upon not one tittle of definite evidence. Positive assertions always receive some credence, however unwarranted the assertions may be, and positions incapable of proof are sometimes incapable of disproof. It would be a futile and useless task to attempt to refute in detail the various statements that have been made regarding the influence of anæmia and hyperæmia of the spinal cord. It will be sufficient to point out briefly what may be reasonably surmised regarding these morbid states.

ANÆMIA OF THE CORD.

Nothing is known of persistent anæmia of the cord from organic obstruction in the arteries lessening the blood supply. Transient anæmia, if it occurs, must be due to arterial spasm, dependent on the vasomotor nerves. This has been assumed as an explanation of certain transient symptoms occasionally observed, loss of power in the legs, and sensory disturbance, tingling, "pins and needles," etc., in the legs and arms, and the tetanoid cramp and tingling in the hands with which patients sometimes wake up. The theory is tenable, but it is also conceivable that such symptoms are due to transient functional states of the nerve cells of the cord. A functional derangement of these cells must be the immediate cause of the symptoms, whether such derangement is primary or is produced by spasm of the vessels.

In general anæmia, such as occurs in chlorosis and pernicious anæmia, the nutrition and function of all organs suffer, and the spinal cord shares the general state. The readiness with which fatigue of the legs is induced in such conditions may be due to the spinal cord as well as to the muscles. The legs often ache, and are sometimes the seat of various disordered sensations. It is probable that these are the result of the impaired nutrition of the nerve elements, in consequence of which their functions are readily deranged. In some patients graver symptoms occur,—weakness of the legs, sometimes of the arms also, which may increase slowly to complete paralysis. Sensation and the sphincters are usually unaffected. The nature of these cases is uncertain. It is probable that many are cases of what is called hysterical paraplegia. Others may be due to organic changes induced by the anæmia. The well-established fact that optic neuritis may result from chlorosis is interesting as proof of the intensity of changes in nerve structures which anæmia may excite. The cases of chlorotic paraplegia are very rare, and need further study.

In the anæmia that results from loss of blood, besides the symptoms just described, paraplegia comes on, under conditions which exclude the idea of hysteria. The source of the hemorrhage has most frequently been the stomach, kidneys, and uterus. It is usually motor only, but Leyden has observed accompanying hyperæsthesia of skin. It comes on sometimes soon after the loss of blood, sometimes only at the end of one or two weeks. Most cases recover. The pathological process which causes the paralysis is not known. The loss of power is comparable to the loss of sight which occurs from the same cause, and under similar conditions, and in which there may be no visible morbid change, or inflammation may be found, sometimes succeeding the loss of sight, and therefore to be regarded as a result of the derangement of the nerve elements, or of the influence to which this is due, rather than the cause of the amaurosis.

The treatment of the symptoms due to general anæmia is, of course, essentially the improvement of the blood state. That of other symptoms will be considered in the account of functional disturbance of the spinal cord.

HYPERÆMIA OF THE SPINAL CORD.

The conditions of the return of venous blood from the cord probably shield it effectually from the mechanical congestion from which almost every other organ of the body suffers when the movement of blood through the thorax is hindered. The only mechanical congestion to which the cord is liable is that which results from the influence of gravitation, and occurs when a person is lying on the back. The distention of the veins outside the dura mater, and also of those of the pia mater, which occurs when the heart has ceased to act, and the blood is free to obey the only mechanical force which then acts upon it, sufficiently proves the power of gravitation to congest the cord. But it is not probable that gravitation has anything like the same influence during life, counteracted, as it then is, by many other forces. Some persons with weak cords (easily fatigued, liable to tingling and pains in the legs) suffer from aching in the legs or spine when they lie on the back. Since the discomfort ceases when the posture is changed, the pain is probably due to the mechanical congestion of the cord or membranes. It is doubtful whether any other symptoms can be, with confidence, assigned to mechanical congestion. Those other nocturnal disturbances in the limbs, that might conceivably be due to mechanical congestion, occur irrespective of posture.

Of active congestion our definite knowledge may be summed up in the statement that it occurs in the early stage of inflammation.* We *know* nothing of it as an independent condition. Nevertheless volumes might be filled by the collected descriptions of the varieties and symptoms of congestion of the spinal cord,—descriptions in which the unrestricted play of "scientific" fancy has elaborated a symptomatology for the separate congestion of every part of the spinal cord. Probably the only case in which we are warranted in suspecting a primary active congestion of the cord or membranes is that in which symptoms identical with those of commencing myelitis, and following a cause of myelitis, pass away in the course of a few days. The cases will be mentioned in the account of inflammation of the cord.

An active congestion, which may be called secondary, seems to result from prolonged and violent action of the nerve elements of the spinal cord. Thus local dilatation and distention of vessels, migration of leucocytes into the sheath and into the adjacent tissue, and even small extravasations, are found in some cases of hydrophobia, of tetanus, and of strychnine poisoning, and it is probable that a similar congestion attends all violent physiological activity, violent and continued muscular exertion, coitus, etc. Coitus several times repeated has been known to cause hemorrhage within the cord. Vascular dilatation, with an increased blood supply, is the physiological attendant of functional activity in all organs, and doubtless also in the cord, but it is morbid only when excessive. It is doubtful whether it causes symptoms unless it goes on, as it probably may, to actual inflammation.

INFLAMMATION OF THE SPINAL CORD—MYELITIS.

Inflammation of the spinal cord is a common lesion. It usually causes a considerable diminution of consistence of the cord. Hence the term "softening of the spinal cord" has been used as a synonym for myelitis, but inaccurately, because inflammation does not always cause softening, and a diminution of consistence may occur without inflammation. The spinal cord differs from many other tissues of the body in that its power of recovering functional integrity, after damage by inflammation, is much less than that of many other organs. The damage produced, and the symptoms which result from that damage, persist for a long time, and are not unfrequently permanent. Hence the distinction between acute and chronic inflammation is to be drawn, not from the whole course of the disease, but from the mode of onset. It is this which indicates the character of the morbid process. *Acute myelitis* is that form in which the symptoms indicating inflammation of the cord come on rapidly and reach a considerable height in a short time, in less than two weeks. When the onset occupies from two to six weeks the inflammation is regarded as *subacute*. When a still longer time elapses before the symptoms reach a considerable degree of intensity the myelitis is termed *chronic*. There is, of course, no sharp distinction between these classes. The subacute and acute forms may be conveniently described together. Other varieties are based on the distribution of the disease. The gray matter may be affected only or chiefly, *polio-myelitis* (Kussmaul, from πολιος, gray), a form that has distinctive characters which make its separate description desirable. When the whole thickness of the cord is affected in a small vertical extent the myelitis is said to be *transverse*. This is one of the most common forms. When an extensive area of the cord is inflamed the myelitis is said to be *diffuse;* when one small area is affected the inflammation is called *focal;* when there are many foci in various parts of the cord the myelitis is said to be *disseminated*. Inflammation of the gray matter, around the central canal, extending into the intermediate gray substance and to a less extent into the cornua, has been termed *central myelitis*. It is said to be sometimes so intense that the gray substance flows out on section, leaving the white substance intact. From this the inflammation of the cornua (commonly limited to the anterior) may conveniently be distinguished as *cornual myelitis*. Both are forms of poliomyelitis, but the latter term has been somewhat anomalously restricted to the affection of the anterior cornu. To this it was originally applied, with the qualification "anterior," but the adjective is now frequently omitted.

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* It may be remarked that the term "congestion" has come to be a popular medical euphemism for a slight degree of inflammation, employed because the public have become acquainted with the word as signifying a less grave lesion than inflammation, and hence the word excites less alarm. Such a use of the word, within limits, is less objectionable than that of many analogous euphemisms, but it is often grossly abused.

ACUTE MYELITIS.

ETIOLOGY.—Little is known of the influence of hereditary predisposition, but myelitis is sometimes met with in members of families in which other evidence of a neuropathic tendency can be traced. Males suffer more frequently than females. Acute myelitis may occur at all ages, but the form which occurs in young children is usually confined to the anterior cornu; other forms of myelitis are most common between ten and forty years. Of exciting causes the most frequent is exposure to cold, especially to wet cold,—exposure to cold winds when the body is heated, lying on damp grass or on snow, bathing in cold water. This cause has been supposed to act by suppression of the functions of the skin, since myelitis has been produced by varnishing the skin of animals,* but it is unlikely that this is the sole mechanism. Exposure of the back to cold sometimes seems especially effective. Myelitis has been produced experimentally in rabbits by freezing the skin of the back with ether spray (Feinberg). Over-exertion is another powerful cause, and is especially effective when combined with insufficient food, and exposure to cold. Hence soldiers on the march are often attacked. The disease has been known to follow a single violent contraction of the dorsal muscles. It followed, in one recorded case, a violent effort to prevent a fall on the ice. It is possible that there is an actual injury to the cord in these cases, or inflammation may be set up in the vertebral column and may spread to the cord. In several cases it has resulted from attempts to lift heavy weights. Thus a man, two days after such an attempt, became rapidly paraplegic and ultimately died, and softening of the cord was found at the level of the fifth and sixth cervical vertebræ. Sexual excesses, and the sudden suppression of the menses and of other habitual discharges, have been supposed to cause the disease.

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* Feinberg, *Virchow's Archiv*, Bd. 59. In Vulpian's laboratory similar experiments failed.

Myelitis, acute and subacute, often results from injuries to the cord of all kinds, lacerations, bruises, and punctured wounds. The readiness with which traumatic myelitis may be produced, has been repeatedly demonstrated experimentally. Simple concussion of the cord may set up inflammation, but the subacute form most frequently results from this cause, and often follows railway accidents. Slow compression is constantly attended by inflammation in the compressed part, as stated in the account of compression; the myelitis thus set up may have an independent course, acute or subacute, and may not be proportioned to the pressure. In cancer of the vertebral bones, for instance, very slight pressure may cause an acute inflammation; there is probably sometimes an extension of inflammation to the cord from the external disease, but such extension is not necessary for the occurrence of the myelitis; the inner surface of the dura mater is often normal. Acute myelitis also results from internal meningitis, in which some degree of invasion of the cord is almost invariable.

Inflammation of the cord occasionally comes on in the course of, or after, some acute diseases, typhus, typhoid, and especially variola, acute rheumatism, and severe puerperal diseases. Whether syphilis is a cause of acute myelitis is a question on which opinions differ. It certainly may cause a subacute form. Attacks of acute myelitis are not uncommon in syphilitic subjects. It seems, on the whole, probable that there is a causal relation between the two, although the nature of that relation, whether, for instance, it is by disease of the vessels, is not known. Subacute myelitis may result from chronic alcoholism, but it is doubtful whether acute myelitis is produced by this cause.

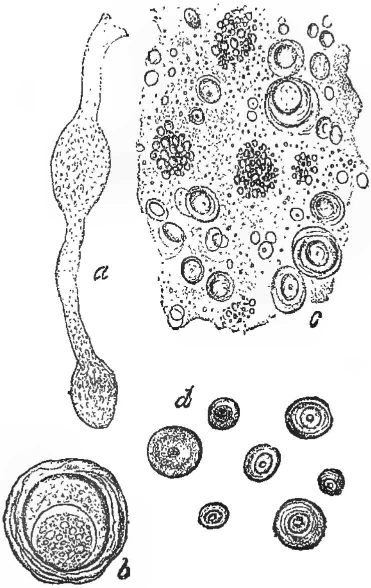
Irritation and inflammation of peripheral organs, as the uterus, bladder, kidneys, intestine, etc., has been supposed to be an occasional cause, but this influence is exceedingly doubtful except in the rare cases in which inflammation of a nerve ascends to the cord. In a considerable number of cases we are able to discover no cause to which myelitis can be ascribed.

Pathological Anatomy.—In recent, acute inflammation of the cord, the pia mater, at the affected part, is usually red and vascular, and the cord itself may be slightly swollen. Its tissue is reddened and often marbled from distended vessels, and the distinction between white and gray matter is indistinct and lost.

The diminution of consistence which occurs in acute inflammation may be so great that the affected part is diffluent, like cream, and flows out when the pia mater is divided. It does not follow that the consistence is lessened to this degree during life; post-mortem softening occurs more readily at an inflamed part. When the vascular disturbance and extravasation are considerable, the condition is termed "red softening:" so much blood may be effused into the tissue that the part looks as if there had been a primary hemorrhage. This form is termed "hemorrhagic myelitis." If the inflammation has existed for some time, the effused blood is altered in tint and the color of the softened part may be rather chocolate than red. After a longer time the changes in the blood pigment cause the color to be lighter, "yellow softening." Ultimately the blood pigment is removed and the softening becomes "white." The period at which these changes of color are found varies according to the amount of extravasation at the onset. This is sometimes very slight, so that the softened part may be white from the first. At a later period the white, opaque aspect is changed to a gray, translucent appearance, from the absorption of the fatty products of degeneration, and an increase in the connective tissue. If the myelitis is very limited in extent, cavities may be formed within the cord. When the destruction of tissue is trifling, the diminution in consistence may be very slight.

The microscopical appearances in myelitis vary much according to the duration and form of the inflammation, and also according to the method of preparation adopted. Observations during the early stage are scanty, since few patients die during the first fortnight. Moreover, the softened tissue does not harden, so that the most diseased part cannot be examined by the most instructive method. In the fresh state the most conspicuous objects are the products of degeneration of the fibres, granules and masses of myelin, and granule corpuscles, with, in older cases, the peculiar bodies termed "corpora amylacea" (Fig. 84, *d*). With these are numerous red blood discs, leucocyte-like corpuscles, and, after the first week, distinct cells, round, oval, spindle-shaped, or angular, with nuclei which resemble leucocytes in size, and are readily mistaken for them. Fragments of axis cylinders may also be seen, granular in aspect, and with irregular swellings (Fig. 84, *a*). After hardening, the appearances vary according to the intensity of the inflammation. The changes in the acute and subacute forms are very similar, probably because, in the parts that can be thus examined, the acute inflammation is less

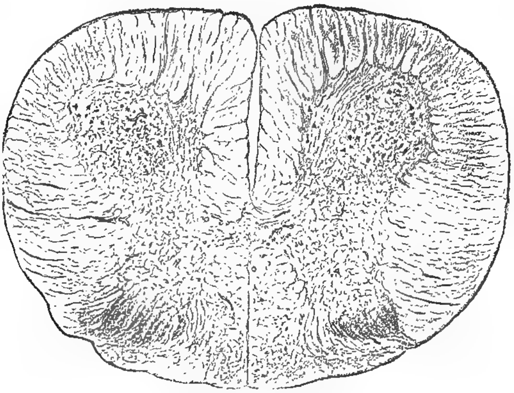
FIG. 84.



Myelitis. *a*, An irregularly swollen axis cylinder. *b*, Section of nerve fibre with swollen axis cylinder. *c*, Section of white substance, with granule masses and atrophied nerve fibres. *d*, Corpora amylacea. (*After Leyden.*)

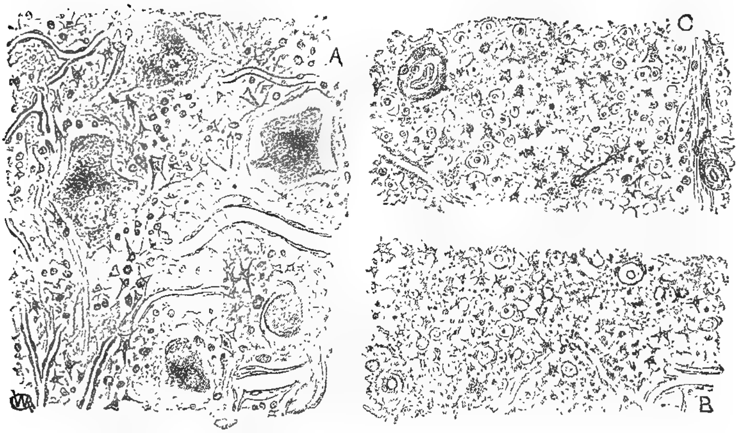
intense than in the softened parts, and because acute cases usually come under pathological observation when the acute stage is over. The products of degeneration are best seen in the fresh state or in sections mounted in glycerine (Figs. 84 and 99). The fragments and globules of myelin in part occupy the position of the nerve fibres from which they have come, in part are aggregated into masses. In sections rendered transparent in the ordinary way, the most conspicuous alteration is the dilatation of vessels, which is often very great. In the white substance it is chiefly the larger vessels that are enlarged, but the gray substance may be crammed with dilated vessels (Figs. 85, 86, A). The nuclei of the capillaries are

FIG. 85.



Subacute myelitis, lumbar region; both white and gray substance uniformly affected. In the latter the numerous distended vessels appear as lines; here and there a larger one is visible.

FIG. 86.

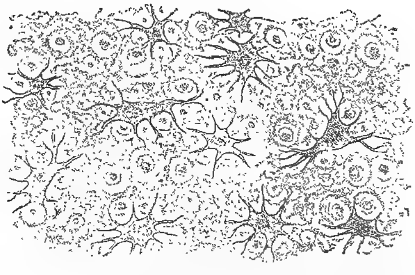


Portions of the section shown in the last figure, more highly magnified. A, from the left ant. cornu; dis- tended capillaries with numerous nuclei; larger vessels with slight distention of perivascular sheaths; numerous round and stellate cells; ganglion cells, swollen, with shriveled processes. C, from front of post. column; scattered nerve fibres separated by amorphous material in which small cells can be seen, round and irregular, some distinct, others indistinct, vessels with thickened walls. B, from near the posterior surface, shows similar changes but with more open spaces from which nerve fibres have per- ished. (*From sections prepared by Dr. Money.*)

enlarged. The walls of the smaller arteries are encrusted with leucocytes in the early stage, and later on, in both white and gray substance, are much thickened by cells, the nuclei of which are often elongated and more or less concentric to the lumen of their vessel (Fig. 89). Outside this thickened wall the perivascular sheath is enormously distended, at first by leucocytes, among which, afterward, other cells are mingled, round, fusiform, angular, with leucocyte-like nuclei (Fig. 89). In transverse section the distended sheath may look, at first sight, like an enormously thickened wall. Red blood corpuscles distend the vessels and are seen also in the adjacent tissue, sometimes uniformly scattered through it (Fig. 89), sometimes aggregated in small extravasations. The gray substance is densely set with round corpuscles, staining deeply, many of which are the nuclei of small, fusiform, or angular cells (Figs. 86, A; 88, B), while the intervening substance is much more granular than in health. The nerve cells are much swollen and granular, often contain distinct globules, strongly refracting, and probably fatty. The cell processes appear shriveled or lost (Fig. 86, A). In slight cases the margins of the cells may be less sharply defined than in health, and the bodies then contain large vacuoles, sometimes wholly within the cells, sometimes partly within and partly outside them in an adjacent albuminous-looking substance.*

In the white substance cleared sections show a great increase and alteration in the interstitial tissue. The change may be uniformly distributed, or greatest in the neighborhood of the vessels (Fig. 89, C). The increase is due in part to an amorphous material, containing nuclei here and there, some of which belong to cells such as have been described in the gray substance; and these may constitute extensive tracts between the spaces where nerve fibres once existed (Fig. 89). In older cases the tissue may have a fibrous aspect in places. The large cells, with many processes, called "spider cells" or "cells of Deiters," are often conspicuous objects (Fig. 87). The processes extend between the nerve fibres. We have seen (p. 124) that their existence in the normal cord has been doubted, but in the inflamed cord, in which they are enlarged, their presence is unmistakable. The changes in the nerve fibres vary much. They always suffer destructive changes, but the products of their degeneration, above described, are scarcely to be seen in cleared specimens. The

FIG. 87.



Subacute disseminated myelitis; part of the posterior column. Nerve fibres separated by granular material and products of degeneration, and by numerous large spider cells. The myelitis in this case was attended by optic neuritis. (*From a section lent by Dr. Dreschfeld.*)

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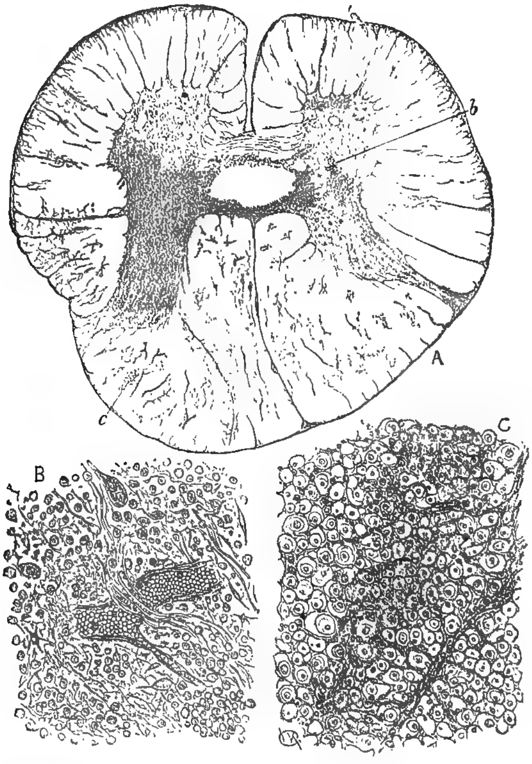
* The significance of this vacuolation has been much discussed. It is probable that the vacuoles form after death, perhaps during the process of hardening, but that they do not form unless the cells have been altered by disease. They have thus some pathological significance.

slightest change in the myelin appears to consist in an alteration in its chemical nature, in consequence of which it stains more readily than in health. Fibres are thus changed adjacent to spots of increase in the interstitial tissue, and they contrast with the unstained healthy fibres in the vicinity (Fig. 88, *c*). With or without this change in the myelin there is an irregular swelling of the axis cylinders, such as is seen on examination in the fresh state. If the fibres are divided transversely at the places where the axis cylinder is swollen this appears much larger than normal (Fig. 84, *b*).

Fig. 88.



Acute transverse myelitis, fatal in three weeks. Carmine preparations. A, Section through most diseased part, at first lumbar segment. Foci of inflammation scattered through the whole area of this cord, most abundant in the posterior columns (the apparent enlargement of which is probably due to the direction of the section). Dilatation of the central canal. B, From the anterior cornu at A, *b*, a distended vessel; the tissue crammed with lymphoid and other cells, among which run normal nerve fibres. C, From the posterior column, A, *c*. The areas staining deeply are seen to consist of thickened trabeculæ, lymphoid cells, amorphous tissue, and also of nerve fibres, the white substance of which stains with carmine while that of the neighboring healthy fibres does not.*

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* I am indebted to Dr. Dreschfeld for the section from which these drawings are made. The patient was a girl aged twenty-three; paralysis, motor and sensory, was complete at the end of two days; the application by her mother of hot bottles to the feet caused such extensive sloughing ulcers that amputation of one leg was deemed necessary three weeks after the onset, and the patient died the next day.

Examples of this will be found in most of the figures. These intense degenerative changes cause the breaking up of the myelin and may involve the destruction of the fibre, the space occupied by it being left empty. In other cases, however, the axis cylinder persists and remains surrounded by a narrow zone of myelin. Often a large number of such shrunken fibres may be found in a part which at first sight appeared devoid of fibres. This shrinking of the fibres occurs not only as a result of pressure on them by the connective-tissue elements, but also where there is little interstitial change and no compression. The relative amount of change in the nerve fibres and interstitial tissue respectively varies very much. In some cases the interstitial increase of tissue seems the primary change and the fibres are altered secondarily, changing, shrinking, and vanishing as they are surrounded and enclosed by the new tissue.

In other cases, or even other parts of the same section, there is but little increase in the neuroglia, although the fibres have suffered extensively; empty spaces are limited by septa but little thicker than normal, although, with more nuclei and often with numerous granules, the nature of which is uncertain. Occasionally there is more diffuse change in the white substance; the neuroglial tissue is increased by small cells and amorphous material in such a manner that all appearance of septa and spaces is lost; a few nerve fibres are included in it, and the spider cells are very conspicuous objects, their processes alone suggesting the septa that exists in the normal condition (Fig. 87).

The distribution of these changes is very variable. The whole thickness of the cord is uniformly affected in pressure myelitis (see "Compression of the Spinal Cord"). In transverse myelitis the alteration is rarely uniformly distributed through a single section, and each part may present variations in the damage in a series of sections taken near together (see Fig. 89*). In meningo myelitis the periphery of the cord is always most affected, and in old cases the thickened pia mater may send tracts of dense tissue into the interior of the cord, from which branching processes of tissue may pass on all sides. An illustration of this is given in the section on chronic myelitis. A similar extension is often conspicuous in cases in which there is no special inflammation of the membranes, and the interstitial changes often extend from the septa that normally pass into the cord and from the walls of the vessels, the course of which may thus be marked out when the vessels cannot be seen. Hence the white columns may be broken up by lines of tissue passing from the surface of the cord toward the gray substance. In dissemi-

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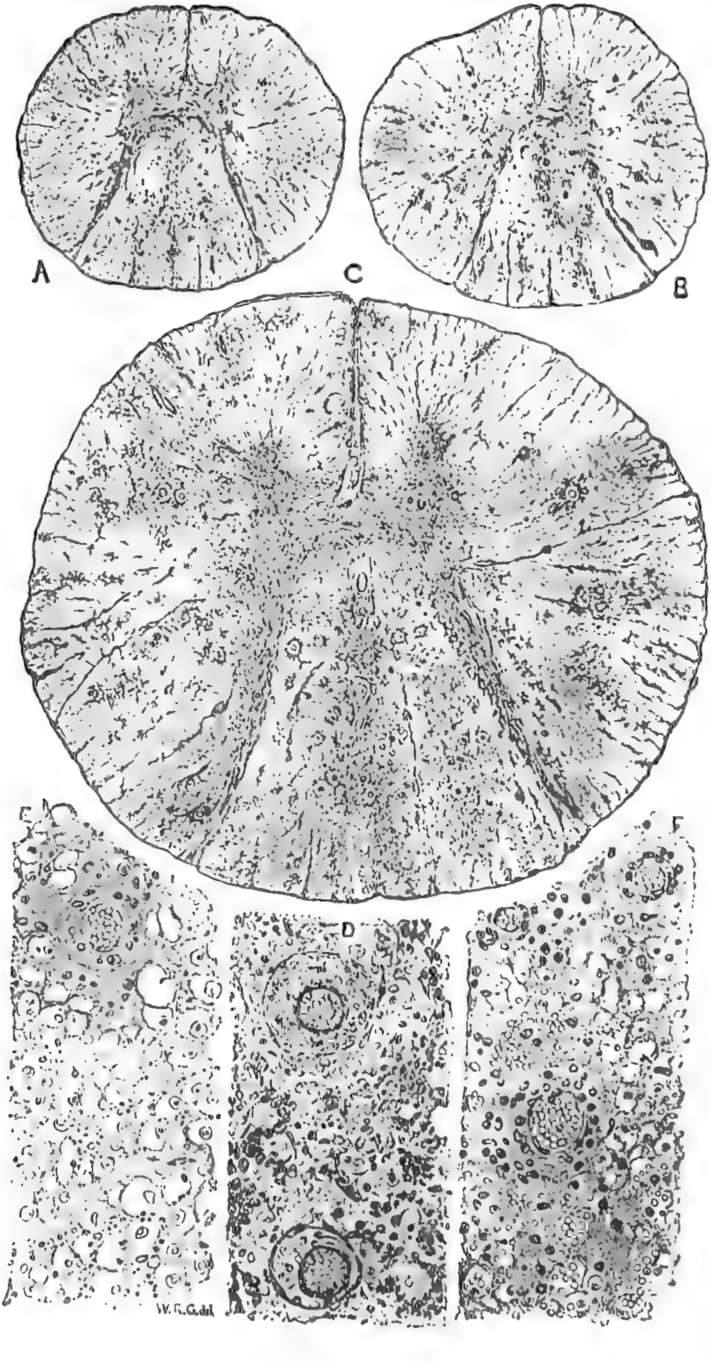
* Fig. 89.—Acute transverse myelitis; from a case fatal in a month. A, B, C, Neighboring sections from the inflamed part, in the mid-dorsal region, to show this varying distribution of the inflammation. (Stained with aniline blue black.) Distended vessels are all surrounded by thick zones of clear tissue, in places divided obliquely, while outside this, ramifying tracts of darker tissue extend into the white substance. In D, from the front of the posterior column, the changes are shown, more highly magnified. The zone around the vessels is seen to consist of the perivascular sheath distended by cellular elements, round, oval, fusiform, angular. The white substance is crammed with similar cells and hardly any trace of nerve fibres or of normal structure can be perceived. In E, from a less affected part of the lateral column, stained with carmine, the alveoli remain, although the tissue between them is thickened, and in many places studded with refracting granules and larger nuclei. In many spaces nerve fibres are seen much smaller than normal in consequence of the wasting of the white substances. F represents a similar condition, with fewer nerve fibres, and extensive infiltration of the alveoli with blood corpuscles. (*From sections prepared by Dr. Money. For the specimen I am indebted to Dr. Hadden.*)

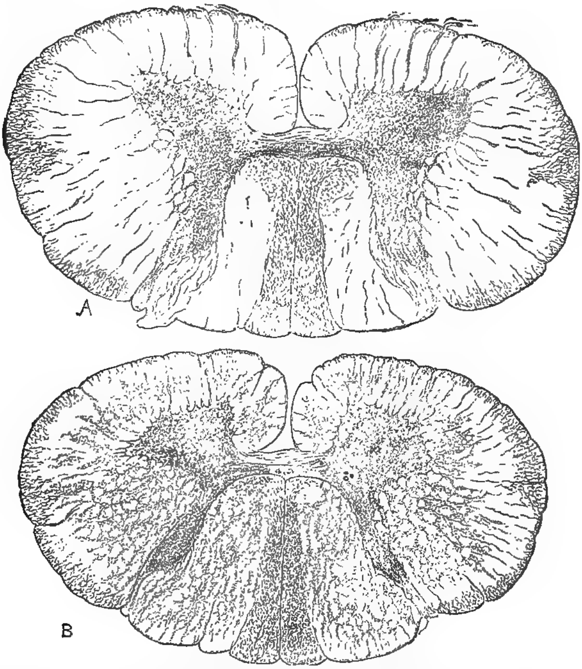
FIG. 89.



nated myelitis foci of inflammation may be scattered through a wide extent of the spinal cord and may appear on the surface as reddish-gray areas, closely resembling those of disseminated sclerosis, but less sharply limited. This form is usually subacute and is that which is often due to syphilis.

The nerve roots coming from a much inflamed part are usually damaged, and may present histological changes similar to those within the cord, distention of vessels, increase of nuclei and of connective tissue about the vessels, breaking up of the myelin, swelling of the axis cylinders. Degenerative and neuritic changes may descend the motor nerves, although rarely to the same degree as in polio-myelitis.

FIG. 90.

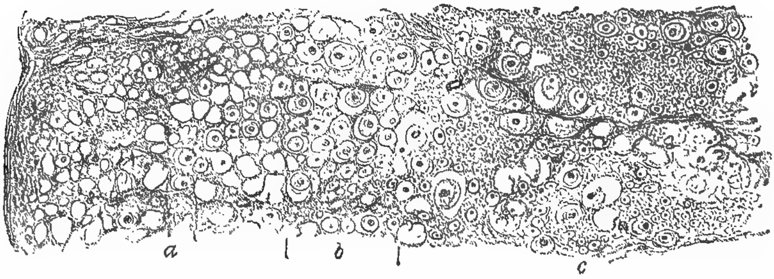


Ascending myelitis from fracture of spine completely destroying the cord in the mid-dorsal region. A, from the seventh cervical, presents only the ascending degenerations in post. med. col., direct cerebel. tract, and ant.-lat. ascending tract. B, from the eighth cervical, presents also numerous tracts of finely granular aspect under a low magnifying power, the structure of which is shown in Fig. 91.

From the foci of inflammation, ascending and descending degenerations pass along the tracts of long fibres that have been already described, downward in the pyramidal tracts, upward in the posterior median columns, the direct cerebellar tracts, and the antero-lateral ascending tract. In cases of transverse myelitis inflammation sometimes ascends the cord for a short distance chiefly in certain tracts. It may, for instance, pass up the pyramidal tracts, so that, an inch or two above the upper limit of the general inflammatory changes, an ascending inflammation of the pyramidal tracts may be combined with an ascending degeneration of the tracts that always suffer secondary ascending degeneration.

If the patient survives, the new interstitial tissue slowly undergoes fibroid changes and contracts, although for a long time cell forms may be distinguished in it. We know very little of the nature of the process that occurs in the cases which slowly recover. The fact that a long period of total palsy may be succeeded by the slow return of considerable power shows that greatly damaged fibres may regain functional capacity. It is easy to conceive that the fibres, which are only so far changed that the axis cylinders are swollen and the myelin stains readily, may speedily recover. The fibres that are shrunken, and in which a narrow layer of myelin surrounds an axis cylinder, may also regain the power of conducting. But we must presume that in these fibres there is no interruption of the axis cylinders. An absolute motor palsy of twelve months' duration must, it would seem by all analogy, involve an absolute interruption of the fibres. But in such a case some return of power may occur. Is this effected by a growth of new fibres such as occurs in nerves? In the lower animals such a growth of fibres has been proved to occur; in man it has not yet been demonstrated. But it is difficult to conceive any other explanation of the clinical facts. The very interesting appearances presented by the section of a cord shown in Figs. 90, B, and 91 suggest strongly a process of regeneration. The cord was crushed in the mid-dorsal region by a fracture of the spine, and motor and sensory paralysis remained absolute, up to the level of the lesion, till the patient's death, six months later. At the seventh cervical the cord presents only the usual ascending degenerations, but at the eighth cervical, opaque tracts extend through the gray matter. Adjacent to these there is everywhere an extensive destruction of nerve fibres. The tracts contain a large number of very minute nerve fibres, each consisting of a fine axis cylinder surrounded by myelin. The fibres are smaller than any met with in the normal cord. The appearance is as if there had been an ascending myelitis, chiefly parenchymatous, which had extended up the cord as high as between the seventh and eighth cervical segments, and from the

FIG. 91.



From the outer part of the front of the lateral column in B, Fig. 90. *a* is the antero-lateral ascending tract, the fibres completely degenerated, except a few, probably of other nature and belonging to *b*, a narrow zone, nearly healthy, of the antero-lateral ground fibres. *c* represents the outer part of one of the finely granular tracts. Under the higher magnifying power these tracts are seen to consist of very minute nerve fibres with a granular material between them in which few cell forms can be distinguished. Adjacent to these tracts there has apparently been extensive destruction of the fibres, large spaces being occupied by products of degeneration and appearing empty in the cleared section.

lower extremity of the normal fibres there had occurred a growth of new fibres such as effects the regeneration of nerves.*

SYMPTOMS.—The symptoms of myelitis vary much according to its degree and form. The most common variety, which may be regarded as the typical form, is acute transverse myelitis, and to this especially the following description applies. The conspicuous symptoms are those which depend on the interference with the functions of the cord, and these, in most cases, are also the first indications of the disease. They are sometimes preceded, and more often accompanied, by general symptoms, such as attend inflammation of other internal organs,—malaise, shivering, headache, depression, loss of appetite, pyrexia,—symptoms which may attend any form of acute myelitis, or may be altogether absent. Their occasional absence is probably explained by the fact that very severe nervous symptoms may result from an inflammation which involves a very trifling extent of tissue.

The spinal symptoms, if not prominent at first, quickly become so. The most obtrusive is the motor weakness, but this may be preceded, for a few hours or days, by sensory symptoms in the limbs—"numbness," tingling, formication, or burning. Occasionally there are rheumatoid pains at the onset, especially in myelitis of the anterior cornua. Pain may be felt in the back, as well as in the limbs, but the vertebral pain is not increased by movement, and soon ceases. Occasionally spasmodic twitchings in the limbs or painful cramps attend the onset. In rare cases, there is a general disturbance of the system. But convulsion occasionally attends the onset of acute myelitis in adults, even when there is no cerebral complication. Three instances of this have come under my own observation.

The motor paralysis usually comes on rapidly, and reaches a considerable degree in the course of a few hours. A patient after such sensations as have been described, or without any warning, finds that his lungs feel heavy; after walking for a few hours he is obliged to sit down and rest. When he tries to walk again his legs feel "as if made of lead." He lies down for an hour, and then finds that he cannot stand, and in a few hours more is unable to raise his legs in bed, although, perhaps, he can still move the toes; next day, even this power is lost. Occasionally the mode of onset is still more rapid, and occupies only a few minutes. The legs are found suddenly to be heavy and tingling, the sufferer sits down on a chair for a quarter of an hour, and then finds that he cannot stand. Such rapid onset resembles that of spinal hemorrhage, and it is probable that in most of these cases there is hemorrhage in addition to inflammation, "hemorrhagic myelitis." Such cases are, however, sufficiently rare not to interfere with the diagnostic rule that a sudden onset means a vascular lesion, and that the characteristic onset of myelitis is rapid but not sudden. Now and then the onset is in the night, during sleep; a patient goes to bed well, and wakes up in the morning with complete paraplegia. On the other hand, the onset of the paralysis may occupy several days, or even a week. When longer than this, from ten to forty days, the myelitis is to be regarded as subacute. Occasionally the onset is by a series of sudden attacks of weakness at intervals of a few hours or days, or even weeks. In the latter case there may be some recovery from one attack before the next comes on. In other instances, there is gradual partial loss of power for a few days, and then complete paralysis comes on rapidly.

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* For these sections I am indebted to Dr. F. G. Penrose. Unfortunately, I have been unable to obtain any other sections of this part of the cord.

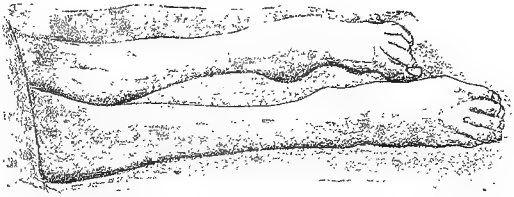
When the paralysis has reached its height, and has ceased to increase, it is usually complete: sometimes it is incomplete, the limbs can be moved, but with little power, or the patient may be able to contract certain muscles but not to move the parts to which they are attached, perhaps only to move the toes. The distribution of the paralysis depends on the position and extent of the disease. In the majority of cases the legs and lower part of the trunk only are affected, because the dorsal region of the cord is the most frequent seat of myelitis. If it is incomplete in the legs, the flexor muscles usually suffer more than the extensors. When the disease is in the cervical region the arms as well as the legs are involved, and the arms are sometimes paralyzed before the legs, probably because the disease, in these cases, begins in the gray matter, and affects the pyramidal tracts secondarily (Hallopeau). The range of motor symptoms, when the disease is at different heights, are indicated in the table of functions given at p. 152.

Sensation is frequently impaired as well as motion. In all cases, except those of trifling degree, it is affected at the onset. It may be absolutely lost up to the level of the lesion. In slighter cases the loss may be partial, and only certain forms of sensation may be impaired. Occasionally there is hyperæsthesia in the limbs at the onset. At the level of the lesion, there is usually a zone of hyperæsthesia, corresponding to the distribution of the nerves that pass through the upper part of the affected region, *i. e,*, through the lowest, merely irritated, portion of the cord above the disease. The hyperæsthesia may readily be detected by passing a hot sponge down the spine; the sense of warmth changes, at the hyperæsthetic zone, into one of pain. Corresponding to this zone of hyperæsthesia there is often a sense of painful constriction, a "girdle pain," which may persist for a long time, even after the loss of sensibility below the lesion has passed away. Its position varies with the position of the disease. It is mostly felt between the umbilicus and ensiform cartilage, sometimes around the lower part of the abdomen, about the anus, or in the legs. It is of much practical importance because it is evidence of the existence of organic disease and also of the upper limit of the lesion. It is sometimes a very early symptom. Any initial pain in the limbs usually ceases when sensation becomes abolished.

The state of reflex action varies, and depends on the position of the disease, in accordance with the laws already stated. An acute lesion in any part of the cord may cause an initial, inhibitory, loss of reflex action in the parts below, but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently there is no initial depression. Subsequently the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately each attains a high degree of exaltation. If the disease involves the lumbar enlargement all forms of reflex action are lost. When in the cervical region, it may be lost in the arms, excessive in the legs. In dorsal myelitis the trunk reflexes are often impaired, and may give important information as to the seat of the disease.

The muscles of the legs are sometimes at first flabby and toneless during the stage of initial depression of reflex action, doubtless from the same influence. This condition soon passes off, if the lesion is above the lumbar enlargement, and as reflex action becomes active, the muscles regain their tone. If, however, the myelitis involves the lumbar enlargement in considerable degree, the muscles of the legs remain flaccid, and lose all influence on the posture of the limbs. The feet fall into an extended position, so that the instep is in a line with the tibia. (Fig. 92.) The muscles also waste rapidly, and present the reaction of degeneration. The wasting in these cases is often extreme. In many cases of myelitis above the lumbar enlargement there is a slighter and slower wasting of the muscles, without any loss of reflex action. The ultimate amount of wasting may be considerable, but it is never so great as when the lumbar gray matter is diseased. There is never the reaction of degeneration, but only a slight change in irritability, similar to each current, at first an increase which, after some weeks or months, gives place to a slight diminution.

FIG. 92.



Posture of feet in myelitis of lumbar enlargement with rapid wasting of muscles.

The sphincters are usually affected from the first, except in very slight cases, and they sometimes afford the earliest indication of commencing lesion. There is usually first retention of urine, and afterward incontinence, overflow or reflex (see p. 163), when the disease is above the lumbar region; if the lumbar centre is diseased there is persistent incontinence from the first. In the former case there is involuntary action, in the latter inaction, of the sphincter ani, and in both cases incontinence of fæces. This may be inconspicuous in the early period, on account of constipation, due to intestinal paralysis. In cases of partial myelitis, as of the anterior cornua, the sphincters are often unaffected. The urine is frequently alkaline in reaction, and the change sometimes comes on so rapidly that it is evidently the result of an alteration in the secretion. In all cases of retention, decomposition in the bladder increases the alkalinity. Cystitis often follows.

The temperature of the paralyzed limbs is usually at first raised one or two degrees above that of the mouth, but it afterward falls, and remains a degree or so lower than that elsewhere. The skin is often dry, sometimes covered with sweat. Its nutrition often suffers and bedsores result. In some cases they are due to long-continued pressure and neglect, but they may occur early and intensely when the disease affects the lumbar enlargement or irritation extends down to it from above. The least pressure on the skin may then cause a bulla containing sanious liquid, and sloughing occurs with readiness, especially over the sacrum. Now and then there is effusion into the knee joints. Occasionally the tendency to sloughing of the skin is so intense that the lesions appear to be spontaneous.

Such trophic disturbances sometimes occur in the cellular tissue of the lower part of the body, especially in the neighborhood of the bladder and rectum. A remarkable example of such disturbance was presented by the case from which Figs. 85 and 86 are taken. Slight symptoms of subacute myelitis were followed by those of cellulitis in the lower part of the abdomen, on account of which the patient was admitted into University College Hospital under the care of my colleague, Mr. Marcus Beck. Subcutaneous emphysema developed about the groins, and suppuration occurred near the rectum. In consequence of this inflammation the patient died, the cord symptoms having slowly increased. After death no lesion of the intestine or local cause of the cellulitis could be discovered. It is probable that the derangement of the trophic influence of the cord facilitates the occurrence of cystitis from retention of urine, and intensifies its effect on the kidneys. Ulceration of the bladder, which sometimes occurs with remarkable rapidity, may be thus produced. A vesico-vaginal fistula may form. Ulceration of the urethra and consequent extravasation of urine have been observed in a similar case.* In another case the inflammation of the bladder was intense, and suppurative cellulitis occurred outside the organ and set up a fatal peritonitis. The kidneys were the seat of suppurative inflammation.†

When the disease is in the cervical region the pupil may be affected. In rare cases of myelitis, optic neuritis has been observed, without any intracranial complication to cause it.‡ It is probably not the result of the inflammation of the spinal cord, but is an associated and similar lesion, the result of the cause of the myelitis. It is noteworthy that most of the cases thus accompanied have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system. In the case of Sharkey and Lawford the optic neuritis reached its height some weeks before the occurrence of the first spinal symptoms, and in the cord were two separate and distant foci of inflammation.

In cases of rapid development the degree attained by the symptoms at their onset may not be exceeded. In some cases a slower increase or extension ensues in the course of a few days. The myelitis, beginning below, may ascend the cord, and the arms and respiratory muscles may gradually become involved. Or inflammation, beginning in the dorsal region, may slowly descend into the lumbar enlargement, probably extending along the gray matter. Reflex action in the early stages is normal or excessive, and afterward becomes lost.

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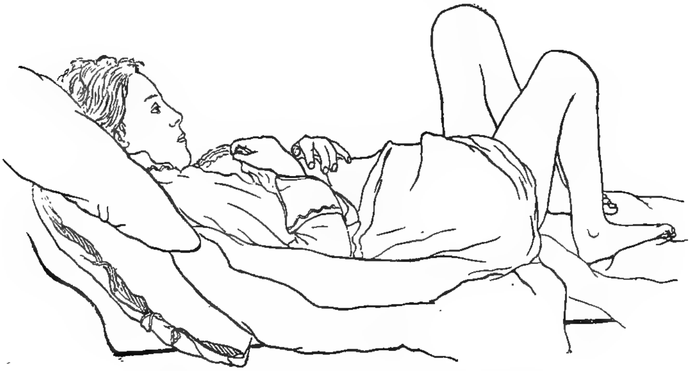
* S. West, *St. Bart. Hosp. Rep.*, vol. x.

† Sharkey and Lawford, *Trans. Ophth. Soc.*, *1883*.

‡ Optic neuritis was present in the case of disseminated myelitis under the care of Dr. Dreschfeld, from which Fig. 87 is taken, and also in that of Sharkey and Lawford just mentioned. See "Medical Ophthalmoscopy," 2d ed., p. 161.

If the patient survives, the course of the symptoms varies according to the intensity of the lesion. The paralysis, motor and sensory, may remain complete. Most frequently sensation is recovered after a few weeks or months, while motor power continues absent for a much longer period. The excessive reflex action may lead to the gradual development of spasm in the legs. This is especially related to the increase of the muscle-reflex action, which follows the secondary descending degeneration of the pyramidal fibres. Ultimately the condition is that termed "spastic paraplegia," and described in detail in another section. The onset of this spasm is not incompatible with the recovery of some voluntary power. The muscles in these cases are often well nourished and may even increase in size; the spasm constitutes a powerful stimulus to their growth. Spasm may, however, coexist with the slow wasting described above (p. 229), but is rarely intense in these cases. Sometimes, especially when the cutaneous reflexes are in excess, and there is little or no recovery of voluntary power, flexor spasm comes on at a later period, at first in paroxysms, alternating with the extensor spasm. Afterward posture may lead to persistent contraction in the flexors of the knee and hip (Fig. 93), which may become so great that the heels are in contact with the buttocks.

FIG. 93.



Flexor contraction of legs in myelitis of the dorsal region.

Death may occur from the extension upward of the mischief, or by failure of respiratory power, often aided by accumulation of mucus in the bronchi, or it may result from the trophic consequences of the disease; bedsores may lead to pyæmia; cystitis and retention may set up suppurative pyelonephritis; or the patient's strength may be slowly worn out, and thus he may die after some weeks or months. When improvement takes place it is usually slow. In cases of moderate severity the recovery may ultimately be complete, but in severe cases it is often imperfect, and some weakness with spasm or wasting is permanent. Rarely there is no improvement. The disease passes into a chronic stage, and the paralysis may remain complete as long as the patient lives, often for years. In some cases, however, improvement occurs even after the paralysis has remained absolute for six or twelve months. In such cases the recovery is never complete, although it is sometimes considerable.

VARIETIES.—The different forms of myelitis, which may be distinguished on pathological grounds, are attended by some differences in the symptoms. The symptoms of *transverse myelitis* have been described as those typical and characteristic of the disease. Strictly speaking, the transverse form is a variety of *focal myelitis*, but the latter term is often applied to the variety in which there is a small area of inflammation not extending through the cord and sometimes limited to a single element. Many cases of anterior poliomyelitis are instances of this form, but these are excluded from present consideration. With this exception, little is known of myelitis limited to a single small focus, because such cases are rarely fatal. Cases are occasionally met with in which symptoms of very limited range come on acutely, and may reasonably be ascribed to such a lesion. They may be one-sided, and motor or sensory, or, as I have more than once seen, there may be acute sensory disturbance with incoördination, limited to a single limb. A much more frequent focal form is *disseminated myelitis*, in which there are two or more foci of inflammation in the same or different parts of the cord. The onset of this form is often subacute, and constitutional symptoms are frequently absent. The most important special symptoms are those which indicate interference with the central functions of the cord in more than one locality. The combinations of symptoms are very variable. In many cases the several foci of inflammation develop successively, not simultaneously, and then we have a corresponding succession of symptoms. Thus, a myelitis in the cervical region, causing atrophy of some groups of muscles in one arm and paralysis of the corresponding leg, may be followed by paralysis of the other leg with such loss of reflex action in it as shows a separate focus of inflammation in the lumbar enlargement, and this again by the development of a girdle pain corresponding to the middle of the dorsal region from a fresh area of inflammation in that situation. When numerous foci of myelitis occur in the dorsal region, as is not uncommon in syphilitic cases and after injury, the symptoms may closely resemble those of a transverse myelitis, because by one or another of these foci each of the elements of the cord is interrupted. The only distinction may be the extensive impairment of the trunk reflexes, or the detection of extensive loss of irritability in the trunk muscles.

In the rare form of *diffuse central myelitis* there is usually rapid loss of power, of sensation, and of reflex action, considerable elevation of temperature, speedy trophic disturbance, and often death at the end of two or three days. Rarely sensation and reflex action have been lowered and not lost. On the other hand, the loss of sensation is said to be sometimes absolute, when motor palsy is incomplete. The symptoms have begun in arms and legs simultaneously, or in either of these, and have accordingly spread upward and downward. Practically nothing is yet known of any slight non-fatal forms of this variety. *Hemorrhagic myelitis* is scarcely a special form, since any acute inflammation of the cord may be attended by a sudden extravasation of blood. Its manifestation is the sudden onset of severe symptoms after slighter disturbance such as indicates a commencing myelitis.

PATHOLOGY.—Very little is known of the pathology of acute myelitis, of the relation of the morbid process to its causes. The spinal cord differs from most organs in that acute inflammation may be very limited in extent. In its great liability to primary inflammation, the cord presents a remarkable contrast to the brain. The special liability of the dorsal region may be associated with the readiness with which this part undergoes post-mortem softening, but we are ignorant of the conditions by which this liability is determined. A question of considerable interest is whether acute inflammation of the cord always begins as such, or whether, in any case or form, it is set up by vascular obstruction, such as thrombosis in a minute vessel. It is conceivable that such an initial lesion may be ultimately lost in the spread of the inflammation it excites. The acute inflammations of the cord that occur in syphilitic subjects seem to have no specific histological characters.

DIAGNOSIS.—Myelitis is recognized by the rapid onset of symptoms indicating structural disease of the cord. The diagnosis may be aided by the presence of such general symptoms as attend the occurrence of inflammation in other organs, but the absence of these is of little negative significance. The position of the myelitis must be inferred from the considerations already described. Its upper limit is indicated by the upper limit of the paralysis; its lower limit by the impairment of the functions of the cord as a central organ (reflex action and muscular excitability) in the parts paralyzed, while the degree to which the various structures of the cord are damaged must be inferred from the character and degree of the symptoms in the affected limbs.

Thus, if called to a patient who has rapidly become paraplegic, the practitioner should first note the degree of motor and sensory paralysis of the legs (indicating whether the lesion is total or partial), and how high up the trunk the symptoms extended. He should then test the reflex action in the affected limbs and trunk, and ascertain the state of myotatic irritability in the limbs. These indicate the condition of the reflex arcs in the lower portion of the cord. Further information on this point is afforded by the state of muscular nutrition, and especially by the evidence of the state of nutrition of the nerve fibres which is revealed by faradaism. It is useless to apply this test until five or seven days after the onset, because four or five days, and often eight or ten, elapse before the degenerative changes occur in the nerve fibres. The examination may then be made without any risk, provided a very gentle current is employed, just sufficient to cause a contraction in the corresponding muscles of a healthy limb, and the current need not be applied to each spot for more than a second. The isolated faradaic shock may be employed with advantage. If no change is found, the examination may be repeated at the end of ten days from the onset. If no muscles, at the end of that time, present any considerable diminution of contractility, it shows that no considerable nutritive change is taking place in the nerves, and that the gray matter from which the nerves proceed is not inflamed. On the other hand, if certain muscles present a diminution of irritability, others being normal, there is a focal lesion in the corresponding gray matter. If all the muscles of both legs present such a failure there is an inflammation of the lumbar gray matter. So, in cases of cervical myelitis with paralysis of all four limbs, the condition of irritability of the arm muscles shows whether the disease involves, or is above, the gray matter in the lower half of the cervical enlargement. If there is impairment of irritability, its extent in the two arms will afford an indication as to whether the myelitis is total, or whether it affects chiefly certain spots in the gray matter (polio-myelitis). This distinction is aided in certain cases by the degree of paralysis of the legs. Cervical cornual myelitis may cause paralysis in the legs, because the inflammation, in slighter degree, involves also the lateral white columns, but the paralysis of the legs is usually incomplete, whereas it is complete if the myelitis is transverse, and sensation also may be lost. The distinction of the other varieties of myelitis depends on their special symptoms which have been just enumerated.

If transverse myelitis has been diagnosed, the question should always be asked, Is it primary, or secondary, to compression? We have seen that myelitis may result, even in acute form, from pressure of the cord. The answer must depend on the discovery of any cause of compression. The spinal column should in all cases be most carefully examined, not merely once, but again and again. When the myelitis is developed there may be no sign of bone disease, and yet these may appear in the course of a few weeks or months. Or the myelitis may be preceded by slight symptoms indicating irritation of the membranes. Myelitis in a cancerous patient should always suggest a secondary growth in the spine. A woman, shortly after the removal of a cancer from the breast, became paraplegic. No evidence of bone disease could at first be found, but in a few weeks the vertebral column became distinctly enlarged from secondary cancer. The diagnosis from spinal *hemorrhage* is chiefly by the mode of onset. In hemorrhage the symptoms develop in a few minutes, and it is only in the cases of hemorrhagic myelitis that any diagnostic difficulty presents itself. The sudden paralysis is then commonly preceded by slight sensory symptoms, tingling, etc., and sometimes by fever. If these are absent in cases of actually sudden onset, we are not justified in diagnosing myelitis.

Cases in which acute myelitis ascends the cord, so that the legs, muscles of the trunk, and the arms are successively paralyzed, bear considerable resemblance to the rare disease termed *acute ascending paralysis*, or "Landry's paralysis," in which no lesion of the cord is found after death. There has been some confusion of the two diseases, as will be mentioned in the account of this malady. The most important distinction is that in such ascending myelitis there is always much affection of sensation, and a strong tendency to trophic disturbance, while, if the patient survives, many muscles waste and present loss of faradaic irritability. In acute ascending paralysis, on the other hand, sensation is unimpaired or is merely dulled on the extremities of the limbs; bedsores do not form, and, if the case is not fatal, there is no change in the electric irritability of any muscles.

In *meningitis* the symptoms of irritation, severe pains, muscular rigidity, etc., are prominent, while they are absent in simple myelitis, in which also the degree of fever is usually less. But in many cases the two conditions coexist, and the predominant lesion can only be decided by the order and degree of the development of the symptoms. In *meningeal hemorrhage* there is severe pain in the back, acute irritation of the nerve roots, and the symptoms are of very sudden onset.

Myelitis is far from rare in patients of the age and sex in which hysteria especially prevails, and many cases are mistaken for *hysterical paraplegia.* The converse error is very rare. The mistake occurs especially in cases of transverse dorsal myelitis, in which there is no wasting of the muscles of the legs. The gradual development of considerable excess of myotatic irritability is a symptom of great diagnostic value, and when this increases to characteristic extensor spasm there should be no room for doubt. Among other conclusive symptoms of organic disease, a girdle pain, and incontinence of urine or fæces, are of especial value. Trophic changes in the skin often sometimes decide, even alone, the nature of the case.

Prognosis.—The danger to life in myelitis depends upon the position of the disease. It is less when the mischief is confined to the dorsal region, greater when the lumbar enlargement is affected, on account of the greater liability to trophic disturbances, which so often cause death. On this account the prognosis is worse in disseminated than in simple transverse myelitis, because some of the scattered foci are so often situated in the lumbar enlargement. The risk to life is greatest, however, when the disease is situated in the cervical enlargement, on account of the danger of fatal interference with the muscles of respiration. The prognosis is therefore rendered worse by indications of a tendency to extension, especially to extension upward. The prospect of recovery of power is influenced by the degree of damage, as shown (1) by the degree of the symptoms, being less when sensation is lost than when there is only motor paralysis; (2) by their vertical extent. Intense general disturbance, high fever and prostration, render worse both the immediate and the ultimate prognosis. Bedsores, if forming early, within the first month, are an unfavorable indication; at a later period they have little influence on the prognosis. The occurrence of cystitis, and especially of any indication of secondary kidney disease, increases very much the gravity of the case. When the acute stage is over, and the disease has become stationary, the prognosis must be influenced by any indications of change that can be detected. As long as sensation as well as motion remain lost, no recovery can be anticipated, but if the sensibility of the skin has become normal, some ultimate recovery of motor power may be looked for. The development of even an intensely spastic state of the legs does not preclude recovery of the power of walking, indeed the spasm often enables the patient to stand with a slighter degree of voluntary power than would suffice if the limbs were supple. If paralysis has remained absolute for about six months, complete recovery cannot be hoped for, but some return of power, sufficient to enable the patient to walk, may occur after even twelve or eighteen months' absolute paraplegia.

The fact of preceding syphilis must be allowed very little influence on the prognosis, provided confidence is felt that the patient is suffering from a primary myelitis and not from any secondary consequence of a syphilitic growth.

TREATMENT.—If a case comes under observation at the earliest period, when only slight sensory disturbance and slight weakness of the legs indicate the commencing process, the question arises whether any treatment can avert the further development of the inflammation. If the symptoms are clearly due to exposure to cold, a hot bath, followed by free diaphoresis, should be employed, and seems sometimes efficacious. If considerable paralysis shows that the process of inflammation is fully developed, little can be expected from these measures, and it is better not to subject the patient to treatment that is incompatible with perfect rest. This, in all cases, is of paramount importance. Both functional excitation of the cord and movement of the spinal column should be avoided. The remarks regarding posture made in the account of the treatment of inflammation of the membranes apply also to that of the cord itself; it is most undesirable that the spine should be the lowest part of the body, and it is rather less difficult to keep the patient off the back in myelitis than in meningitis. If there is any reason to suspect hemorrhage, or if there are indications of rapid extension of the inflammation, the prone position should be adopted for a time, if possible.

The removal of blood from the skin of the back over the affected region, by leeching or wet cupping, is an old measure, which finds some theoretical justification in the fact that the blood from the structures behind the spine passes into the same venous plexuses as the blood from the spinal cord itself. Hence this measure may conceivably have some influence on the circulation in the cord. If the patient's strength is not such as to render the abstraction of blood desirable, dry cupping may be employed, or the vessels of the skin may be dilated by hot fomentations, or a mustard plaster, or hot-water bags. By stimulating the cutaneous nerves, these agents may also influence, in a reflex manner, the vessels of the cord. The application of cold to the spine, as by a spinal ice bag, has also been recommended. Contrary as these therapeutic agents seem, it is probable that each moderates local inflammation in the same manner, by causing first contraction and then dilatation of the vessels of the inflamed part, and so lessening the tendency to stasis of the blood, on which some effects of inflammation depend. Unless there is reason to suspect hemorrhage, the application of warmth seems the safer of the two. At the very onset of inflammation mild counter-irritation is unquestionably useful, and even a blister may be employed, but when the process has reached a considerable degree, it is very doubtful whether counter-irritation has much influence until the acute stage is over.

In other respects the treatment of acute inflammation of the cord must be guided rather by the nature of the process than by the character of the organ in which it occurs. A nutritious but unstimulating diet, aperients, and diuretics, are desirable in all cases. It is extremely difficult to ascertain the influence of the drugs which are supposed to exert a special influence on the inflammation. Ergot was recommended by Brown-Séquard chiefly on theoretical grounds, and has been extensively employed. In rare cases it has seemed to do good. In cases of hemorrhagic myelitis it may reasonably be given with greater confidence, or ergotin (3 to 5 gr.) may be injected beneath the skin. Belladonna has also been recommended, but its influence is doubtful. Mercury has been largely enployed, given by the mouth and by inunction. The influence of mercury on the inflammation of internal organs does not seem so great as upon the inflammation of fibrous tissues and the structures that invest organs. Certainly in myelitis its effect is less distinct than it is in many cases of meningitis. Iodide of potassium seems to be even less efficacious than mercury. In cases of myelitis occurring in syphilitic subjects the treatment for syphilis seems to have but little influence on the morbid process. It is true that such treatment is rarely adopted at the very onset of acute myelitis, but after the disease is developed energetic treatment does not seem to modify the subsequent course of the disease. This is true, also, of the subacute disseminated myelitis which often occurs in the subjects of syphilis. I have known this form, concurring with syphilitic disease of the cerebral arteries, to develop and run its course to a fatal termination in spite of continuous antisyphilitic treatment. It has already been pointed out that no form of acute myelitis in syphilitic subjects presents any special histological characters. Whatever be the relation of non-specific morbid processes to syphilis, the influence of antisyphilitic treatment seems to be for the most part confined to lesions which present a specific character. Hence, in cases of myelitis in syphilitic subjects, while it may be desirable to treat the patient in accordance with the constitutional state, much effect on the morbid process must not be anticipated from the special treatment. The conclusion is that, as stated at the onset, in acute myelitis our chief reliance must be on the general measures suggested by the process of inflammation rather than on special agents. At the same time it may be thought well to use the latter in order to employ all possible means which afford even a chance of lessening a process, the most trifling diminution in which may have a profound influence on the ultimate condition of the patient.

In the general management of a case of myelitis, two points are of extreme importance. One is to avoid, by extreme cleanliness and care, the exciting causes of bedsores. The skin should be most carefully watched, and any indication of deleterious pressure met by a change of position or an alteration of the mode of supporting the part. Cotton wool is very useful for this purpose. If there is a marked tendency to trophic changes the patient should be placed on a water bed. When there is incontinence of urine, the difficulty of avoiding irritation of the skin is greatly increased.

For males, a bed urinal is sometimes useful, but often it causes irritation and sloughing of the prepuce, and then does more harm than good. A quantity of boracic absorbed cotton wool, changed as often as it becomes saturated, is one of the best means of meeting this difficulty. It must be remembered that the prevention of bedsores is the prevention of one common cause of death. Should offensive sores unfortunately form, a quantity of picked oakum, placed outside the immediate dressing, is a cheap and most effective means of preserving the air of the room from the fœtor of the sores, and therefore of increasing the comfort and well-being of the patient and his attendants. The second point in management is the treatment of retention of urine. If there is either simple retention or overflow incontinence, the bladder must be regularly emptied by the catheter and great care exercised to prevent the introduction of contaminating germs. If the bladder is left full and the urine allowed to dribble away, inflammation is sure to be set up, and probably also pyelo-nephritis. If cystitis occurs, antiseptic washes must be used to lessen, as far as possible, the decomposition of the urine.

When the disease of the cord has become stationary, the patient may be allowed to move and a more tonic treatment may be adopted. Iron, quinine, or arsenic may be given. Strychnia must be given only in very small doses if there is any excess of reflex action. Occasional counter-irritation may be employed, and frequently if any improvement seems to result. The limbs may be rubbed, and any muscular wasting treated with electricity. It is not desirable to use electricity as a therapeutic agent while the cord disease is in an acutely active stage. There is no evidence that the application of electricity to the spinal column has any influence on the process of recovery of the cord. Its chief value is to maintain, as far as possible, the nutrition of any muscles of which the nerves have undergone degeneration. In cases of dorsal myelitis, in which the legs are well nourished, and the reflex action is excessive, it is better not to apply any form of electricity. The unavoidable stimulation of the sensory nerves tends to increase the reflex over-action. Careful attention should, in all cases, be paid to the position of the limbs, during the stage of helplesssness, so as to avoid as far as possible the development of contractions. For the condition of active spasm which often develops after severe myelitis, not much can be done; such special treatment as can be adopted is described in the chapter on "Primary Spastic Paraplegia."

ABSCESS OF THE SPINAL CORD.

Simple inflammation of the spinal cord scarcely ever goes on to the formation of pus. In polio-myelitis, leucocytes may accumulate at certain points of the gray matter so densely as to constitute microscopic collections of pus. In very rare cases of this kind such minute abscesses have been sufficiently large to be visible to the naked eye. Pus only forms in the substance of the cord in considerable quantity in cases of purulent meningitis. In most instances the purulent meningitis has been of septic origin, rarely traumatic. In the former case, suppuration within the brain may coincide with that in the spinal cord. The latter may be very extensive. The symptoms are those of an acute irritative spinal lesion, but they are often lost in those of the purulent meningitis which precedes the disease of the cord itself. In the diagnosis of the condition, the presence of a cause of septic suppuration is the most important element in the determination of the nature of the acute spinal symptoms. A good example of the disease is a case recorded by Nothnagel. A patient suffering from cough and most offensive expectoration was suddenly seized with severe pains on both sides of the abdomen, attended by a sense of constriction, and quickly followed by paralysis of the bladder and of the legs, with loss of sensation and of reflex action. An abscess of the cord was diagnosed. After death there was extensive purulent spinal meningitis, and the dorsal and lumbar cord contained an extensive collection of gangrenous offensive pus, which seemed to occupy the central part of the cord, from the cervical enlargement downward. Some abscesses were found also in the white substance of the brain.

EMBOLISM OF THE SPINAL CORD.

The occurrence of embolism in the spinal cord has not yet been proved, but a few cases have been recorded which suggest the possibility that the process has been the exciting cause of an acute myelitis in very rare instances. In a young man with mitral regurgitation, considerable weakness of the right leg came on suddenly, with transient spasm. The onset idicated an acute lesion in the cord, which might well have been the embolic obstruction of a small vessel. In a case recorded by Weiss, a boy, aged sixteen, with chronic mitral disease, was suddenly seized with complete paraplegia, followed by bedsores, etc. He died four months after the onset, and the lumbar enlargement was found completely softened, with old coagula in the arteries. There was embolism of the kidneys and spleen, and the cortex of each cerebral hemisphere presented small foci of softening. Such cases justify a suspicion that embolism may be the cause of a sudden lession of the cord in a patient in whom a source of embolism exists and the process has occurred in other organs.

CHRONIC MYELITIS.

The spinal cord is occasionally the seat of chronic inflammation, which develops slowly in the course of a few or many months. The lesions, which can be regarded with most accuracy as of this nature, resemble acute myelitis in seat and distribution, and intermediate cases connect them with the analogous subacute forms. They differ from acute inflammation both in the longer time occupied in their development, and in the absence of the considerable vascular disturbance which forms part of the acute process. Such chronic myelitis may be focal, disseminated, or diffuse. In the former case it may involve the whole thickness of the cord at a certain level, *chronic transverse myelitis,* or only part of it, sometimes one-half, occasionally for a considerable vertical extent. The *chronic disseminated myelitis* may resemble the corresponding subacute form in distribution, many points of inflammation being scattered through a certain region of the cord, or it may affect almost all parts of the cord, and then ultimately become diffuse by the union of the areas of disease.

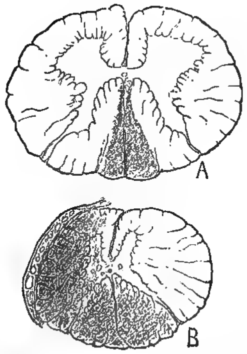
The term "chronic myelitis" has, however, been used in a much wider sense than that here indicated. There has been a tendency in recent pathology to extend the conception of inflammation so as to include all morbid processes which do not consist in an actual new growth. The extension is chiefly based on the difficulty of drawing a dividing line between inflammation and other processes. But it is often useful and even necessary to distinguish where we cannot divide, and it may be doubted whether any clearer conception of pathological processes has been obtained by a generalization which is hardly more than the forced extension of an elastic name. The application of this method to the spinal cord has caused the term "chronic myelitis" to be applied to all chronic diseases other than tumors. Thus the chronic lesions that are limited to certain structures of common function, and probably begin by an atrophy of the nerve elements ("system diseases"), have been described as "chronic parenchymatous myelitis" (Hallopeau and others). Such processes have so little in common with the forms of chronic inflammation above mentioned that it is more convenient to follow the example of those who consider them separately, as a class of "degenerative diseases," in spite of the fact that, in rare cases, the morbid process may develop with some rapidity, and indications of a secondary inflammation may sometimes be discovered. For practical purposes, it is best to base our distinctions on the salient differences of general character and course. We are not therefore precluded from recognizing the points of contact between the various groups of diseases, or the presence of common elements in those that are, in the main, dissimilar. Hence we will restrict the term "chronic myelitis" to the processes already indicated as those to which it is strictly applicable.

The irregular distribution of the lesion in "disseminated" or "insular sclerosis" may seem a special justification for including it, as many have done, under "chronic myelitis," but it presents so many peculiarities that it also is most conveniently described apart.

Etiology.—In the causation of chronic myelitis an inherited neuropathic tendency can sometimes, although rarely, be traced. The disease is most common in the male sex and in early and middle adult life, but it occurs occasionally in old age. Many cases are met with in the subjects of constitutional syphilis, so that a causal influence of syphilis is highly probable. In most instances the primary syphilis was contracted many years before the myelitis came on. Various conditions which lessen constitutional strength predispose to the disease. The exciting causes of acute myelitis seem also capable of giving rise to the chronic form, usually when acting for a considerable time, or frequently repeated. Thus, while a single intense exposure to cold may produce acute myelitis, frequent habitual exposure may cause the chronic form. Repeated over-exertion has also seemed effective in some cases. Sexual excess has been thought to be a cause, but on less clear evidence. Chronic alcoholism certainly sometimes excites chronic myelitis, usually in association with chronic meningitis; the inflammation that results from this cause is often more rapid in onset than most other forms, and sometimes deserves the name subacute rather than chronic. Lead poisoning is said to be also an occasional cause. Chronic myelitis may be secondary to other processes, as, for instance, to chronic meningitis. The inflammation that results from pressure on the cord may be chronic as well as acute. Lastly, acute inflammation may pass into a chronic stage, in which the damage to the cord not only persists but slowly increases.

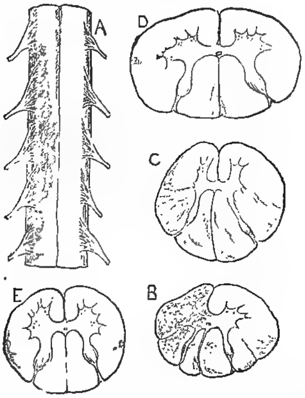
Pathological Anatomy.—The morbid appearances vary much in the different forms. In most cases the disease is visible to the naked eye by a gray discoloration of the white substance, from which the gray matter may be indistinguishable. The tint may be visible on the surface, even through the pia mater, which may be thickened at the spot. The consistence of the affected area may be lessened or increased; when diminished, small cavities sometimes exist in it. Rarely there is a slight increase in the size of the affected part. In some recorded cases in which there was a great increase in

Fig. 94.



Chronic myelitis. (*After Charcot and Gombault.*) B, section at the level of the third dorsal, shows both the posterior columns and whole of left half inflamed, except the inner part of the anterior column. The affected part was gray and uniform in aspect, vascular, and firmer than normal. Pia mater and arachnoid thickened. A, section from cervical enlargement, showing secondary degeneration in the post. med. columns. Similar foci existed in the pons, etc.[*]

Fig. 95.



Chronic sclerotic myelitis. (*After Troisier.*) A, posterior view of the affected part, lower dorsal region. The diseased areas were gray in aspect and are indicated by shading. B, middle of lesion, showing extensive damage; C, 2½ cm. higher up, slighter damage; D, cervical region ascending degeneration, post. med. cols., and a spot of disease in lateral column (probably part of the ascending ant.-lat. tract, secondarily degenerated). E, lower part of lesion. (*See next figure.*)[†]

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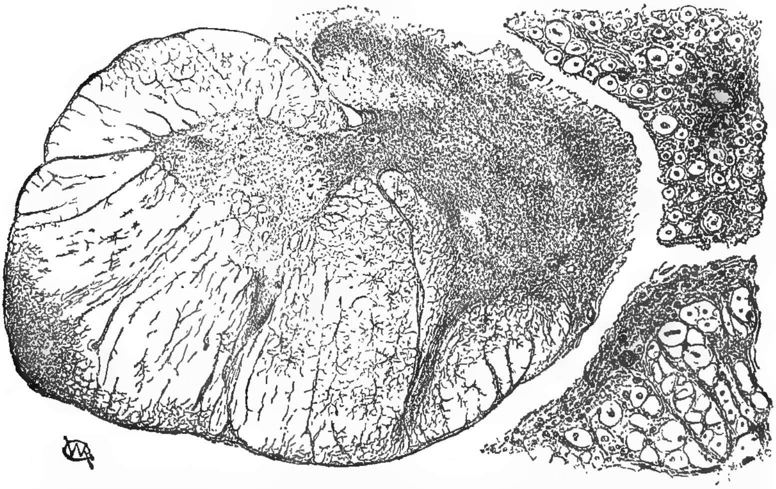
[*] The patient had had syphilis twenty-one years before. The spinal symptoms slowly developed during fifteen months and consisted in paralysis with hyperæsthesia on the left side, with loss of sensibility without paralysis in the right, and a band of anæsthesia to pain around the thorax at the level of the lesion, but the mental dullness of the patient prevented accurate observation during the last few months of her life. (Charcot and Gombault, *Arch. de Phys.*, *1873*, vol. v, p. 143.)

[†] The patient was a woman forty years of age. The symptoms slowly developed during the three years before death, and consisted of weakness with rigidity in the left leg, and impaired sensibility in the right, first of the temperature sense and afterward of touch. No observations were made on sensibility during the last fifteen months of the patient's life, during which the lesion was slowly progressing. (Troisier, *Arch. de Physiologie*, *1873*, vol. v, p. 716, Case 2.)

size, it is probable that an infiltrating glioma has been mistaken for chronic inflammation. In many cases, especially when the affected part is indurated, it is also shrunken, and the shape of the cord may be changed, as in Fig. 95, B, in which almost the whole of one-half of the cord is intensely damaged. When there is one considerable focus of disease there are often other slighter foci in the neighborhood. In what is called the diffuse form, areas of disease may be scattered through almost the whole extent of the cord. It begins at many separate places, and so is, strictly speaking, disseminated, but the areas of inflammation may blend, so that, in the ultimate distribution, it is a diffuse inflammation. In the secondary form, the inflammation is usually confined to the neighborhood of the morbid process which causes it. That which is secondary to meningitis is most intense in the peripheral part of the cord, sometimes, however, extending deeply, especially when the cause of the meningitis is one, such as alcoholism, capable also of giving rise to myelitis. In this condition almost the whole thickness of the cord may be involved (cf. A and B, Fig. 97). Now and then, when there is no primary meningitis, the superficial layers of the cord are chiefly damaged, a form that has been termed *chronic annular myelitis* or *annular sclerosis* because the inflammation extends like a ring around the cord.

FIG. 96.



Chronic sclerotic myelitis, same case as Fig. 95; section near B, reversed. Almost the whole left half of the cord (right in the figure) is changed into a dense mass of shrunken connective tissue, and the right half is being invaded in many parts by the same process. The upper small figure is from the posterior column, showing a thickened vessel surrounded by nucleated tissue, among which lie nerve fibres, many of them much smaller than normal and some with swollen axis cylinders. The lower small figure is from the right anterior column (left in fig.), the pia mater is thickened and contains many nuclear elements; from it thick trabeculæ extend into the white substance and enclose spaces, from many of which the nerve fibres have perished.*

The microscope shows that the inflammation is essentially interstitial. The affected areas stain deeply with carmine in consequence of the increase in the connective tissue. The parts most altered may be occupied by a dense ibrous reticulum, in which few nerve elements can be discovered, as in Fig. 6. In less affected parts of the white columns there is an irregular increase f the interstitial tissue, which has in part a fibrillated aspect, in part the ppearance of an amorphous substance, studded with nuclei (Fig. 96, upper mall figure), while in other parts there may be many nucleated cells, oval, usiform, or stellate. The large stellate "spider cells" are sometimes very

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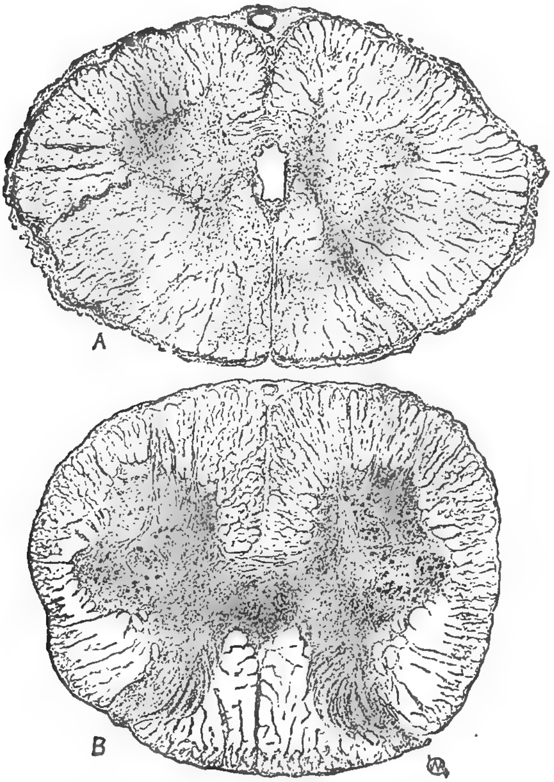
* From a section by Prof. Pierret, kindly lent me by Dr. Dreschfeld.

Fig. 97.



Chronic meningo-myelitis, due to alcoholism. Syringo-myelia. A, lowest part of cervical enlargement; pia mater thickened, and from it tracts of tissue extend into the cord. The greater part of the section is the seat of a diffuse myelitis, in which the nerve fibres could be seen with difficulty, and were separated by inflammatory products. The gray matter was also affected, and the nerve cells much changed. The clear spaces in the right ant. cornu appeared occupied by products of degeneration. Central canal greatly dilated and surrounded by new growth, which in B (lumbar section) has obliterated the canal and thus caused its dilatation above. In this section the myelitis is chiefly peripheral and the gray matter has, for the most part, escaped.

onspicuous. The nerve fibres waste before the interstitial process. Some-mes they at first undergo the change mentioned in the account of acute yelitis, by which the myelin stains more deeply than normal; afterward e white substance becomes narrowed, and the axis cylinder irregularly wollen, and ultimately many or all the fibres disappear from the affected rea. Here and there the destruction of fibres may be out of proportion to the interstitial growth, so that thickened and granular trabeculæ enclose empty spaces. This is seen in the lower small illustration in Fig. 96, which also shows the thickening of the pia mater over the affected area. In the recent state, products of degeneration, granule corpuscles, etc., may be found abundantly near the altered regions. The walls of the vessels are thickened, and sometimes the lumen of small arteries may be obliterated. Around the arteries the increase of interstitial tissue is often more intense, and it seems to spread thence into the adjacent structures. Undue significance has been attached to this condition in old patients with arterial degeneration; it has been regarded as evidence that arterial disease sets up the myelitis (Demange), but it is a common appearance in all forms of chronic myelitis and also in the later stages of acute inflammation. In the gray substance there is a similar increase of connective-tissue elements, so that the altered part takes a deeper stain with carmine than the normal gray substance. The nerve elements become atrophied, the ganglion cells, at first swollen and globular, afterward shrink, until ultimately they may be reduced to small angular bodies, and they even may disappear. The nuclear corpuscles about the central canal are often increased in quantity, and the canal obliterated, but this condition is so common in otherwise normal cords, that no importance can be attached to it. A considerable increase of tissue about the central canal, closing it below and causing it to be dilated above (Fig. 97), "syringo-myelia," has been ascribed to chronic inflammation around the canal, but the new tissue is probably of the nature of a growth, and it is doubtful whether the condition results from simple inflammation.

In chronic meningo-myelitis (Fig. 97) the pia mater is greatly thickened, and dense tracts of tissue may pass from it into the superficial layers of white substance. From these a slighter interstitial overgrowth extends in various directions. As already mentioned, this change may be accompanied by a deeper and sometimes by a more diffuse inflammation, as in A, Fig. 97, in which the affection of the cord is greater and that of the membranes less than in B. The chronic myelitis that occurs in syphilitic subjects may present no specific characters. In some cases, however, the cell formation has been abundant and has been associated with a tendency to fatty degeneration in the older portions of the lesion. The condition is, however, clearly one of chronic inflammation, and not of a syphilitic growth.*

The less chronic the inflammation, the larger is the proportion of cells and nuclei in the interstitial tissue; the more chronic, the greater the fibrillation it presents, and the firmer the consistence of the altered part. The very chronic indurating forms are sometimes called "sclerotic myelitis," or "focal sclerosis of the cord." From the various areas of inflammation secondary degenerations proceed, ascending or descending, and these may complicate the aspect of the disease at higher or lower levels.

Symptoms.—The symptoms of chronic myelitis vary much according to the character of the morbid process. Different cases may manifest almost every symptom that can be produced by disease of the spinal cord, acute spasm alone excepted. Severe pain, however, is not common. There may be various motor and sensory paralyses, contractures, and even muscular atrophy. The common feature of all cases is the slow, gradual, and often successive development of the symptoms. In one of the most common forms, focal myelitis of the dorsal region, partial or transverse, the symptoms are paraplegic. They are the same if there is a single area of disease or if several foci develop near together. Motor power is usually impaired far more than sensibility. Subjective sensations are often prominent and early symptoms, whether there is anæsthesia or not,—tingling, "pins and needles" in the legs, a sensation as if fur or wool covered the skin, sometimes with dull pain in the legs and back, especially after exertion, and commonly also a well-marked girdle pain at the level of the lesion. The onset of the motor weakness is very gradual; the patient gets tired sooner than before, and actual weakness gradually forces itself on the patient's notice. Months, sometimes years, pass before the power of walking is considerably impaired. As the legs get weak, excess of reflex action usually develops, with the myotatic excess that indicates a descending degeneration of the motor fibres. The knee jerk becomes increased and a foot clonus can not only be obtained, but occurs from time to time when the legs are in certain positions, and a tendency to spasm gradually develops. The sphincters usually, but not always, share the impairment of voluntary power. Sensation may be unaffected, but more often some loss is found, by a careful examination, to touch, or pain, or temperature, and sometimes the impairment is considerable in degree. When there are several foci of disease, some functions may be much more impaired than others. Thus the usual rule may be reversed, and sensibility may be more affected than motion. The symptoms may come on simultaneously in the two legs, or one may be affected before the other, and occasionally the paralysis reaches a high degree in one leg while the other still possesses fair strength. This is the case when one-half of the cord is affected in considerable degree, as in the lesion shown in Fig. 96. There may then even be a crossed paralysis of motion and sensation.

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* See Moxon, *Guy's Hosp. Reports, 1871*, and Charcot and Gombault, *Arch. de Physiologie, 1873*.

If chronic myelitis develops in the cervical and lumber enlargement, the symptoms are often very irregular in distribution, and muscular wasting is usually added to the other symptoms. This occurs in the widespread, diffuse form. In this, irregular muscular wasting may be the most prominent symptom, but it is always combined with other symptoms, palsy and anæsthesia, also irregular in extent and seat. The muscular atrophy may be attended by a slow failure of electrical irritability to both currents, but often some voltaic irritability in the muscular fibres persists longer than the faradaic irritability of the nerve fibres, and sometimes there is a well-marked degenerative reaction. The symptoms may commence in the arms or in the legs, but they ultimately become general in most cases, and, according to the place of their commencement, may seem to have an ascending or descending course. The cases were described by Duchenne under the name of "*general spinal paralysis.*" In rare cases of this character, in which the disease is limited to the white substance of the cord, there may be little or no muscular wasting, but extensive paralysis and contracture, with increased myotatic irritability. Occasionally the disease begins in the dorsal region, and after a time, perhaps years, it spreads to the enlargements.

The course of chronic myelitis is usually slow and progressive. The symptoms often increase very gradually, and may only attain a considerable degree at the end of two or three years. At any stage the progress of the disease may undergo arrest. The chronic course may be varied by occasional more rapid increase of the symptoms, due to subacute or even acute processes in the diseased part. Every degree of chronicity is met with, and subchronic cases, in which the symptoms develop in the course of a few months, effect a gradation to the acute and subacute forms of myelitis. The duration of the disease varies from six months to twenty years or more. Thus in one case the symptoms slowly increased during about seven years, and then became stationary, and the patient died twenty-three years after the onset.

Diagnosis.—The diagnosis of chronic myelitis rests on the slow development of symptoms indicating a random process in the spinal cord, *i.e.*, a process which damages irregularly structures of various functions, and thus cannot be looked on as a "system disease." The maladies from which chronic myelitis has to be distinguished differ according to the seat and character of the inflammation. Dorsal transverse or focal myelitis may be confounded with compression of the cord and with primary spastic paraplegia. The distinction from compression rests on the absence of a cause of compression, such as bone disease, and the absence, also, of the signs of a morbid process outside the cord, preceding those of damage to the cord itself. Such signs are chiefly the irritation of nerve roots; in myelitis such irritation is never intense and is often absent, whereas it is usually a prominent symptom in all cases of compression. A tumor within the spinal cord also causes, in most cases, more irritation of the nerve roots than does chronic myelitis. If myelitis involves one-half of the cord much more than the other, the symptoms may closely resemble those of an intra-medullary growth. But a tumor never causes considerable damage to one-half of the cord without interfering considerably with the functions of the other half, and the symptoms in chronic myelitis may be strictly unilateral. The distinction from primary spastic paraplegia, the so-called "primary lateral sclerosis," is often one of great difficulty. The motor state of the legs may be identical in the two diseases; in each there is the same extensor spasm, and in each there is a slow, gradual, and apparently simultaneous onset of the weakness and spasm. The distinction is that in primary spastic paraplegia the symptoms are purely motor; there is no indication that the lesion extends beyond the motor structures. In chronic myelitis, on the other hand, there is such indication, either by the impairment of sensation or by the presence of a girdle pain. This distinction is sufficient in the majority of cases. But, as we shall see when we consider primary spastic paraplegia, the pathological distinction between the two diseases is not well defined, is indeed less definite than is, in the majority of cases, the clinical distinction.

The diffuse form of chronic myelitis has to be distinguished chiefly from pachymeningitis and degenerative muscular atrophy. In pachymeningitis, the muscular wasting may be similar, but anæsthesia is usually much more considerable in range and in degree, and there is often much more pain in the back. If there are similar symptoms in both arms and legs, myelitis is far more probable than pachymeningitis, since chronic inflammation of the membranes is less extensive than that of the cord. From progressive muscular atrophy the chief difference is in the random distribution of the wasting, the absence of symmetry, and the indications of irregular damage to other structures in the cord. Sensory symptoms are rarely absent in this form of chronic myelitis, while in progressive muscular atrophy they are limited to occasional dull rheumatoid pain.

PROGNOSIS.—Chronic myelitis is a very grave disease, on account of the intractability of the morbid process, and the persistence of damage which is slowly produced. In acute inflammation there is not only a possibility of repair, but there is a tendency to it, which is absent in the chronic process. Hence, while arrest of the disease occurs not unfrequently, considerable recovery is rare. The prospect of improvement is equally small, whatever is the seat or extent of the disease. The prognosis, however, is less unfavorable the less chronic the development of the disease, and the shorter the time the symptoms have lasted. Preceding syphilis does not materially modify the prognosis; hence the great importance of the diagnosis from syphilitic growth, in which suitable treatment has a most certain effect. The danger to life is least in focal myelitis in the dorsal region; in the diffuse form the ultimate danger to life is great, but I have seen several cases in which the morbid process was permanently arrested. In chronic myelitis, as in all other diseases of the spinal cord, the prognosis is rendered worse by the presence of any of the complications which so often terminate life.

TREATMENT.—The first and most important measure is the improvement of the general health, by rest, change of air, and tonics. All causes of physical and mental depression must, as far as possible, be removed. Over-exertion, and even fatigue, should be avoided, and the patient should be kept, as far as possible, from exposure to cold. Absolute rest for a short time is often useful at the outset of the treatment, especially when there has been a somewhat rapid development of symptoms. In cases of purely chronic course, absolute rest should not be maintained for more than ten days or a fortnight, or the patient may find it hard to regain his former muscular power. Counter-irritation at the situation of the disease is often useful, and most so in cases in which there is spinal pain or tenderness. Repeated sinapisms, blisters, or a mild form of the actual cautery may be employed. A hot douche to the back, at a temperature of 100° to 104° F., applied daily, has been strongly recommended by Brown-Séquard. Warm brine baths, and various thermal mineral waters, have been also said to do good.

A sea voyage is often of service, combining as it does the maximum of fresh air with the minimum necessity for exertion. Of tonics, quinine and iron are the most useful. Drugs have, unfortunately, but little direct influence on the morbid process. Those which most deserve a trial are arsenic, very small doses of mercury (such as $\frac{1}{24}$ gr. of the red iodide), and iodide of iron. Energetic mercurial treatment rarely does good, even when the patient has had syphilis, and sometimes it does harm. Iodide of potassium seems to have little influence on the disease. Nitrate of silver, ergot, and phosphorus have been recommended. Strychnia is chiefly useful in cases in which there is muscular wasting. The treatment of symptoms is that suitable in primary spastic paraplegia and progressive muscular atrophy, and described in the account of those diseases.

COMPRESSION OF THE SPINAL CORD.

Compression of the spinal cord is a common consequence of various morbid processes. Inflammation is almost always excited by the pressure, and interference with function occurs, partly from the pressure and partly from the resulting myelitis. The symptoms produced have, in different cases, many characters in common, although they vary according to the character of the compression and the acuteness of the inflammation.

CAUSES.—The morbid processes that may compress the cord are numerous. Most of them have been already described. They are as follows: Disease of the bones of the spinal column, especially caries; growths in the spine; aneurism eroding the bones and then compressing the cord; growths in the membranes; thickening of the dura mater. These processes have usually only a small vertical extent, rarely exceeding a few inches. Occasionally their vertical extent is greater, but even then the chief pressure is usually confined to a small area.

PATHOLOGICAL ANATOMY.—The cord usually presents evidence of the compression it has endured in considerable narrowing at the spot compressed, where it may be indented, flattened or cylindrical. Sometimes the reduction in size is extreme; for an inch or so the cord may be reduced to one-third of its normal diameter, and it has even been found no thicker than a crow-quill. An example of flattening is shown in Fig. 98. On the other hand, there is sometimes very little narrowing visible after the removal of the cord. At the compressed part the cord is usually gray in tint; its consistence is lessened in early cases, and increased in those of long duration. The change in color and consistence are due to the inflammation of the substance of the cord which always results from pressure, and may often be traced for some distance above and below the compressed part. When there is much compression there is always much inflammation, but considerable myelitis may occur when the amount of compression is slight. The inflammation may be chronic and slow, developing in proportion to the pressure, or it may be subacute or acute, even when the pressure is gradual. The signs of inflammation are very distinct on microscopical examination, and resemble those in other forms of myelitis. There is a general increase in the interstitial tissue; in this, at first, various cell forms may be found, but it ultimately presents the appearance of a dense reticulum. The nerve elements undergo degeneration, and abundant masses of myelin, granule corpuscles, and corpora amylacea are visible in the fresh state or in glycerine preparations of the hardened cord (Fig. 99, C). Many nerve fibres persist, however, with a narrowed sheath of myelin, and it is probable that these regain the power of conducting, in spite of the persistence of considerable and even dense sclerosis in the part. In extreme cases all the fibres seem to be destroyed at the point of chief compression, but there is never an actual division of the cord itself. The gray matter can scarcely be distinguished from the white, even with the microscope, and the ganglion cells become shrunken and atrophied. The walls of the small vessels are often thickened by a growth of spindle cells, more or less concentric to the cavity, which is encroached upon and may be obliterated (Fig. 99 A, B), a process that must add to the degree of damage to the cord. The signs of inflammation gradually lessen above and below the compressed part, but often extend for some inches in each direction. Beyond its limits the usual ascending and descending degenerations may be traced (Fig. 98). The nerve roots passing by the seat of compression suffer from the pressure, and from interstitial inflammation excited in them. They are usually gray in tint and ultimately waste, and may even be reduced to fibrous threads. The microscope shows increase of the interstitial tissue, and wasting of many nerve fibres (Fig. 99, D), and enlargement of some axis cylinders.

FIG. 98.

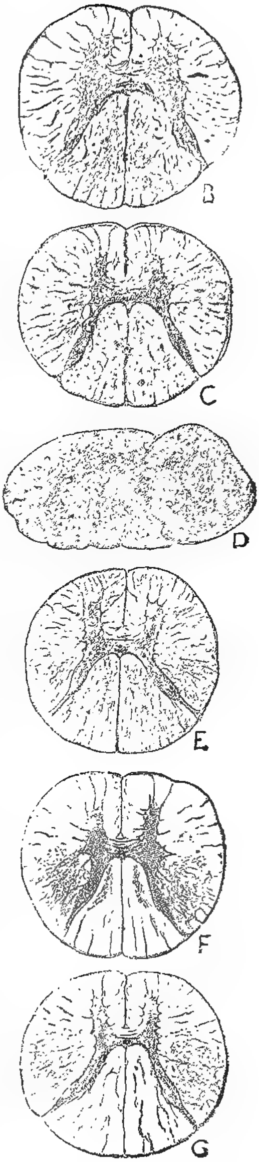
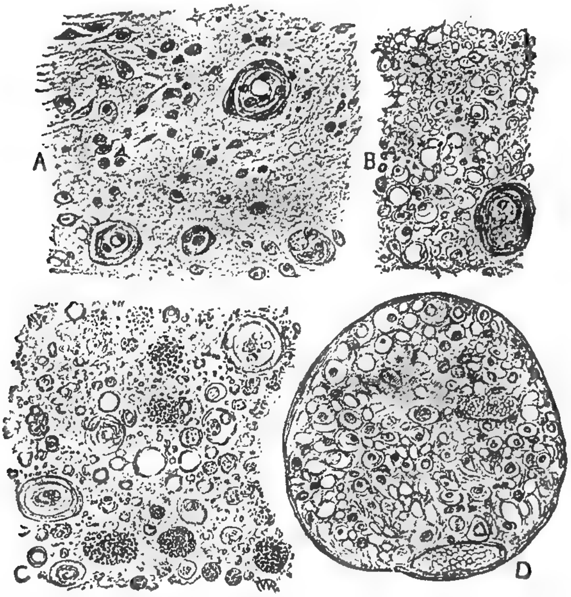


FIG. 98.—Compression of the spinal cord and pressure myelitis in a case of caries of the spine. D, Mid-dorsal region, near the chief point of greatest compression; the cord is narrowed from before backward, and is uniformly damaged by inflammation, so that the gray substance can scarcely be distinguished. C, 1½ inches higher up, shows a slighter degree of myelitis, still extending through the whole thickness of the cord. B, First dorsal; the myelitis is much slighter and the chief disease is in the posterior columns, in which ascending degeneration occupies the post.-median columns and extends outward into the post.-external columns. E, 1½ inches below the point of greatest pressure; inflammation still extending through all the elements of the cord. F, 2 inches lower down, shows only descending degeneration in the pyramidal tracts, anterior and lateral, and also the "comma-shaped" descending degeneration in the anterior part of the post.-external column. In G, at the lowest part of the dorsal region, the comma-shaped degeneration has ceased, and only that of the pyramidal tracts remains. (*From preparations by Dr. F. G. Penrose.*)

SYMPTOMS.—The effects vary much according to the degree of pressure, its rapidity, its direction, the amount and character of the inflammation produced, the amount of damage to the nerve roots, and the position of the disease. The symptoms in most cases enable the fact of slow compression to be inferred, even when there is no indication of the cause of the compression.

The symptoms are of two classes, and it is the combination of these that is the special characteristic of the process. The first is the interference with the function of the nerve roots at the level of the morbid process; the second is the interference with the functions of the cord itself. We may term them the "root symptoms" and the "cord symptoms." The cord symptoms consist chiefly in impaired conduction, manifested in the parts below the lesion. The central functions of the cord (reflex action, etc.) at the level of the lesion are abolished by the pressure, but the symptoms of this abolition are often lost in those of the damage to the nerve roots.

FIG. 99.



Pressure myelitis: portions of the section D in Fig. 98 more highly magnified. A, from the gray matter, numerous angular and fusiform cells: vessels with walls thickened by a growth of cells, narrowing the cavity, which in some is obliterated. B, from the white column; thickening of the interstitial tissue, nerve fibres in part destroyed, in part narrowed; a vessel with thickened walls. C, glycerine preparation from white substance, abundant products of degeneration of the nerve fibres, in part aggregated into granule masses. D, section of a nerve root passing by compressed area; increase of interstitial tissue, many nerve fibres narrowed, some with swollen axis cylinders.

The first class consists of symptoms of irritation and interruption of the nerve roots. The most constant is pain, extending along the course of the nerve fibres, and through the area of their distribution. The seat of these pains depends on the position of the disease; they may be felt in the arms, around the thorax or abdomen, or in the legs. They are usually sharp pains, resembling neuralgia in character, sometimes attended with tender points. Occasionally, when felt in the limbs, they are referred especially to the joints. The pain may be intermitting or constant. It is sometimes increased in a remarkable degree by movement, especially when the cause of the irritation is a growth in the bones of the spine. With the pain there is usually hyperæsthesia of the skin, often intense. After a time anæsthesia develops in irregular areas, in spite of the persistence of the pain, "*anæsthesia dolorosa.*" Irritation of the motor nerve roots may cause painful contracture of the muscles, but this is, on the whole, rare, and the chief motor symptoms are due to the interruption of the fibres—muscular weakness, gradual in onset, and accompanied by wasting. The rapidity of the atrophy varies much, and with it the electrical reaction. When slow there may be merely a progressive diminution in irritability to faradaism and voltaism; when rapid there is often the degenerate reaction, and sometimes the "mixed form," from the partial damage to the fibres that supply a muscle. Occasionally, without any actual loss of power, the patient moves the part but little, on account of the increase of the pain that movement produces,—a "pseudo-paralysis," as it has been termed.

These local root symptoms are usually the first, and to them are added, after a time, the indications of interference with the function of the cord itself. There is a weakness in the parts below the lesion, commonly developed gradually, but sometimes rapidly. Paralysis may even come on in the course of a few hours, when an acute myelitis is set up by the compression. With the weakness there is a marked and early increase in the superficial reflex action, more constantly conspicuous than in most other diseases of the cord, and therefore of some diagnostic importance. The myotatic irritability is also increased, the foot clonus being almost always present. Pains in the legs may occur, even when the disease is above the lumbar enlargement, usually dull and aching. There is often formication and tingling. There may be no impairment of sensibility in the parts below the lesion, even when there is complete motor palsy; in many cases, however, there is some sensory loss, complete only when the spinal lesion is very severe. Conduction of painful impressions may be delayed, sometimes for thirty or even forty seconds. When the pressure is lateral, one leg may be first and most affected, but the other side is usually soon involved, since neither the mechanical effect nor the inflammation remains limited to one side of the cord. The sphincters are often affected. The tendency to the formation of bedsores is rarely great, unless the lumbar enlargement is compressed or myelitis descends into it.

The course of the symptoms varies according to that of their cause. When an acute myelitis has been set up, improvement may occur for a time even if the cause of pressure is slowly progressive. This is true also of root symptoms when they are rapidly developed. If the compression ceases to increase, the cord may recover its conducting function, although it remains narrowed. Sensation is often regained in the legs when motion remains absent, but both motion and sensation may be regained although the narrowing of the cord persists. It has even been found no larger than a goose quill at the compressed spot, although the paraplegia had passed away. In such a case many narrowed nerve fibres are found in the section of the compressed portion, and it is probable that many axis cylinders persist with an envelope of myelin so narrow as to be recognized with difficulty, but which suffices for their conducting function. Under slow pressure the axis cylinders may not suffer interruption, although the myelin wastes. Regeneration of axis cylinders, as in the peripheral nerves, is also possible (see p. 227). In some cases, especially when a considerable myelitis has been set up, the motor path may recover and the posterior columns remain affected, so that power returns, but without coördination, "secondary ataxy" resulting.

The DIAGNOSIS of slow compression rests on the concurrence of the root and cord symptoms already described. Of the former, the sensory symptoms are the most characteristic. In many cases other indications of the compressing disease assist the diagnosis. These vary in character; the most frequent is considerable local tenderness of the spine. If root symptoms are absent the diagnosis is much more difficult, unless the morbid process manifests itself externally. If there is a suspicion of compression in a case of slow paraplegia, an early and considerable excess of superficial reflex action gives additional weight to the suspicion. Even without any other symptoms to indicate compression, this is suggested by symptoms which begin on one side and gradually spread to the other.

The chief disease from which compression of the spinal cord has to be distinguished is a subacute or subchronic transverse myelitis. Besides the indications of a cause of compression already mentioned, the chief distinction depends on the presence of symptoms indicating irritation of the nerve roots, and especially on the fact that these symptoms precede those of the lesion of the spinal cord itself. True chronic myelitis is occasionally attended by more irritation of the nerve roots than the subacute form, but this succeeds, instead of preceding, the damage to the cord, and the course of chronic myelitis is often much slower than is that of most forms of slow compression. The distinction from a growth within the spinal cord rests chiefly on the early occurrence of impairment of the central and conducting functions of the cord, and the partial distribution, and often slower course of the symptoms. The effects of an intra-medullary growth vary widely in different cases, and if there is an early irritation of root fibres, the distinction may have to depend on the presence or absence of the signs of a compressing process.

The diagnosis of the cause of compression is often much more difficult than that of its occurrence. The facts that the patient is in the first half of life, and that he inherits a tubercular tendency, suggest caries. The absence of root symptoms is also in favor of caries or a tumor within the spinal cord. The presence of root symptoms does not, however, render caries less likely, unless the pain is extremely severe, and is greatly increased by movement, characters that should always suggest a growth in the bones of the spine.

Root symptoms extending over a considerable vertical area suggest pachymeningitis. Additional details of the differential diagnosis will be found in the account of the several compressing diseases.

Prognosis and Treatment.—The prognosis depends on the cause, and the amenability of this to treatment. As a general rule, however, the more rapidly the cord symptoms develop, the more chance there is of improvement, because the greater share does inflammation take in their production. The treatment is that of the cause of the compression and that of myelitis.

ANTERIOR POLIO-MYELITIS. ATROPHIC SPINAL PARALYSIS.

In certain important groups of diseases of the spinal cord, wasting of the muscles is a prominent symptom. In all the lesion involves, exclusively or in part, the anterior gray cornua, and the muscular atrophy depends on the destruction of the nerve cells, from which the axis cylinders of the motor nerve fibres proceed. The fibres degenerate, and it is apparently by the agency of this degeneration that the atrophy of the muscles is produced. The occurrence of the wasting does not depend on the nature of the lesion in the gray matter; it occurs equally whether the nerve cells are destroyed by hemorrhage, by inflammation, acute or chronic, by slow degeneration, or by the growth of a tumor. But the rapidity with which the lesion develops influences materially the character of the symptoms. Time is required for the process of nerve degeneration, and for the effect of this on the nutrition of the muscles; one or two weeks elapse, even in the most acute cases, before muscular wasting is distinct. The nerve cells also form part of the voluntary motor tract. Their disease at once interrupts this path. Hence, in acute cases, paralysis is rapidly developed, and the atrophy succeeds it after an interval. In chronic lesions, on the other hand, the changes in the cells and fibres progress so slowly that the weakness and wasting appear to come on together. The wasting may even seem to precede the weakness, but a careful examination will show that loss of power exists as soon as there is distinct atrophy. It is customary to distinguish the two groups by terming those in which the palsy precedes wasting, "atrophic spinal paralysis," and those in which wasting is apparently simultaneous with weakness, "spinal muscular atrophy."

The atrophic paralyses are due to an acute or subacute process in the anterior cornua. The lesion is probably, in most cases, inflammatory in character, a cornual myelitis, or polio-myelitis (Kussmaul). In some cases, which differ only in the suddenness of the onset, it is probable that the lesion is hemorrhage and not inflammation. The chronic atrophies are due to a slow degeneration of the nerve cells in the gray substance. By some writers it is regarded as a parenchymatous inflammation, but, in accordance with the method here adopted, the chronic disease will be classed among the spinal degenerations.

ACUTE ATROPHIC PARALYSIS; ACUTE ANTERIOR POLIO-MYELITIS; CORNUAL MYELITIS (INFANTILE PARALYSIS; ESSENTIAL PARALYSIS OF CHILDREN).

Acute atrophic paralysis is a disease in which voluntary power is lost in the course of a few hours or days, and some of the paralyzed muscles undergo rapid wasting, while others recover. The onset of the palsy is often preceded or accompanied by indications of general disturbance. The muscles which waste usually remain weak, and contraction of their opponents leads to permanent distortion of the paralyzed limbs. The disease is the most common form of paralysis in young children, and has hence received the name of "infantile paralysis." Before anything was known of the lesion on which it depends, it received (from Rilliet and Barthez) the unmeaning designation of the "essential paralysis of children." The spinal lesion was first demonstrated by Prevost (1865) and soon afterward by Lockhart Clarke, and by Charcot and Joffroy. Its constancy has been abundantly proved by numerous observations during the last ten years. The occurrence of similar symptoms in adults was noted by Vogt (1858) and Duchenne (1864).

Etiology.—The disease occurs at all ages, and although its relative frequency at different periods of life cannot yet be definitely stated, it is certainly six times more frequent in the first ten years than in all the rest of life. It is moreover especially a disease of later infancy. Of the cases under ten, three-fifths occur in the first two years of life, and four-fifths in the first three years. It is less frequent in the first than in the second year, but it occasionally comes on soon after birth, at the twelfth day in a case observed by Duchenne. The friends often date the disease from birth, but there is no valid evidence of its intra-uterine development. Its onset may readily be overlooked in a young child. In adults it may occur at any period, up to sixty years of age, but is most frequent between ten and thirty. Boys are somewhat more prone to suffer than girls, in the proportion of four to three, but (to judge by a series of fifty consecutive cases which I have tabulated from my own note books) it is only under two years that a greater liability in boys can be traced. In adults, however, the disease is far more frequent in males than in females. The influence of heredity is small, but a family tendency to suffer may now and then be traced; two and even three brothers or sisters have been affected (Seeligmuller, etc.). One of my own patients had two cousins affected with the same disease. The inherited tendency may be indirect; of a patient in whom the disease came on in adult life, two brothers had suffered from hemiplegia, and an uncle from paraplegia.

The disease occurs far more frequently in summer than in winter. Sinkler, who first called attention to the fact, found that in Philadelphia four-fifths of the cases commenced in the five hot months, May—September. Of my own cases, two-thirds were attacked during the hottest third of the year, June—September. This relation to season is seen in adults as well as in children. The fact is the more important because cold has long been regarded as the most potent exciting cause. That exposure to cold is an occasional cause can scarcely be doubted, but it is more traceable in adults than in children, and the extent of its influence has certainly been over-estimated. The relation to season shows that it is not the mere degree of cold which is influential, but probably the chilling of the heated body. Thus one patient, aged sixteen, when perspiring from a long ride after the hounds on a hot day in September, lay down on a sofa beneath an open window from which a draught blew on his back. Two days afterward the first symptoms commenced. I have twice known the disease to be due to sitting on damp grass. Two or three days is the interval which usually elapses between the exposure to cold and the onset of the symptoms.

Over-exertion has been supposed to be an occasional cause, and Lange associates the frequency of the disease in late infancy with the functional excitation of the cord in walking. Over-walking is perhaps an occasional cause in young children. A traumatic cause, such as a fall, can occasionally be traced, although the statements of friends on this point may readily mislead. "We suppose that the child must have had a fall" is a common statement, when there are no grounds for the supposition. But now and then the disease does follow a fall, at an interval of a few days, in a manner which at least suggests that the injury was its cause. Thus in one severe case, a boy eight years old was thrown over the head of a donkey, and the onset occurred five days afterward. In another instance, a child fell over the back of a chair on which she was standing; she was stunned for a few seconds and then seemed all right. Two days afterward febrile disturbance came on, and paralysis was discovered on the fourth day from the fall. It would seem probable that the injury, in such cases, merely serves to excite the disease in a predisposed subject.

Since the disease develops most frequently during the period of the first dentition, teething has, by many, been supposed to be its chief cause. But there is no proof of any relation between the two. Their coincidence in time would be significant if we knew of any mechanism by which the process of dentition could produce myelitis, or if no other conditions existed, during the period of dentition, to which the frequency of the disease might be due. But, on the one hand, we know of no such mechanism (for the theory of a reflex vasomotor disturbance is a pure hypothesis), and, on the other hand, the period of dentition coincides with the rapid functional development of the nervous system, which succeeds its structural development in the first few months of life. Moreover, this period is often one of deterioration of health, in consequence of changes of diet and other causes. The disease occurs before and after the period of teething, and it exhibits no increase in frequency at the period of the second dentition. Of fifty cases occurring under ten, in only two did the disease occur between six and ten.

Many children are perfectly well at the time when the disease occurs. A few are in conspicuously defective health. Thus in one case the child had been rendered feeble by long-continued diarrhœa. I have only once seen the disease in the subject of inherited syphilis. On the other hand it is occasionally secondary to acute febrile diseases—scarlet fever, measles, bronchitis, pneumonia, and, in older children and adults, to typhoid fever and ague. It may develop during the height of the acute disease or during convalescence. But the frequency of this cause has been unquestionably overrated. The initial general disturbance is constantly mistaken for an independent general disease, and the opinion is often maintained after the discovery of the paralysis, which is then supposed to be of secondary origin. Thus in one case initial pyrexia, headache and vomiting were supposed to indicate scarlet fever. Paralysis of all four limbs came on, with difficulty in swallowing, and the latter was supposed to render certain the diagnosis of scarlet fever, although there was no sore throat or rash. In older children and adults the disease is often thought to be secondary to rheumatic fever, in consequence of the rheumatoid character of the pains. The error is the more likely to occur if the symptoms follow exposure to cold. There is no evidence that the disease is ever secondary to articular rheumatism. Chronic alcoholism has been supposed to be a cause in adults, but it is probable that most cases supposed to be due to this cause have been instances of multiple neuritis.

SYMPTOMS.—The general characters of the symptoms have been already mentioned,—an acute onset, often with general febrile disturbance; paralysis, at first widespread, afterward passing away, except from a region in which the muscles rapidly atrophy, and in which, although partial recovery may occur, more or less weakness and wasting usually persist.

The general disturbance at the onset may be severe, slight, or absent. When present there is commonly pyrexia, with its usual accompaniments, headache, prostration, loss of appetite, restlessness. The elevation of temperature is rarely considerable, 100° or 101°, but it sometimes reaches 103°, or even, as I have seen, 104°. These symptoms occur in both children and adults. In the former there is sometimes more pronounced cerebral disturbance, convulsions, and delirium. In older children and adults transient diplopia occasionally occurs, chiefly when the disease is in the cervical region of the cord. Giddiness is sometimes complained of. The convulsions and delirium are usually ascribed to the fever, and compared to those which may occur at the onset of an acute specific disease, but it is possible that the cerebral functions are sometimes more directly deranged. These symptoms of general disturbance usually last for a few days only, sometimes for only a few hours, and they are not unfrequently absent. Before they have entirely passed away, the paralysis comes on, sometimes a few hours, sometimes a few days, after the onset of the symptoms. Thus a common history is for a child to seem ill and feverish, to be put to bed, and the next morning to be found to have lost power. As an example of more deliberate onset may be mentioned the case of a medical student who found, one day, that he saw double; on the next he felt giddy, and became feverish (100°–102°); he slept for forty-eight hours; and on the fourth day found his right arm was weak, and atrophy of the deltoid, etc., followed. When the initial stage is prolonged, the paralysis is often not discovered for some time. The immobility of paralysis is mistaken for the inertia of prostration. When general symptoms are absent, the paralysis often comes on in the night.

In older children and adults, another common initial symptom is the rheumatoid pain already mentioned, sometimes referred to the muscles, sometimes to the limbs generally, occasionally to the course of the nerves, but never to the joints. Pain may be felt also in the back. In character it is dull and aching, or acute and tearing or burning. It is often very severe. Instead of being initial, the pain may succeed the onset of paralysis, and occasionally continues for several weeks. There may also be tingling or formication in the limbs.

The paralysis is always rapidly developed. It varies much in its initial range. Only part of a limb may be affected, or there may be universal loss of power. In the majority of cases the paralysis is intermediate between these extremes. Two or three limbs are affected—both arms, both legs, or the legs and one arm. When all four limbs are paralyzed the neck muscles also may be weak, and even swallowing may be impaired. The other muscles supplied by the cranial nerves escape, and, as a rule, the sphincters are unaffected. To the latter, exceptions are occasionally met with, always in severe cases, and the paralysis of the sphincters, when it occurs, often lasts a long time. For instance, a child aged two and a half years woke up one morning with headache, fever, and weakness of the legs, which rapidly increased to complete paralysis. Four days later the arms also became weak, and in a day or two more the urine escaped involuntarily. The arms began to recover in six weeks, and were well in six months; both legs wasted and remained permanently paralyzed; the incontinence of urine lasted for a year. A similar affection of the sphincters is occasionally met with in adults.

The paralysis usually commences in one limb and quickly spreads, reaching its maximum extent in from one to four days, and sometimes in a few hours; occasionally it occupies a week in its development. When it does not reach its height until more than a week has elapsed, the case is regarded as "subacute." As another example of a common mode of onset may be mentioned the case of a boy, aged a year and four months, who one day seemed ill, was sick, and was put to bed. The next day he could scarcely stand alone; on the following day he could neither move the legs, nor sit up in bed. In about ten days the left leg began to improve, but there was enduring paralysis and wasting of the whole of the right, and of the lower part of the left leg. Again, another child seemed ill, and was kept in bed for five days; on the third day it was noticed that the left arm was not moved so much as the right, and by the fifth day the arm was quite powerless; at the end of a fortnight improvement commenced. In the adult, the mode of onset presents nearly the same characters. A lady, twenty-five years of age, sat down for some time on wet grass. Two days later general rheumatic pains came on, very severe in the legs on the following day, and the legs were distinctly weak. In the course of the next forty-eight hours the arms also became feeble, and at the end of a week she could scarcely move her arms, and her legs were absolutely paralyzed. In a fortnight improvement commenced, first in the arms and then in the feet; but the hip and thigh muscles remained paralyzed and rapidly wasted.

Cases are sometimes seen similar in other respects to those now under consideration, but in which the paralysis comes on suddenly, and without general disturbance. Weakness commences, for instance, in one arm, and spreads so quickly that in the course of a quarter of an hour there may be universal paralysis, which afterward passes away, except in one region, where there is rapid wasting. The suddenness of the onset makes it difficult to regard the lesion as inflammation; it suggests hemorrhage. Moreover, in most cases of actually sudden onset, febrile disturbance is absent. We shall see that hemorrhage may occur in early childhood. Hence, while we must class these with the other acute spinal atrophies, we are not justified in regarding them as cases of polio-myelitis.

The following cases are instances of such sudden onset. A girl, aged seventeen, suddenly felt tingling in the left hand. In a few minutes the whole arm was powerless, and then the right arm became paralyzed. She had a strange sensation at the back of the neck, and went up stairs. As she was going up, her legs became weak, first the left and then the right. In less than half an hour from the time of the first symptom there was absolute universal paralysis, with difficulty in breathing and in swallowing. Improvment (in the opposite order to the onset) commenced the same evening and was complete in about six weeks, with the exception of the muscles of the left forearm and hand, which rapidly wasted and were permanently paralyzed and atrophied. Another girl, while walking across a road, suddenly felt a "sort of shock," as if some one had given her a knock between the shoulders. She became giddy, and instantly felt tingling in both arms, especially in the right, which became weak before she got to the other side of the road. Ultimately the arms recovered, but permanent paralysis and wasting of the intrinsic muscles of each hand were left. A similar sudden onset is also seen occasionally in children. Thus a child, aged two, was walking along, when he suddenly said he could not stand, and fell down on his knees. Both legs were powerless. Slow improvement followed in the left leg, and wasting in the right. A sudden onset is perhaps more common than histories show, because sudden onset while a child is resting eludes recognition.

When the paralysis has reached its height, it remains stationary for a time, which varies from two to six weeks, and then lessens. The first improvement occurs in the parts last affected, and it gradually spreads until, usually at the end of from one to three months, all parts have recovered except those which are permanently affected. In these the muscles are toneless and flaccid from the first, and in two or three weeks there is distinct wasting, which rapidly increases until the shape of the limb is changed, and, in extreme cases, scarcely any of the volume of the muscle can ultimately be detected. In fat children, the appearance of the limb may be less altered, and it seems that, in some of these, an interstitial growth of fat makes up for the diminution of muscular tissue. In older children and adults the muscles are often tender to the touch during the process of wasting.

When the atrophy is distinct the muscles no longer contract to faradaism, and, if the motor nerves are tested, they also will be found to have lost irritability. The loss depends on degeneration of the nerves. The muscles usually present the degenerative reaction in characteristic form (see p. 66). The loss of faradaic irritability is distinct, in severe cases, as early as the end of the first week, and even by the fifth or sixth day. In a patient, for instance, who is universally, and apparently uniformly, paralyzed, one or more groups of muscles may be found to have lost irritability, and in these we know that there will be lasting paralysis and wasting, while the other parts will recover. In a severe case, in which the muscles most affected atrophy completely, the loss of faradaic irritability may be permanent. The voltaic irritability remains, increased in degree, for two, three, four, or six months, then slowly falls, as the muscular fibres themselves degenerate, and ultimately, at the end of one or two years, it disappears. More commonly, after six or twelve months, some faradaic irritability returns. It may be slight, due to the recovery of a few fibres, insufficient in number to restore any bulk or power, but the few fibres which recover do so perfectly, so that the irritability becomes normal in degree, although the contraction which can be thus produced is very slight in amount. In other cases considerable recovery occurs, so that some power and volume are regained, although the muscles remain below the normal size. Rarely recovery is perfect. In the muscles which do not waste there is no degenerative reaction, but there may be slight diminution of excitability to each current.

The paralysis is motor only. There may be at first tingling or formication in the limbs, but there is no loss of sensation. Reflex action from the skin, however, is abolished. At first it is lost wherever there is weakness, but it returns with or soon after the recovery of power in the less affected parts. Where there is persistent paralysis it remains absent. The myotatic irritability is lost in the same or even greater degree. For instance, no knee jerk can be obtained if the extensors of the knee are affected even in slight degree. The loss depends on the interruption of the muscle-reflex centre by the disease in the gray matter. In rare cases of severe cervical polio-myelitis, which is not limited to the gray matter but affects in less degree the lateral white columns, there is wasting of the arms, but paralysis without wasting in the legs, and in the latter the myotatic irritability may be increased above the normal, so that the foot clonus may be obtained. Such cases are, however, very rare, and are intermediate between polio-myelitis and transverse myelitis.

A curious effect of the disease is to retard the growth of the bones in the affected limb, so that this gradually becomes shorter than its fellow, and after a time the compact tissue of the bone is less than on the other side. It is a retardation of growth, and there is scarcely sufficient ground for calling it an atrophy comparable to that of the muscles. There is no evidence that any wasting of bone takes place in adults. Strange to say, Seeligmuller has observed the opposite effect in children: an actual elongation of the bones, apparently due to the fact that the growing epiphyses suffer traction instead of the normal compression. The effect on the growth of bone is not proportioned to the muscular paralysis, and cannot therefore be regarded as the effect of the latter.

Those joints that depend for their support on tendons which pass over them become lax when the muscles are paralyzed, and the articular surfaces may be no longer kept together. Thus when the deltoid is wasted, the head of the humerus may fall out of the glenoid cavity.

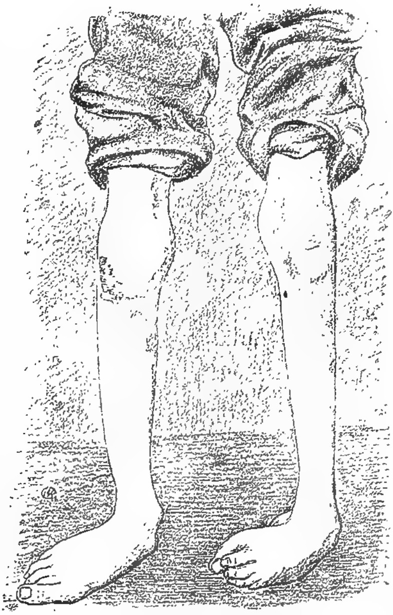
Bedsores are almost unknown, even in the acute stage of the disease. A slight local elevation of temperature in the most affected parts has been observed in the early stage, but subsequently the limb is constantly colder than the other. This is due, in part at least, to the want of the aid to the circulation which is, in health, supplied by the muscular action.

During the chronic stage of the disease there is great liability to the occurrence of permanent shortening of muscles, with consequent displacement of the parts to which they are attached. Grave deformities are thus produced. Unquestionably the chief mechanism by which these are produced is the shortening of muscles which are less paralyzed than their opponents. The less affected muscles retain their contractility; they are no longer subject to the normal extension by their opponents, and hence gradually become shorter and less extensible. This may occur in muscles which are unaffected, and it is uncertain how far it is favored by a slight degree of paralysis. The latter is attended with some increase of the interstitial tissue of the muscle, and the growth of this may readily fix the contracture of the proper muscular fibres. Posture often aids the production of these muscular contractions, and so also in the case of the foot does the relative shortening of the limb.

The persistent paralysis may occupy the whole or part of one or more limbs. In half of the cases one leg only is permanently affected. It is less frequent for both legs or for one arm to suffer, and still rarer combinations are those of both legs and one arm, of both arms, of the arm on one side and the leg on the other, while the rarest of all is the affection of leg and arm on the same side. The limbs on the left side of the body are more often permanently affected than those on the right.

When the paralysis of a limb is incomplete, the part involved varies much in different cases, and as different parts of two or more limbs may be affected, the combinations of palsy which result are extremely varied. The grouping of adjacent muscles is sometimes distinctly that of functional association, more often it is random. In the legs the paralysis, in very severe and fortunately very rare cases, may be absolute, involving all the muscles of both limbs. Usually it is partial, and then the muscles below the knee suffer more often than those above the knee. The calf muscles are much less frequently affected than the muscles in front of the tibia or the peronei. Hence talipes equinus is a common deformity, and its occurrence is aided by the shortening of the limb, in consequence of which the foot has to be extended to bring the ball to the ground. Either the tibialis anticus or the peronei may be most affected, with the result, in the former case, of talipes valgus, in the latter of talipes varus. In the case shown in Fig. 100 the peronei have suffered most in the left leg and least in the right, producing an unsymmetrical but corresponding distortion of the feet. This affords an illustration of another fact, namely, that when there is a partial affection of both legs the paralysis scarcely ever corresponds in the two in its incidence or its degree. Much less commonly the calf muscles suffer more than the others, and there results talipes calcaneus, as in the case shown in Fig. 101. The intrinsic muscles of the foot often suffer with those of the leg. In the thigh the extensors of the knee are affected more frequently than the flexors, and hence flexor contraction is very common and may even cause subluxation of the joint. The flexors of the hip often suffer, with the extensors of the knee or alone. Less commonly the glutei are involved, but never alone.

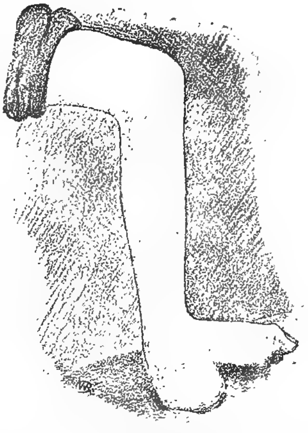
FIG. 100.



Old atrophic spinal paralysis. Talipes valgus in the right leg from paralysis of the anterior tibial; t. varus on the left from paralysis of the peronei.

In the arm almost all the muscles are sometimes affected, as in the case shown in Fig. 102, but all are never entirely paralyzed. The intrinsic muscles of the hand often suffer: either the thenar muscles or the interossei may be most damaged. An instance of such paralysis of the interossei is shown in Fig. 103. The forearm muscles are frequently affected, but the supinators may escape when the other muscles are involved. The deltoid suffers more frequently than any other single muscle of the arm. It may be paralyzed alone or in association with other muscles; occasionally the deltoid, supra- and infra-spinatus, biceps, and supinators are all involved in the "upper arm type" of palsy of Erb* (see p. 96). But other muscles than these may

FIG 101.



Atrophic spinal paralysis affecting chiefly the calf muscles, with resulting contracture of the flexors of the foot causing talipes calcaneus.

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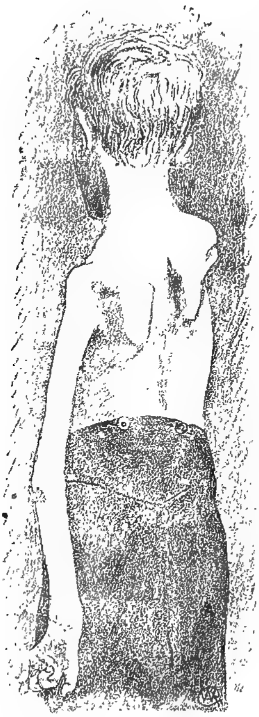
* See a paper by Dr. Beevor, *Med.-Chir. Trans., 1885.*

be associated with the deltoid. In the case shown in Fig. 103 the deltoid and intrinsic muscles of the hand were wasted, and no other muscles. The serratus magnus is occasionally affected, as in Fig. 102; in this case, although the left arm had suffered severely as the figure shows, on the right side only the serratus was involved. With the serratus the upper part of the pectoralis major is sometime associated in the paralysis (as it is in normal function, see p. 49); the lower part being unaffected. It was so in this boy. The middle part of the trapezius and other scapular muscles are occasionally involved. The neck muscles rarely suffer, but the diaphragm is occasionally paralyzed. Although the intercostals and other trunk muscles are so often weakened in the early stage, considerable permanent atrophy is very rare. Curvature of the spine is, however, often produced in consequence of the patient being allowed to sit up while the muscles are weak. It may be a lateral or an antero-posterior curve, according as the weakness is on one or both sides. I have once seen considerable depression of the lower part of the wall of the thorax on the left side from paralysis of the intercostal muscles at the spot, perhaps combined with hindered growth of the ribs. The muscles supplied by cranial nerves are scarcely ever affected. I have once seen a complete paralysis of one side of the face. It was associated with wasting in the limbs.

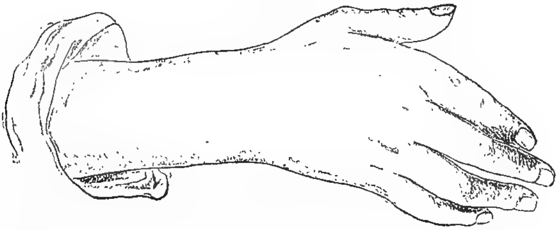
Fig. 102.



Atrophic spinal paralysis, infantile form. Wasting of all the muscles of the left arm from the deltoid downward, and of the right serratus magnus.

The *course* of the disease has been already sketched. There are (1) an initial stage of paralysis rapidly increasing, often accompanied with fever: the duration of this stage is from a few hours to a week; (2) a stationary period, which lasts from a week to a month; (3) a stage of "regression," during which the palsy passes away, except from certain parts in which

Fig. 103.



Atrophic spinal paralysis (adult form) affecting the interossei and thenar muscles, and also the deltoid.

wasting occurs: this regressive period usually occupies from one to six months; (4) a chronic stage, during which the atrophy continues, slight improvement may occur, but contractures and deformities are developed. The duration of this stage is indefinite. Improvement often occurs after three, six, or twelve months, but is then usually slight, and often more than counterbalanced by the interference with growth, and by the occurrence of deformities. Even in the slightest cases complete recovery is extremely rare. On the other hand, death from the disease is equally uncommon, and occurs almost exclusively in the early stage. It is not improbable that some children die from the initial disturbance before its nature is recognized, especially in cases attended by severe convulsions. Now and then death occurs at the end of the first week or ten days, from universal paralysis or from some cerebral complication. I have seen one example of the latter; paralysis in the arms came on upon the third day of the initial fever, reached its height on the fifth day, and then remained stationary. Faradaic irritability on the seventh day was lost in many muscles of the left arm. There was a little rigidity of the neck. On the tenth day the patient began to vomit, and complain of increased headache, and had hallucinations. On the eleventh day he became comatose and died. It is probable that other cases of the same kind occur, in which the exact nature of the spinal affection is not recognized.

Sequelæ, beyond those mentioned as part of the symptoms of the disease, are very rare. As a rule, the general and nervous health of the patients and the duration of life, are uninfluenced by the local palsy. In a few cases, however, some other chronic affection of the spinal cord has come on when the subjects of infantile paralysis have reached adult life. Progressive muscular atrophy has been several times observed to start from the paralyzed limb; acute and subacute polio-myelitis in adult life has also been observed, and I have twice seen the symptoms of lateral sclerosis, without atrophy, slowly developed, in one case at seventeen, in the other at twenty-eight. In the spinal cord of the subject of old infantile paralysis there seems thus to persist a disposition, slight though it be, to fresh disease, and the cases of lateral sclerosis mentioned show that the liability to disease is not limited, as has been thought, to the gray matter.*

PATHOLOGY.—For a long time infantile paralysis was believed to be a peripheral affection having its seat in the muscles. The opinion rested on the absence of any knowledge of the spinal changes, and on the fact of the muscular wasting. But improved methods of examining the nerve centres have shown that changes in the spinal cord invariably exist, and, in early cases, have the characters of an acute inflammatory lesion; and we now know that muscular atrophy usually depends on central disease. Every gradation exists between the severe and the slight form of the affection, and all known facts point to the conclusion that acute atrophic infantile paralysis is invariably a disease of the spinal cord. The perfect correspondence between the infantile and adult forms make it certain that the latter have the same origin, although pathological facts are more scanty than in the cases of the former.

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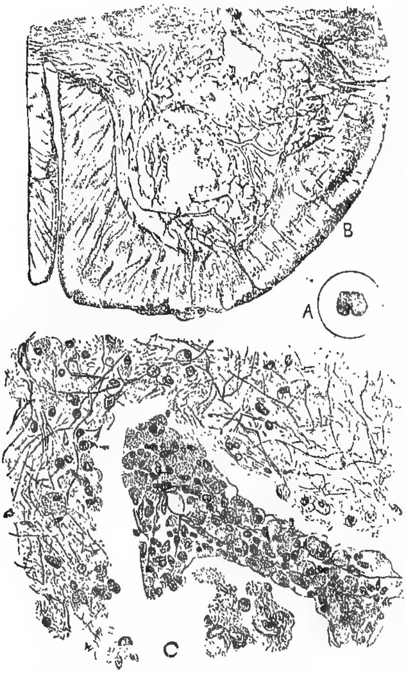
* Compare Ballet and Dutil, *Revue de Médecine*, January, 1884, p. 18.

FIG. 104.



Acute anterior polio-myelitis in a child 2½ years old, six weeks after the onset. (*After Charlewood Turner.*) A. Section through the lowest part of the lumbar enlargement, showing a cavity visible to the naked eye, on the left side. B. Left anterior half of the cord under a low magnifying power, showing destruction of almost the whole anterior cornu. C. Portion of anterior cornu in the upper part of the lumbar enlargement. Numerous corpuscles lie in the granular protoplasm and reticulum of fine fibres. A small artery is encrusted with several layers of corpuscles and nuclei.

Observations on the changes in the spinal cord in the early stages are few. A case, recorded by Dr. D. Drummond, in all probability one of this disease, is by far the earliest on record, and is indeed the earliest possible.* A child, five years old, died after a few hours' acute illness. The spinal cord in the region of the fourth and fifth cervical nerves, presented undue redness of the anterior gray matter. The vessels running from the surface to the cornu were distended with blood. The microscope showed distention of capillaries and minute extravasations in the gray substance, swelling of the neuroglial elements and of the ganglion cells, which were granular with indistinct processes. Another important early observation is that of Dr. Charlewood Turner,† six weeks after the onset (Fig. 104). In this, and other somewhat later cases, acute changes have been found in the anterior cornua, more advanced than in the case of Dr. Drummond. They are often widely spread in slight degree, and attain considerable intensity in one or more foci, usually in the cervical or lumbar enlargements or both. At these spots the anterior horn is softened; sometimes there is hemorrhagic infiltration, sometimes an actual cavity (Fig. 104, A, B). The microscope shows extravasated blood, often massed along the vessels (Fig. 104, C), and scattered through the gray matter with other cells such as are met with in myelitis. There are also granule corpuscles and other products of degeneration of the nerve elements. These elements, and especially the motor nerve cells, have almost entirely disappeared. Where the change is slighter in degree there is a leucocytal infiltration, a few granule corpuscles are seen, while the nerve cells may be structurally intact, but swollen, and granular. The morbid appearances may be confined to the anterior cornua, or may extend in slighter degree into the adjacent antero-lateral column. The posterior columns are always unaffected. In very severe cases there may be signs of slight local meningitis corresponding in position to the region chiefly affected. This region corresponds to the origin of the nerves supplying the muscles in which there is most wasting, and the anterior nerve roots, which arise from this part, present the signs of acute degeneration. Although early observations on the morbid changes are few, a large number of cases have been examined at a later period, years after the onset. The appearances presented by these cases accord with those in the early stage, allowance being made for the difference in time. The anterior cornu at one or more places is shrunken (Fig. 105), and in part stains deeply with carmine, in consequence of its nervous structures being replaced by connective tissue, in part less deeply in consequence of "granular disintegration" of the substance. The motor nerve cells are absent, partially or entirely. Sometimes a few shrivelled cells remain, sometimes one or more groups may be unaffected; or one or two nerve cells of normal appearance may alone remain (see Fig. 105). The antero-lateral column is also smaller than normal and may present slight sclerotic changes, sometimes chiefly contiguous to the gray substance, sometimes extending into the pyramidal tract. In consequence of these alterations, the affected half of the cord is conspicuously smaller than the other, even to the naked eye. The consequent alteration in the shape of the cord is greatest where the damage to the gray matter is most intense. The anterior nerve roots at the most affected parts are small and gray, and the degeneration of the motor fibres may be traced down the nerve trunks. Often, however, a few fibres present a normal appearance, although the rest have perished.

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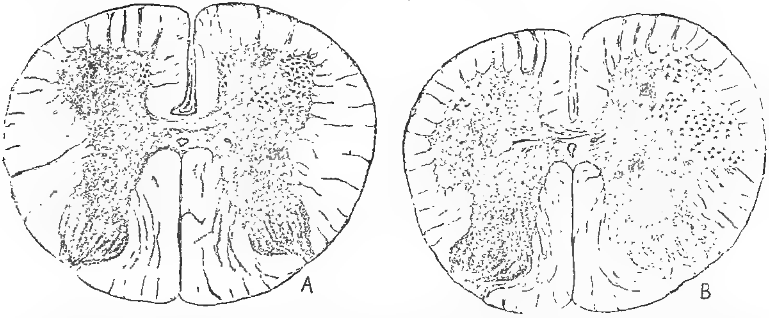
* *Brain*, April, 1885.

† *Path. Trans.*, vol. xxv, p. 203.

FIG. 105.



Atrophic spinal (infantile) paralysis. A. First lumbar; the left anterior cornu is smaller than the right, and its ganglion cells have disappeared with the exception of the inner group. B. Fourth lumbar: the whole left half of the cord is smaller than the other: in consequence of the diminution in size of the ant. horn. Of the ganglion cells only a few remain, belonging to the antero-lateral group. Similar changes existed throughout the lumbar enlargement.*

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* For these sections I am indebted to Dr. H. Humphreys, of St. Leonards. The paralysis came on at one and a half years of age, two years before death, and involved most of the muscles of the left leg, those of the calf in greatest degree, so that talipes calcaneus had developed.

The muscular fibres, in early cases, have been found narrower than normal, and in a state of granular degeneration, with an increase in the nuclei of the sheath and of the interstitial tissue. Granules and pigment masses accumulate between the sarcolemma sheaths. In extreme cases the degeneration proceeds to complete disappearance of the fibres, the place of which is occupied by fibrous tracts, developed partly from the sarcolemma sheaths and partly from the interstitial connective tissue. It is common to find, here and there, a fibre presenting normal appearances. Sometimes fat accumulates in the interstitial tissue, so that the reduction in the bulk of the muscle may not be so great as the actual wasting. In slighter cases, in which partial recovery takes place, some of the fibres slowly regain their normal appearance, even though they have undergone slight granular degeneration. Usually, however, many fibres perish; those which recover are smaller than normal, and the interstitial tissue remains excessive. Thus the original volume of the muscle is rarely regained.

The brain has been almost always found normal. In one case with extensive paralysis, of long duration, some atrophy was found in the motor region of the cortex (Sanders).

The symptoms of acute atrophic paralysis indicate disease of the motor nerve cells with consequent degeneration of the motor nerve fibres which spring from them. The pathological facts fully corroborate the conclusion, and the slighter widespread changes in the anterior cornua agree perfectly with the extensive initial palsy, which passes away. Both the character of the lesion and the mode of onset, suggest that it is inflammatory in character in the majority of cases. In rare instances, as already stated, the almost instantaneous onset suggests that the lesion is different in character, is vascular in its nature, probably hemorrhage, although the character of the symptoms makes it probable that the seat of the lesion is the same as in those of the common type. These cases have, moreover, the character of a primary hemorrhage, not of hemorrhagic myelitis (see p. 281). Signs of inflammation do not precede the sudden onset. Thus these cases, while they belong to the clinical group of acute atrophic paralyses, cannot at present be placed in the pathological category of polio-myelitis. They differ from the inflammatory cases in another respect, that the lesion is confined to one part of the cord, whereas in the cases that are certainly myelitic, it is frequent for different parts of the cord to be damaged.

The wide distribution of the lesion in the latter cases, and its varying degree in different parts of the gray matter, seem to preclude the theory that the mischief is set up by an initial vascular lesion, such as thrombosis. It is to be remarked, however, that when the disease is in the cervical region, paralysis of the legs does not show that the lumbar enlargement is also diseased unless there is loss of irritability in the leg muscles or abolition of reflex action in the legs. They may be completely paralyzed for a time by the damage, in the cervical region, to the adjacent lateral columns, which occurs in rare cases in which the inflammation of the gray matter is very intense.

DIAGNOSIS.—This rarely presents any difficulty except in the early stage. When the initial paralysis is passing away, and the wasting is distinct, the nature of the case is sufficiently evident, and is corroborated by the electrical reactions, by the loss of reflex action, and by the absence of any impairment of sensibility. At the onset, however, the symptoms may readily be misinterpreted. Initial febrile disturbance is naturally ascribed to some general cause until nervous symptoms manifest themselves. Even then there is risk of error. On the one hand, as we have seen, a diagnosis of a general disease is not always relinquished when paralysis appears, but the latter is regarded as secondary. On the other hand, especially in young children, the existence of paralysis is often overlooked at first, and it is supposed that the child does not move because it is prostrate. This error will not be made if it is remembered that mere prostration never causes total immobility, and *à fortiori* it does not produce local immobility. When the pyrexia ceases, and the loss of power persists and increases, the existence of paralysis is always unmistakable. The initial general disturbance must then be regarded as part of the disease, unless there is the clearest evidence of its independent nature.

In adults, the danger of mistaking paralysis for prostration is considerably less, but, on the other hand, the general symptoms are as likely to be misinterpreted as in the case of children. The rheumatoid pains, which are so common, are usually regarded as evidence of acute rheumatism, especially when the affection follows exposure to cold. When severe rheumatoid pains are not localized in the joints, and especially when they are spontaneous, and not influenced by movement, the possibility of their spinal origin should be remembered, and other indications of spinal mischief, such as local loss of power, tingling or formication, should be carefully watched for and receive due weight.

Of other spinal cord diseases, all chronic lesions are distinguished at once by their slow onset and gradual development. There should thus be no danger of confusing the disease with any chronic form of muscular atrophy. From other acute diseases, moreover, the distinction is only difficult in the early stage. As soon as the initial palsy begins to clear, and the muscles in one part begin to waste, the nature of the case admits of no doubt. In the early period of the paralytic stage, the disease may be suspected if the paralysis is of wide extent, and the diagnosis may be made with certainty about the end of the first week by the electrical reaction. Care is necessary in making the examination, but if properly conducted it is not attended with the slightest risk. Faradaism will give all the information required, because if we find acute loss of faradaic irritability, it is certain that the characteristic alteration in voltaic irritability will follow. The isolated faradaic shocks may be employed with advantage, because the condition can be thus ascertained with much less stimulation of the sensory nerves than if the current is employed.

Acute transverse myelitis, affecting the dorsal region, causes paralysis of the legs, but, the lumbar enlargement being intact, there is no loss of electric irritability. Moreover, reflex action is not lost (except during the first day or two) as it is in the polio-myelitis, and at the end of a week the myotatic irritability becomes excessive, giving rise to an increased knee jerk and the ankle clonus. Sensation is usually impaired at first. The sphincters are usually affected, but since they are sometimes paralyzed in polio-myelitis, this indication is not absolute. If wasting occurs in these cases at a later stage, it is moderate and general, and is attended with no change in electric irritability or only with a slight diminution to both currents. But transverse myelitis, if seated in the cervical or lumbar enlargement, causes symptoms which very closely resemble those of severe cornual myelitis, since the gray matter is always implicated, and rapid wasting occurs with the same electrical reactions. Indeed, the two conditions cannot always be separated, since a severe cornual myelitis involves the adjacent lateral columns, and so is, in a degree, transverse. And yet the diagnosis may often be made—by the affection of the sensory tract, and by the greater degree of affection of the lateral columns, and the occurrence of bedsores, in transverse myelitis, or by the indications of slighter disease elsewhere in cornual myelitis. Thus transverse myelitis in the cervical region causes total and long-continued paralysis of the legs, often with anæsthesia; in cervical cornual myelitis the paralysis of the legs, if it occurs, is incomplete, and soon improves, while sensation is unaffected. In cornual myelitis of the lumbar enlargement there may be slight and transient paralysis of one or both arms—an indication that the morbid process is disseminated in distribution, which is absent in transverse lumbar myelitis. In extensive spinal hemorrhage the paralysis is absolute; there is pain in the back, and affection of the sphincters, while bedsores rapidly form, and the onset is sudden. Cornual hemorrhage, as already stated, is distinguishable from cornual myelitis only by the suddenness of the onset. The separation of subacute from acute myelitis of the gray substance is, to a large extent, arbitrary. The symptoms are similar, but the mode of onset is less rapid and occupies more than a week. The distinction of diphtheritic paralysis and of multiple neuritis is from the subacute rather than the acute spinal atrophies, and is considered in the next section.

The diagnosis from paralysis of cerebral origin is usually easy. It has to be made chiefly when the palsy is bilateral or hemiplegic, or, in the chronic stage, when the arm is paralyzed and the case simulates hemiplegia with recovery of the leg. Careful attention to the symptoms will generally prevent error. Loss of faradaic irritability never occurs in cerebral paralysis, nor does extreme muscular wasting. The skin reflexes may be lost in each, but the muscle-reflex action (myotatic irritability) which is lost in the spinal affection is always preserved, and often excessive, in cerebral palsy. In the latter, in children, mobile spasm and spastic incoördination are common in the hand, while in spinal infantile paralysis they are always absent. Slight paralysis (or spasmodic over-action) is common in the face of cerebral hemiplegia. Any cerebral symptoms which may attend the onset of polio-myelitis, convulsions, delirium, diplopia, headache, are brief in duration, whereas in cerebral lesions they may be very severe and continue for some weeks. The convulsions which attend the onset of infantile spinal paralysis are general, while in a cerebral lesion they are usually unilateral or commence unilaterally.

The exceedingly slow onset of pseudo-hypertrophic paralysis, developing gradually, as it does, with the child's growth, ought to render its confusion with polio-myelitis impossible. Cases in which the muscles are small instead of large, are sometimes confounded with acute spinal paralysis, but only by inattention to the absolute distinction of the mode of onset.

Diseases outside the nervous system which have been mistaken for infantile paralysis are chiefly those in which local pain interferes with the movement of the limbs, and the child is young. I have known, for instance, the mistake to be made in the case of hip-joint disease, necrosis of the femur, and the affection termed "scorbutic rickets," in which there is enlargement of the shafts of the long bones, extreme pain on movement, and spongy gums. In all these cases, a careful examination will show that movement is interfered with only by the pain; there is no actual paralysis, and there is no interference with reflex action. The preservation of the myotatic irritability is often of considerable assistance in these cases. It is common, for instance, for the extension of the knee to be interfered with, but the preservation of the knee jerk at once proves that atrophic paralysis is not the cause of the defect.

Prognosis.—The danger to life, in cases in which the nature of the disease is recognized, is extremely small, but it is possible that children sometimes die from the severity of the initial general disturbance, before the development of the characteristic paralytic symptoms. In the stage of paralysis there is danger to life only when the chief disease is in the cervical region. Death may occur at the end of a week or ten days from interference with respiration; the degree of which is a measure of the danger to life. Definite cerebral symptoms are also of very grave significance. In the vast majority of cases the disease entails no immediate danger to life. But children are left with little power of resistance to other morbid influences, and occasionally succumb to some other illness, as an acute specific disease, or an attack of bronchitis, a few weeks or months after the onset of the paralysis.

As soon as the paralysis has become stationary, *i. e.*, has not increased for twenty-four hours, the danger of further extension is extremely small. But the question at once arises, and is anxiously asked, What will be the permanent condition? Will there be lasting paralysis? An answer cannot be given until the end of the first week or ten days, and then only by means of an electrical examination. Whatever muscles, at the end of that time, have lost faradaic irritability, will certainly waste and remain for a long time paralyzed. On the other hand, if there is no loss of irritability at the end of ten days, but it is apparent at the end of a fortnight or three weeks, the wasting will be slighter in degree, and considerable ultimate recovery may be confidently looked for even in the most affected part. Where there is no loss of irritability, the paralysis will pass away in the course of a few weeks, or at most of a few months. Where irritability is lost tardily, there will be wasting and paralysis for several months. Where irritability is lost early the wasting will be rapid and great, the paralysis will last for one or several years, and it is unlikely that perfect recovery will take place. Without an electrical examination it is necessary to wait longer before a prognosis can be given, until distinct wasting on the one hand and improvement on the other, indicate the region in which the paralysis will persist and in which it will pass away.

In the chronic stage the prospect of ultimate recovery depends on the rate at which wasting develops, on the electrical reaction, and on the duration of the case. Where the wasting is great and has been rapid, and faradaic irritability is entirely absent, although some recovery may occur, it is not likely to be complete, and if this condition exists a year after the onset, it is improbable that more than very slight improvement will occur. On the other hand, if, at the end of one or two months, some faradaic irritability can be still detected, although low in degree (*i. e.*, elicited only by a strong current), considerable improvement is probable, and actual recovery is possible at the end of six or eight months. It is necessary, in the case of children, to remember, and warn the friends, that the growth of the most affected limb will be hindered, and that this, in the case of the leg, may render the effect of the paralysis more obtrusive by its interference with the gait, although real improvement may be taking place.

TREATMENT.—The treatment of the acute stage of the disease is essentially the same as that of myelitis, already described, and the rules and principles already stated need not be here repeated. In the initial stage it should be that of general febrile disturbance, consequent on a local inflammation. In such a disease, in which there is a natural tendency for the morbid process to cease to spread, and then to lessen in extent, the difficulty of ascertaining the actual effect of the drugs employed is extremely great, and hence the very different opinions which have been expressed regarding their influence.

When the acute stage is over and the condition of the patient is stationary, the general treatment should be tonic, iron and quinine being the most useful. The influence of strychnine is also probably beneficial, although its use is undesirable while the motor elements of the cord are the seat of an active morbid process.

Electricity has been strongly advocated, and largely used, in the treatment of this disease, and there is reason to believe that it is useful, although its influence has been much exaggerated. In no sense is it a curative agent, and there is no evidence that its application to the spine is capable of increasing the degree or accelerating the course of the recovery of the nerve elements. Nor is it easy to obtain evidence of its influence over the muscles. If the wasting is rapid, this progresses in spite of daily and sedulous applications. Nevertheless, its demonstrable effect on the muscles in causing their contraction must have an influence in the right direction upon their nutrition. Faradisation is useless, since the muscles cannot respond to it, but to the voltaic current they act readily, and their daily stimulation by its means tends to maintain their contractility. This is, it is true, of no avail if no recovery takes place in the spinal cord, but in most cases some recovery in the cord does occur, and when, after months, the nerve elements regain the power of conducting the voluntary impulse, the muscles are in a better condition to respond to it, if they have been regularly galvanized, than if they have been left alone, a prey to unhindered degenerative processes. The influence of electricity is clearly shown by cases in which no treatment has been adopted for six or twelve months after the onset. No response may at first be obtained to the current, but after a few applications contractility returns, and is often followed by slight voluntary power. The mode of application should be to place the positive electrode on the upper part of the muscle and to stroke the negative down it. The application can be made perfectly well by an intelligent nurse, since the diffusion of the voltaic current is so great that there is not the same necessity for the exact localization of the electrodes, as in the use of faradaism. Large well-wetted sponges should be used. The current should be no stronger than will cause distinct contraction in the muscle. In young children the application sometimes occasions considerable emotional disturbance. This may often be avoided by commencing with a very weak current. It is better, indeed, to commence with no current at all, applying the sponges in the way directed, so that the child may be familiar with them, and cease to fear them. Then two or three cells may be used, and the strength gradually increased day by day. In this way a current strong enough to cause contraction will often be tolerated. If, however, in spite of these precautions, this strength cannot be employed without distressing and disturbing the child, the attempt to obtain muscular contraction should not be persevered in, but only such a current employed as does not disturb the child. The influence of the current, if slighter, is still in the right direction, and the slightly greater influence of the stronger current is more than counterbalanced by the harm caused by daily severe emotional distress. The electrical treatment may be commenced at the end of the third or fourth week after the onset. It should not be used earlier, lest it excite increased disturbance in the spinal cord. The application need only be made to those muscles in which faradaic irritability is lowered or lost. Other muscles will either recover perfectly without its aid, or (as in the case of paralysis of the legs from implication of the lateral columns in the cervical region) will be uninfluenced by electricity.

Another measure which should be employed is systematic rubbing of the limbs. This stimulates the circulation, which is always defective, as the blue, cold surface shows. It no doubt also increases the movement of the fluids in the tissue outside the vessels, and so probably increases the interchange of material. The muscles should be daily rubbed, and kneaded, and gently pinched. The rubbing should be especially upward, so as to expedite the movement of the blood in the veins. No liniment is needed; the uncovered hand answers best. Cod-liver oil is sometimes rubbed in. There is no other objection to this than that it is apt to distract the attention of the rubber from the real object, and therefore from the proper method of the rubbing. But the oil does not reach the deeper structures of the limb sooner or in greater degree than if taken into the stomach. Great care should be taken, at all times, to keep the affected limbs as warm as possible.

In all cases in which the back muscles are weak, even in slight degree, bronchial catarrh must be guarded against with great care. In all such cases the muscles of respiration are weakened, and although normal breathing may not be impaired, the diminution in strength may render an acute bronchial catarrh rapidly fatal, and this even months after the onset of the paralysis.

A very important element in the management of the chronic stage is the prevention and treatment of the muscular contractions, and of the deformities to which these give rise. The contractions cannot be entirely hindered, but they may often be prevented reaching a high degree by careful attention to the position of the limbs, and by watching for and treating the earliest indication of shortening of the muscles. It is needless to discuss in detail the varied influence of posture in these cases; careful and frequent examination of the patient will show whether any deviation from normal relations is being developed, and its counteraction is chiefly a matter of common sense, and a little practical ingenuity. Of especial importance, however, are the prevention of the curvatures of the spine which are caused by allowing the patient to sit up before the back muscles have regained the necessary power, and the prevention of the contraction of the flexors of the hip and knee joints which occurs when the patient is allowed to lie in bed with the legs drawn up. More difficult of prevention is the contraction of the calf muscles, often aggravated in consequence of the lessened growth of the limb. Something may be done during the process of rubbing, for the prevention of deformities. While the contracted muscles are rubbed upward, they should be extended gently but firmly. Thus, if the calf muscles are shortened the foot should be steadily pressed upward while the calf is rubbed. A light Scarpa's slipper, worn at night, often aids in overcoming the contraction of the ankle joint. For developed deformities, due to great shortening of the muscles, surgical treatment by splints or tenotomy is usually necessary.

Mechanical appliances are of unquestionable value in the treatment of these cases during the chronic stage. They tend to counteract deformities, and to supplement weak muscles. Thus they often enable a child to walk, who could not do so without their aid. Achieving this they also do more. If a child cannot attempt to walk, muscles are unused which have regained some power, and power is often increased to a remarkable extent when instrumental aid enables these muscles to be brought into active use. In all these cases, however, the instruments require frequent attention and alteration to adapt them to the changes due to growth. Much patience and perseverance are required in the management of the case through its long and tedious course.

SUBACUTE AND CHRONIC ATROPHIC SPINAL PARALYSIS

(SUBACUTE AND CHRONIC POLIO-MYELITIS).

Under this designation cases have been described in which paralysis, followed by muscular atrophy, comes on less rapidly than in the acute form, its development occupying from ten to thirty days, in the cases which are termed "subacute," and from one to six months or even more, in those which are called "chronic." The shorter the period occupied by the development of the disease, the more distinctly does weakness precede wasting, and the more marked is the degenerative reaction in the muscles. But the cases included under this name differ much in their characters, and certainly belong to several distinct forms of disease. Some are diseases of the spinal cord, others are diseases of the nerves. The spinal forms are rare. The majority of instances of the subacute form are examples of multiple neuritis. The cases of atrophic paralysis in which the onset is not acute may be placed in three classes.

(1) Cases of subacute cornual myelitis which differ from the acute form, already described, only in their slower development. They present the same initial general disturbance, and wide extent, and the same recovery, except in a limited region in which muscular wasting occurs. Most of the sufferers have been adults. Little is known of the exciting causes of this form further than that it certainly sometimes follows exposure to cold. Frequently, however, no cause has been traced. The symptoms and general history of these cases present no important difference from the acute form.

(2) Cases which differ from the type just described in the important characteristic that their course is progressive instead of being regressive. The onset is subacute or subchronic, occupying from a fortnight to several months, but instead of arrest followed by improvement, the more or less rapid onset is followed by slower gradual increase, until at last widespread chronic musclar atrophy is developed. This variety was first described by Duchenne in 1849. By some it has been regarded as merely a chronic form of the acute affection, but the difference in course makes it probable that, as Erb suggests, it is really a distinct malady. This form also is chiefly confined to adults. It occasionally results from cold, and sometimes from injury, as a fall on the back. Intemperance and venereal excesses are also said to cause it. The widespread muscular atrophy which sometimes results from lead-poisoning is probably of the same nature.

In the subacute cases there may be slight fever at the onset. Pain in the back is common, and sometimes there is tingling or formication in the limbs. Loss of power usually commences in the legs, and steadily increases, with loss of all reflex action, but with no affection of sensibility. Muscular wasting occurs irregularly, some groups of muscles being chiefly involved. The upper limbs are affected after the lower, but in a similar manner. The muscles usually present the degenerative reaction, loss of faradaic irritability with increased and altered voltaic irritability, but in some cases, in consequence of the unequal affection of the nerve fibres, there is the mixed form of degenerative reaction (p. 45), faradaic irritability is preserved in the nerve, while in the muscles voltaic irritability is increased and altered. In many of the more chronic cases, however, there is a simple loss to both faradaism and voltaism. The sphincters almost invariably escape. Loss of power usually precedes wasting. In some cases, the affection exhibits a persistently progressive character, muscular atrophy slowly increases and extends, until the case ultimately resembles one of progressive muscular atrophy, and the patient dies at the end of one or two years from exhaustion, or from interference with the respiratory movements. In other cases, the atrophy, after slowly progressing for many months, becomes stationary, and considerable improvement may ultimately take place. This course is seen especially in traumatic cases. Few observations on the pathological anatomy of the affection have been made. Cornil and Lépine found, in one case, at the end of four years, softening of the lower part of the spinal cord, chiefly in the anterior cornua, disappearance of the ganglion cells, increase of the connective tissue, and sclerosis of the white substance around the anterior horns. The diagnosis from the first form rests on the slow progressive onset and course of the disease, and the fact that it either does not become stationary at all, or not until after many months. Although the degenerative reaction may be present in the muscles, it is frequently absent, and this is another distinction from the acute variety. On the other hand, the fact that paralysis precedes wasting is a distinction from progressive muscular atrophy. In the most chronic form, however, this distinction fails, and cases are met with which present a gradation between the two diseases. The prognosis is grave except in traumatic cases, but is influenced by the observed rate of progress, and the presence or absence of any indications of arrest. When the result of injury, considerable improvement is not uncommon, and it is remarkable how great a degree of paralysis and atrophy may, in these cases, ultimately pass away almost completely. The treatment of the disease must be influenced by the rapidity of its onset. In the subacute stage and form it should be conducted on the same principles as that of acute cornual myelitis. In the chronic form the treatment must be the same as for progressive muscular atrophy.

(3) There is no doubt that many subacute and chronic cases, which have been described as atrophic spinal paralysis, are peripheral, not central in their nature, and are cases of multiple neuritis, the symptoms and diagnostic indications of which have been already described. The history of the subacute spinal disease has been largely written from cases of multiple neuritis.

ACUTE ASCENDING PARALYSIS.

Several diseases cause rapid palsy, which commences in the legs and ascends to the muscles of the trunk, the arms, muscles of the neck, the diaphragm and the pharynx. Such ascending paralysis may be caused by meningeal hemorrhage, and by ascending myelitis, but it occurs also in cases in which the other symptoms of these lesions are absent, and in which the spinal cord, after death, appears healthy to both naked eye and microscopical examination.

The pathological nature of these cases is mysterious, but they cannot be classed with other recognized lesions of the spinal cord. The affection has been provisionally termed "acute ascending paralysis" or "acute general paralysis," in the absence of any evidence of its nature. It was first described by Landry in 1859, and hence is often called *Landry's paralysis.* Acute ascending paralysis is a formidable malady, most cases proving fatal in a few days.

CAUSES.—The remote etiology of the disease resembles, in general, that of acute myelitis. The disease affects males more frequently than females. It occurs chiefly between twenty and forty years of age, but has been observed, in rare cases, in older and younger persons, and even in children. Severe exposure to cold has been the exciting cause in many instances. The symptoms have occasionally come on during convalescence from some general disease, smallpox, diphtheria, typhoid fever. The affection has been ascribed to syphilis, and the symptoms have been said to pass away under antisyphilitic treatment. Difficult as it is to conceive by what mechanism this cause can be effective, many cases of the kind have been described.

SYMPTOMS.—Premonitory symptoms have been noted in a few cases—general malaise, pains in the head and back, tingling in the extremities—for a few days or a week before the onset. The first definite symptom is usually weakness of the legs, often commencing in one and spreading to the other. The weakness increases rapidly, so that the power of standing is lost, sometimes in a few hours, sometimes at the end of two or three days, and it goes on to complete paralysis, with relaxation of the muscles. As the legs become motionless the muscles of the trunk become weak, first those of the pelvis, loins, and abdomen, then of the thorax. The weakness next invades the arms; either the upper arm muscles or those of the forearm and hand may be first attacked, and one arm is often weakened before the other. The paralysis of the arms may become absolute, like that of the legs, or some power of movement may remain. The diaphragm and neck muscles then suffer, and difficulty of swallowing comes on, sometimes with paralysis of the palate, and often speech becomes difficult, nasal, and indistinct. The inability to swallow may become so great that the patient has to be fed through a tube, and the paralysis may involve the muscles of articulation to such an extent that utterance may be altogether unintelligible. There is dyspnœa from the weakness of the muscles of respiration.

The tingling and analogous subjective sensory disturbance have been followed, in some cases, by hyperæsthesia of the skin, and tenderness of the muscles. There may be some blunting of sensibility in the extremities as the paralysis becomes absolute, but there is no considerable loss of sensation. A firm touch can usually be perceived anywhere; perception of painful impressions and of heat or cold is sometimes delayed. At first reflex action is lost in the affected limbs, both cutaneous reflex action and myotatic irritability. In cases rapidly fatal the loss has continued till death. In cases that have recovered, reflex action has returned, but recorded cases have presented considerable differences in this respect, and, as there is some doubt as to the nature of many non-fatal cases, there is some uncertainty as to this point. In some the myotatic irritability has soon returned and has even become excessive. In others it has remained absent, and not until all paralytic symptoms have disappeared, has the knee jerk returned.

In spite of the early flaccidity of the muscles, if life is prolonged they present neither wasting nor change in electrical irritability. A trifling reduction in size may occur, but there is no muscular atrophy such as occurs in polio-myelitis, and even after several weeks the most careful examination fails to reveal any abnormal electrical reaction. According to observations by Remak and others, this may be regarded as characteristic of the disease. The sphincters, moreover, escape in the vast majority of cases, but not in all ; there is no tendency to the occurrence of bedsores. The cerebral functions are not involved, and the state of the patient who, with unimpaired intellect, cannot express himself either by speech or gesture, is painful in the extreme.

As a rule there is no elevation of temperature, even during the rapid development of the symptoms. It is not, however, certain that there is never initial fever. In one or two cases, which were not fatal, brief pyrexia attended the onset, and in a few others febrile disturbance occurred during the subsequent course of the affection. In at least one febrile case the symptoms passed away with a rapidity and completeness that seem incompatible with an ordinary inflammation of the cord. An acute enlargement of the spleen was first noted by Wesphal, and has since been observed in several other cases, so that it is perhaps constant.

Thus the chief feature of the disease is an almost purely motor paralysis, progressive in character, with relaxation of the muscles during the acute stage of the disease. Although usually ascending in course, the arms are occasionally involved before the legs, and the bulbar symptoms have been known to precede the others. The muscles of respiration are sometimes little affected until after the paralysis has reached the throat. Very rarely other cranial nerves have suffered in slight degree ; there has been diplopia, paralysis of accommodation, or dilatation of one pupil. In one case the face was paralyzed on both sides. The disease varies considerably in the rate of its progress. It may run its course and end in death in forty-eight hours. A large proportion of the fatal cases last less than a week. On the other hand, the disease may only attain its height at the end of two, three, or even four weeks. In some cases, apparently of the same character, the paralysis of the limbs has not been complete and bulbar symptoms have been absent. There may be a rapid extension of the paralysis to a certain point, and it may then cease to spread, and the limbs last and least affected may slowly regain power, the order of recovery of the muscles being the opposite to that of their invasion. When recovery has occurred, two or three months have usually elapsed before the weakness entirely disappeared, but in a few cases there has been a more rapid improvement and the patient has been well in a few weeks. It is probable, however, that some cases recorded as recoveries from this disease have been cases of myelitis, or of rapid multiple neuritis.

PATHOLOGY.—In several cases the most careful and skilled examination has failed to discover any morbid appearance in the spinal cord, nerves or muscles. Minute hemorrhages, met with occasionally, have probably occurred during the last moments of life. In a few cases, apparently similar to the others, slight scattered indications of inflammation have been found in the gray matter of the cord, with degenerative changes in the ganglion cells (Immermann). In addition to the enlargement of the spleen observed during life and found also after death, the abdominal glands have been found swollen. Bacteria were found in the glands by Baumgarten, but others (Wesphal, Kahler and Pick) have looked for them without success.

In the absence of any constant anatomical changes the nature of the disease is a matter of speculation, which is the more uncertain since the clinical separation of this from other maladies is often difficult. The traces of inflammation in the gray matter, which have been found in a few cases of ascending course, have been regarded by some writers, in France especially, as an indication that the disease is really a form of polio-myelitis, inseparable from the common form of acute atrophic paralysis. But the fact that, in patients who survive, there is no wasting and there is no change in the electrical irritability of the muscles, seems opposed to this view, and the common absence of fever makes it very difficult to regard the lesion as a primary inflammation. These considerations, coupled with the frequently rapid course of the malady, have suggested to many pathologists the idea of a toxic influence acting on the cord; and this idea receives support from the discovery that acute swelling of the spleen is common, and of the lymphatic glands not rare. Such lesions indicate a morbid blood state. The fact that paralysis of accommodation has been observed affords some confirmation of this view, since the isolated acute paralysis of a functional centre, not anatomically separate from others, is known only as a consequence of toxic influence. If this hypothesis is admitted as probable, the question remains —on what nerve structures is this toxic influence exerted? It is clearly upon some part of the motor path. The absence of atrophy or altered reaction of the muscles shows that the interruption is not in the lower segment of the motor path (p. 130), while the flaccidity of the muscles and the prolonged impairment of myotatic irritability suggests that the structures on which the morbid influence acts are not far from the lower segment, are in or near the gray matter in which that segment begins. Curara abolishes the function of the termination of the lower segment of the motor path, *i. e.*, of the nerve endings in the muscles. It may be that, in this disease, some toxic influence impairs in an analogous manner the function of the termination of the upper segment, paralyzes the ramification in which, as we have seen (p. 130), the pyramidal fibres must end in the gray matter, and by which they are connected with the lower segment.* It has been already pointed out (p. 154) that the nutritional stability of the termination is probably lower than that of any other part of each segment, since it is the part farthest from the nerve cell from which the fibre proceeds, and on which its nutrition depends. The greater length of the fibres for the lumbar region may render their termination more susceptible than that of the fibres for the arm, and hence the ascending course of the paralysis. It is, moreover, readily conceivable that the effect extends in slight degree to other structures in the gray matter; those, for instance, which intervene between the sensory and motor cells, and thus the loss of reflex action and myotatic irritability are explained, as well as the muscular relaxation, since, as we have seen, muscular tone must be ascribed to a reflex process. The structure affected may vary somewhat in different cases, and thus the state of reflex action may vary. The facts of diphtheritic paralysis also show that the effects of such toxic influence may lead to a secondary inflammation, and we can thus understand that indications of such inflammation in the gray matter may be occasionally met with. The bulbar symptoms come under the same explanation. The motor path through the nuclei of the medulla has the same pathological relations as that which passes through the gray matter of the spinal cord. The separate affection of nerve structures, distinct from others only in function, is common in the phenomena of toxic actions. There are no facts to show the nature of the blood state, its causes, or alliances, except that on the one hand the frequent relation of the disease to cold suggests an analogy with rheumatic blood states, while, on the other, its occasional relation to acute specific diseases is noteworthy. Perhaps the malady to which acute ascending paralysis bears the closest analogy is diphtheritic paralysis. The supposed relation of some cases to syphilis is as obscure as are most other points in the pathology of the disease.†

DIAGNOSIS.—The disease is recognized by the rapid development of ascending palsy with relaxation of the muscles, and with loss of reflex action, without fever, considerable pain, or loss of sensation, and if the patient survives, without wasting of the muscles or change in electrical irritability. The latter characteristic distinguishes the disease from acute atrophic paralysis, while the absence of pain in the back and of spasm is a distinction from meningeal hemorrhage. The diagnosis from general ascending myelitis has been already considered (p. 234).

PROGNOSIS.—The affection is one of extreme gravity. The danger to life is in proportion to the interference with respiration and with the functions of the bulbar nerves, and also to the rapidity with which the palsy develops. But the cases which develop with comparative slowness are not devoid of danger. Even when the symptoms only reach their height at the end of three or four weeks, death may occur in the same way as in the more rapid cases. On the other hand recovery has been known, although the patient has lost all power of motion at the end of the second day. The danger is great as long as the symptoms are increasing, and only when distinct improvement can be recognized is it justifiable to anticipate recovery.

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* The symptoms of lathyrism suggest that such an effect is actually produced in that toxic state.

† If a blood state arises in the course of syphilis, capable of abolishing the function of certain nerve structures, the fact is of great interest in connection with the relation to syphilis of degenerations of the spinal cord.

TREATMENT.—During the early stage of an attack of acute ascending paralysis, the treatment must be that suitable for acute myelitis, since, at the onset, the diagnosis between the two diseases can never be certain. A warm bath, or still better a vapor bath, should be given if the symptoms followed exposure to cold. It should be followed by counter-irritation over the spine. A long, narrow mustard plaster may be applied to the entire length of the spinal cord. More energetic counter-irritation, even the actual cautery, has been recommended. The little that is known of the pathology of the disease suggests that the dorsal position may be less injurious than in myelitis; but until the nature of the case is clear, the patient should be kept on the side if this does not involve frequent change of posture. The body should be kept in as perfect rest as possible. In very few cases have drugs appeared to exert any influence on the course of the disease, and the malady is so rare that experience accumulates slowly. Salicylate of soda seems to deserve a trial in cases that follow exposure to cold. Ergotin has been given, and one case in which it was used deserves special mention. The patient was a man aged fifty-seven, who, a week after exposure to cold and wet, complained of a feeling of weight and weakness in the legs; the temperature rose to 103°; the loss of power gradually became complete in the legs and spread to the arms, without loss of sensation. At the end of the second day there was a difficulty in swallowing, in articulation, and in breathing, and death seemed near. Ergotin was given every hour, and during the night the patient took twenty grains. In the morning the bulbar symptoms were better, the arms stronger and there was a trace of motor power in the legs. The patient rapidly improved, and at the end of a week was well.* If swallowing becomes difficult, care must be taken to administer a sufficient amount of nourishment, either by the rectum or by the nasal tube.

PARALYSIS FROM LESSENED ATMOSPHERIC PRESSURE: DIVERS' PARALYSIS.

Divers, and those who work in caissons, at such a depth beneath water that they are exposed to considerable pressure, sometimes become paraplegic soon after their return to the surface. Apoplectic attacks and hemiplegia also sometimes occur, but paralysis of the legs is by far the most common effect, and it is, therefore, clear that the spinal cord suffers in greater degree than any other part of the nervous system. Miners have been said to suffer also, but this is doubtful, because a pressure equal to at least an additional atmosphere seems necessary for the production of the symptoms. Most of the subjects of the disease have worked at a depth of from forty to ninety feet below the surface of water. The symptoms come on, not during the exposure to the increased pressure, but from half an hour to an hour after the return to the surface, and only if the individual has been previously exposed to the increased pressure for more than an hour. Those who are not used to such work seem especially liable to suffer, and there are also considerable individual variations in the degree of liability.

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* Sorgenfrey, *Neurologisches Centralblatt*, *1885*, p. 198.

Pains in the limbs, and often in the joints, precede the onset of the paralysis, which is usually sudden. In a few minutes the legs may be powerless. Sensation is often lost as well as motion, and in all severe cases the sphincters are affected. The arms are seldom involved. If the paralysis is incomplete the power may return in the course of a few days, but in severe cases the palsy usually lasts for weeks or months, and may be permanent. Death may occur quickly from acute cerebral symptoms, or after some weeks from bedsores, cystitis, etc.

A careful examination of the spinal cord has been made in only two cases (Leyden, Schultze). In neither was there any trace of hemorrhage, but in each there were indications of a slight disseminated myelitis in the dorsal region. These cases teach us nothing of the nature of the primary lesion. It is clearly related in some way to the diminution of the pressure to which the body has been exposed. It has been suggested that gases may escape from the blood and block up vessels, but it is difficult to see why this should occur especially in the spinal cord, and why such an effect should be delayed until an hour after the increased pressure is removed. Moxon has associated the effect with the imperfect blood supply to the lower part of the cord, and suggested that the long small arteries which pass along the nerves of the cauda equina may be compressed by the cerebro-spinal fluid. But it seems to be the dorsal and not the lumbar region in which the morbid process occurs. The pressure drives the blood from the surface, and probably lessens the amount in all vessels to which its influence extends. On the removal of the increased pressure the conditions must be reversed, perhaps in a degree proportioned to the duration of the pressure. Hence, the paralysis is perhaps due to an acute revulsive anæmia of the cord, analogous to that which sometimes follows loss of blood. The dorsal cord, to judge by inflammation, is more prone to suffer acute disturbance of nutrition than any other part of the central nervous system.

The treatment most suitable to this disease is probably that for acute myelitis.

HEMORRHAGE INTO THE SPINAL CORD: HÆMATOMYELIA.

Primary hemorrhage into the spinal cord, sufficient to cause symptoms, is a very rare disease, and it is even more rare than is suggested by the cases now and then recorded as such. We have already seen that hemorrhage may accompany myelitis; a considerable extravasation may occur when the inflammation is only commencing, during the stage of congestion, and while the symptoms are slight. Such cases are easily mistaken for primary hemorrhage. The risk of error is not always removed by pathological observation. Inflammation results from hemorrhage, and when indications of myelitis are found about a clot, it may be impossible to say whether these are primary or secondary. It is probable that many cases of secondary myelitic hemorrhage have been regarded and described as primary, and it is possible that a few cases of primary hemorrhage have been regarded as secondary. One writer, Hayem,* goes so far as to deny the occurrence of primary non-traumatic hemorrhage; but such an exclusive view is unwarranted. It is certain, however, that the history of primary hemorrhage has been largely written from uncertain data, and will need extensive revision when a sufficient number of exact observations have accumulated.

Etiology.—The rarity of hemorrhage into the cord is especially great in comparison with the frequency of hemorrhage into the brain. The difference probably depends on the tortuous and long course of the arterial path to the cord, whereby the vessels are preserved from the high pressure which is the chief cause of the degeneration, dilatation and rupture of the cerebral arteries. Miliary aneurisms are not found within the spinal cord. Hemorrhage is far more common in males than in females. It may occur at any age, but is most frequent between twenty and forty. It has been seen in young children, even so early as seven months,† and many of the subjects have been in advanced life. It is possible that the apparent preponderance of cases between twenty and forty is due to the inclusion of many instances of myelitis, which is especially common at that period. Of immediate causes, injury is the most frequent, especially falls which involve a severe concussion of the spine; the spinal column may or may not be injured at the same time. Over-exertion and exposure to cold have in rare cases preceded the onset. Chronic alcoholism and sexual excess have been thought to predispose. In a case related to me by Dr. Radcliffe, an extensive hemorrhage into the gray substance at the top of the lumbar enlargement, resulted from coitus four times repeated, the symptoms commencing suddenly during the fourth act. Minute extravasations are often found after death from diseases which interfere with respiration and cause venous congestion, and they are especially frequent in maladies which, at the same time, cause functional excitement of the cord. Thus they are common in tetanus and in convulsions. They cause no symptoms, and are probably produced during the last moments of life. They have been termed "*accessory.*" The diseases of the cord that lead to *secondary* hemorrhage are chiefly inflammation, tumors, and cavities in the cord. Thus we may distinguish four chief classes of hemorrhage: primary, secondary, traumatic, and accessory.

Pathological Anatomy.—The minute extravasations just mentioned, as met with after asphyxial and convulsive diseases, are found in both the gray and white substance, but especially in the former. They are usually microscopic, or visible to the naked eye as minute red points, distinguishable from distended vessels only by their slighter resistance to a stream of water. The extravasation may occupy the perivascular sheath, or the cavity in which the vessel lies, or extend between the nerve elements. The larger non-traumatic hemorrhages, which cause symptoms, always begin in the gray substance, and are often confined to it, extending into the white columns only when large in size. The vessels of the gray substance are more numerous than those of the white, have less external support, and probably undergo more considerable changes in state. The effusion forms a cavity in the cord, sometimes more or less rounded in transverse section, and half an inch or more in vertical extent. Sometimes the extravasation is irregular in shape. The cord is enlarged at the seat of the hemorrhage, and this may be visible externally as a dark swelling the size of a nut or a bean. Very rarely the hemorrhage tears the layer of cord which limits it, and blood, usually only in small quantity, escapes into the membranes. The tissue of the cord adjacent to the clot is usually broken down, stained, and softened, and inflammatory changes may be recognized in it with the microscope, just as in the neighborhood of hemorrhages into the brain. As in the latter, the effused blood slowly undergoes changes in tint, becoming first rusty and then yellow, and ultimately a cyst may remain. Several extravasations may coexist, usually in the same part of the cord. In contrast to this *focal hemorrhage* there may be an infiltration of the gray matter with punctiform extravasations, which appear, until closely examined, to be a single hemorrhage. The tissue between these small extravasations is broken down. It is probable that this form is always secondary to myelitis. In such secondary cases a careful microscopical examination usually reveals indications of inflammation much more extensive than the area affected by the hemorrhage. A growth into which hemorrhage occurs is usually a glioma, unaffected parts of which will be found in the neighborhood of the extravasation. The cavities in the cord into which blood may escape are sometimes of considerable vertical extent. I have known a fissure in the posterior column to be filled with blood through almost the whole length of the cord.

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* "Des Hémorrhagies Intra-rachidiennes," Paris, 1872.

† Clifford Allbutt, *Lancet, 1870*, vol. ii, p. 84.

Symptoms.—Slight symptoms, "prodromata," have been observed in some cases, chiefly in the form of trifling sensory disturbance, tingling, etc., in the limbs afterward paralyzed. They have existed for a few hours or days, or even for two or three weeks before the onset. It is probable, however, that these have been cases of secondary myelitic hemorrhage, and that there are no premonitory symptoms in primary extravasations. The actual onset is always sudden; the symptoms attain a considerable height in the course of a few minutes. Occasionally there has been transient loss of consciousness without any cerebral lesion, or rarely the onset has been by a series of successive sudden augmentations of the symptoms. Sometimes the symptoms come on during sleep. The suddenness of the onset is the characteristic of the disease; the symptoms which thus develop vary according to the seat and extent of the extravasation. In the majority of cases there is paraplegia, complete motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Pain commonly but not invariably accompanies the sudden palsy; it may be felt in the spine, or in the sacrum, or round the trunk, or in the legs. If in the spine it is local, and does not extend through a considerable length of the spine, as in meningeal hemorrhage, and there is not the initial spasm and rigidity which characterize the latter disease. The spinal column may be tender at the seat of the hemorrhage. Usually the paralyzed muscles are relaxed. Sometimes they are the seat of early clonic contractions, or these may come on a few days after the onset. When the hemorrhage is in the cervical region all the limbs are powerless; one arm is often affected before the other. The state of reflex action varies according to the seat of the disease; if it is at first abolished it quickly returns to the leg (unless the hemorrhage is in the lumbar enlargement), and it soon becomes excessive. Vasomotor and trophic changes in the skin are common; there is often vascular dilatation; bedsores readily form, and cystitis often results. Frequently the secretion of sweat is increased for a time. The temperature is normal at the onset, but it usually rises in the course of a few days from secondary inflammation in the cord.

The palsy developed at the onset usually continues for a week or ten days, although the pain may lessen. The symptoms do not always increase during the stage of inflammation, perhaps because this only involves the structures which are already impaired by pressure; but if the patient has been brought near to death by the primary hemorrhage, the secondary inflammation may end life. Occasionally, moreover, symptoms of an ascending or descending myelitis may come on, and the former may cause death by its interference with the muscles of respiration. It is probable that this extension only occurs in cases of myelitic hemorrhage, in which the extravasation is merely an incident in the course of a commencing inflammation. In cases of primary hemorrhage the symptoms pass into a chronic stage, improvement being usually slow. Some lasting loss of power remains in most cases, and there is often some permanent muscular wasting on account of the frequency with which the hemorrhage is in the cervical or lumbar gray matter. Occasionally there is rapid recovery up to a certain point; a hemorrhage of small size may abolish conduction in the white columns by the suddenness with which it compresses them, and the effects of the pressure may quickly pass away.

Diagnosis.—The diagnosis rests on the actually sudden onset of the symptoms. It must be remembered, however, that we cannot assume that symptoms which come on during the night's sleep are of sudden onset. Neglect of this consideration sometimes causes a mistake in diagnosis. The mode of onset is a sufficient distinction from all other organic diseases, except hemorrhagic myelitis and meningeal hemorrhage. The former is distinguished by the existence of slight symptoms before the sudden attack. We are not justified in regarding as primary hemorrhage any case in which premonitory symptoms existed for more than a few minutes, unless such symptoms were so pronounced and sudden in onset that they might have been due to a small extravasation. Initial fever in any case makes myelitis probable. The distinction from meningeal hemorrhage has been mentioned in the account of the symptoms.

Prognosis.—In all cases in which the symptoms are considerable in degree or wide in range, the danger to life is great, and remains great until they begin to subside. The prognosis is better when the disease is in the dorsal region than when it is in the enlargements. It is better when sensation returns in the course of a few days, but if the enlargements are affected other sources of danger remain considerable. Early trophic changes also render the prognosis worse. After the onset is over it is guided by the general principles that determine the prognosis in myelitis.

Treatment.—The treatment of hemorrhage into the substance of the cord is the same as that of hemorrhage into the membranes (p. 213). Absolute rest is essential, and the prone position desirable. Ice should be applied to the spine opposite to the seat of the hemorrhage. Full doses of ergot or ergotin may be given: ʒss of the liquid extract or five grains of ergotin may be given by the mouth, or three grains of ergotin may be injected under the skin, and the dose may be repeated two or three times, at intervals of two hours. A few large doses are probably more effectual than smaller doses continued for a longer time, since the hemorrhage probably does not go on for long. The after-treatment must be that for myelitis.

DEGENERATIONS OF THE SPINAL CORD.

A large and important class of diseases of the spinal cord consists of those in which there is a slow degeneration of the nerve elements, with an overgrowth of connective tissue, and in which structures are affected that have a common function, while others that have a different function escape even when they are adjacent to the elements that are diseased. Affecting thus functional "systems," they are termed "system diseases." This fact affords strong reason for believing that the primary change is in the nerve elements themselves, and that the overgrowth of interstitial tissue is secondary. The process is thus analogous to that which occurs in the secondary degenerations, in which the first change is certainly in the nerve elements, the destruction of which is followed by an overgrowth of connective tissue, a "sclerosis" as it is termed. We have seen (p. 239) that the process is by some regarded as a slow, parenchymatous inflammation, an inflammation beginning in the proper functional elements of the organ. To term a process "inflammation," which proceeds so slowly that five or six years may pass before it attains a considerable degree (as is sometimes the case), and in which there is commonly no primary vascular disturbance, involves a change in our conception of inflammation; but so far as the process is concerned the question is one of name rather than of nature.

Another question of much greater interest is the relation of the two elements in the process, the wasting and the growth, the atrophy of the nerve tissue, the hypertrophy of the connective tissue. The failure of nutrition in the one causes an increased energy of nutrition in the other. We have seen this relation in the nerve fibres. The degeneration of a fibre is attended by an active growth of its nuclei and protoplasm. The nutrition of the two elements, the neural and the adventitial, is connected in the closest manner, but in inverse degree. It is important to recognize the fact that the process of growth of the connective-tissue elements is an active manifestation of the nutritional energy, which, when once excited, may be to some extent independent of its cause. It may, when very active, pass beyond its original limits. It may, when very rapid, have some of the characters of an interstitial inflammation.

The degenerative diseases are not numerous, but they are of great importance. They are degeneration of the anterior cornua and anterior root fibres, causing muscular atrophy; degeneration of the posterior columns and posterior root fibres, causing locomotor ataxy; and degeneration of the pyramidal tracts, causing spastic paraplegia, a disease the exact pathological position of which is not quite certain. These degenerations may be variously combined.

In every form of spinal degeneration there is a tendency for the morbid process to extend beyond the system in which it begins and to which it is, for a time, confined. This tendency is much more marked in some degenerations than in others. It is apparently due to two influences. One is the tendency of the secondary interstitial process to independent extension, by which it may invade structures adjacent to, but distinct from, those which first undergo degeneration. But the extension cannot always be explained by contiguity. More distinct systems of structure may also suffer, apparently because the tendency to decay involves them also, although in slighter degree and at a later date than the elements first affected. In most degenerative diseases, in cases of severe degree and very long duration, almost all the elements of the cord may be in some degree involved.

LOCOMOTOR ATAXY (TABES DORSALIS: POSTERIOR SCLEROSIS).

The malady thus named is the most common chronic disease of the spinal cord. It consists in a degeneration in the posterior columns of the spinal cord, or the peripheral sensory nerves, or both, and is manifested, when considerable, by incoördination of movement, peculiar pains, and defective sensibility. The name "locomotor ataxy" was given to the disease by Duchenne; "tabes dorsalis," or "wasting of the back," is a term applied by Hippocrates to certain symptoms supposed to be due to venereal excess, and long ago limited in Germany to symptoms believed to depend on atrophy of the spinal cord. It then included all chronic paraplegias, but was further restricted to this disease by Romberg. It has lately obtained wider use on account of the discovery that incoördination may be absent when the disease is slight in degree.

The symptoms vary much in different cases. Besides the varieties thus produced, there are two forms which present many and important differences from that commonly met with. One is the so-called "hereditary ataxy." The other is that in which both weakness and incoördination coexist from the first; "ataxic paraplegia" it may be called. Each differs from other varieties with sufficient constancy to merit separate consideration. These varieties are not included in the following account.

History.—The incöordination of movement and other symptoms of the disease were frequently noted, during the first third of this century, in cases of disease of the spinal cord, but such cases were not distinguished from those with actual loss of power. The incoördination was found to be associated with disease of the posterior columns by Stanley.* The first really exact account of the disease was published in 1847 by Todd,† who distinguished the cases with incoördination and without weakness, from simple paraplegia, and apparently not aware of Stanley's observation, he inferred (from the character of the symptoms and his theory that the posterior columns contain fibres connecting segments of the cord at different levels) that the posterior columns would be found diseased; and he verified this inference by finding in two cases disease of these columns. The credit of the discovery of the disease belongs, if to any one, unquestionably to Todd. Four years later (in 1851) Romberg described the disease and the lesion in the posterior columns, but he failed to exclude loss of power from the symptoms. Russell Reynolds in 1855 gave an accurate description of the symptoms, and was the first to attribute them to muscular anæsthesia.‡ A series of cases was described by Gull in 1856 and 1857. Türck first observed, with the microscope, the wasting of the fibres in the posterior columns. Duchenne in 1858–9 published an independent and very able analysis of the symptoms of the disease, and gave it the name "locomotor ataxy." He obtained for it (with the help of Trousseau) the recognition that previous descriptions had failed to secure.

Causes.—The disease is much more frequent, in this country at least, in urban than rural populations. Inherited influence is to be traced only in a small proportion of the cases, perhaps in not more than 10 per cent. It is usually a general neurotic heredity, manifested by the occurrence in relatives of such diseases as insanity, epilepsy, and other degenerative diseases of the nervous system. Instances of this are—father epileptic; father insane; two sisters insane. Direct inheritance of the disease is extremely rare (the special hereditary form being always excluded). A boy with distinct symptoms, and optic nerve atrophy, was the son of a man who presented characteristic indications of the early stage of tabes (Remak).

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* *Med. Gazette*, Feb., 1840, and *Med.-Chir. Trans.*, vol. xxiii.

† "Cyclopædia of Anatomy and Physiology," vol. iii, p. 721.

‡ Reynolds, "Diagnosis of Diseases of the Brain, etc.," 1855, "Anæsthesia Muscularis." Certain words are worth quoting: "It appears most probable that the centripetal tract of fibres is affected; and that the locality of lesion is very variable."—P. 165.

Males suffer far more frequently than females, the proportion being about ten to one. When every allowance has been made for the different incidence of causal influences, the fact seems to indicate a proclivity inherent in the male sex. The same preponderance of males obtains in a disease that has some alliances with tabes—general paralysis of the insane. The middle period of adult life is that in which the disease usually commences. No less than half the cases begin between thirty and forty, one-quarter between forty and fifty, and rather less than a quarter between twenty and thirty. It rarely begins after fifty, but I have once known it to develop at sixty-six. Under twenty it is still more rare, but cases have been seen as early as ten and twelve.

Among the individual causes, one overshadows all the rest—the influence of syphilis. A very large proportion of the sufferers have had, at some previous time, constitutional syphilis, either distinct secondary symptoms or an indurated sore. The proportion is almost as large in the upper and middle classes as in the lower. Of fifty consecutive cases of the disease in men, seen in private consulting practice, no less than twenty-nine, or 58 per cent., gave a history of a chancre known to be hard, or of secondary symptoms; and eight others had had a venereal sore of unknown nature. In the lower classes the proportion is higher, and has been estimated at 70 or 80 per cent., or even more. When a deduction is made for possible accidental coincidence, there remains at least one-half of the cases in which numerical coincidence must depend on causal relationship. It is probable indeed that, taking all cases, a causal proportion of two-thirds would be nearer the truth. In women, also, antecedent syphilis can often be traced, although rather less frequently than in men. At the same time the full facts are difficult to ascertain, since in married women syphilis often runs a latent course. But in the case of tabes that succeed syphilis the lesion is not syphilitic in histological character. In these, as in the cases in which syphilis can be excluded, the disease is a degenerative one. It is, moreover, rarely influenced by anti-syphilitic treatment. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. It is probably the influence of syphilis that determines the greater incidence on the urban than on the rural population, and the preponderance of cases in middle life. Inherited syphilis also seems capable of causing the disease. In several cases of tabes in young persons, indications of inherited syphilis have been present. I have seen one example of this. The interval between the primary syphilis and the first symptoms of the disease varies from two to twenty years. It is, however, rarely less than five years, and in most cases it is between six and twelve years. Occasionally the malady develops as a sequel to some more distinctly syphilitic lesion of the cord.*

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* Although an occasional relation to syphilis had been noted by several preceding observers, Fournier was the first (in 1876) to assert the wide extent of this relation. His statements were received with doubt, because syphilitic patients constituted his field for observation, but they were confirmed from the neurological side by myself (*British Med. Journal*, March 1, 1879) and Erb (*Arch. f. klin. Med.*, July, 1879); abundant corroboration has

It is certain, however, that syphilis is not the only cause of the disease. In a few cases, perhaps 10 per cent. of the whole, it can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex.

One of these causes, which can sometimes be clearly traced, is injury, such as involves concussion of the spine. The immediate results of the injury, whether slight or grave, transient or lasting, are followed by the symptoms of the degenerative malady. In one case a man fell from a height on to the deck of a ship; transient paraplegia resulted, but six weeks after the accident the man presented extreme ataxy, with good power, and no knee jerk (Arnold). I have known the symptoms to develop gradually a few months after a fall from a horse. Exposure to cold and wet has occasionally preceded the onset so directly that it must be regarded as a cause. In other cases the disease has succeeded excessive fatigue and over-exertion, and also certain acute diseases, especially acute rheumatism and typhoid fever. The latter is known to cause focal myelitis; except by such an agency acute diseases can hardly do more than excite to activity a preëxisting tendency. Alcoholic excess has been noted in rare cases, and the influence of this cause is intelligible since it is known to cause neuritis, and sometimes such a form of neuritis as occurs in tabes. Indeed, it is strange that this cause cannot be traced more frequently. Sexual excess has been supposed, by some, to be a cause; but its influence can rarely be traced, and its significance is uncertain, since sexual excitement is undoubtedly sometimes an early symptom of the disease.

Secondary Tabes.—Symptoms of locomotor ataxy sometimes succeed other diseases of the spinal cord, and such sequence is especially common in syphilitic subjects. Myelitis and syphilitic gummata may be thus succeeded by tabes. It is not surprising that a degenerative tendency, effective alone, should be determined in its operation by local disease. Thus an officer in India, who had had syphilis, having suffered for a day or two from pains in the back, took a bath in snow water, and in a few days his legs were absolutely powerless. He gradually recovered power, but could not coördinate the movements: as power returned, lightning pains came on, and a year afterward he presented the typical condition of locomotor ataxy. Again, a man, twelve years after syphilis, had a severe fall, followed by gradual loss of power, so that at the end of three weeks he could scarcely stand. The

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been since afforded; many who at first doubted have been convinced by fresh observations. The difference in the character of the lesion from that of changes known to be syphilitic has been urged as an objection by many writers, but such theoretical considerations must yield to facts, and instead of denying that this or that lesion can be produced by a given cause, we may have to widen our view of the operation of that cause. Other degenerations of the nervous system seem to have a relation to syphilis, and, as I wrote some years ago, "it seems that one effect of constitutional syphilis may be to induce a neuropathic state in which certain degenerative diseases of the nervous system readily occur" (*Lancet*, Jan. 15, 1881).

legs remained weak for a month, and then improved, but ataxy came on; three months later, power was good, incoördination extreme. A similar succession may occasionally be observed in those who have not had syphilis. In many cases, however, muscular strength does not rise to the normal standard, and the condition is one of combined weakness and ataxy.

In recognizing the fact that tabes is occasionally secondary to a primary myelitis, it is necessary to note that myelitis may also occur in the course of tabes. We must, therefore, ascertain the absence of preceding tabetic symptoms before the disease can be regarded as purely secondary.

SYMPTOMS.—A typical case of developed tabes presents certain motor, sensory, and reflex symptoms. There is incoördination of movement of the legs, sometimes of the arms also, without loss of power or muscular wasting. There are pains in the affected parts, especially sharp momentary "lightning" pains; there is some loss of sensation; there is often loss or diminution of reflex action from the skin, and almost always entire loss of the myotatic irritability that is revealed by the so-called "tendon reflexes," and especially by the knee jerk; there are retention or incontinence of urine, constipation, and often loss of sexual power. Of this group of symptoms, two usually precede the others—the pains, and the loss of the knee jerk. These may exist alone, even for years, before incoördination comes on. Thus the symptoms are far wider in range than the name "ataxy" suggests, and while incoördination, if it exists, is the most obtrusive objective symptom, it is not the essential symptom of the disease, and, indeed, may never be developed. Hence physicians have extensively fallen back on the older term "tabes dorsalis." But recent expansion of our knowledge, especially the discovery of the extent to which the symptoms may depend on disease of the peripheral nerves, shows that even the qualifying "dorsalis" narrows the term within the pathological conception of the disease.

Besides the symptoms above enumerated, others are occasionally present. Of these the most important are atrophy of the optic nerve, and occasionally of other cranial nerves, trophic changes in the skin, the bones and the joints, and an ultimate extension to motor paralysis and muscular wasting. The combinations of symptoms present in different cases vary much. It will be convenient to consider first the symptoms in their various degrees, and then their grouping and sequence.

Motor Symptoms.—The characteristic incoördination of movement develops gradually. It is always increased (as Romberg first pointed out) by closure of the eyes, and at first may only exist when the guiding influence of vision is thus withdrawn. Before it causes ataxy of movement, it may render difficult the maintenance of equilibrium when the base of support is narrowed by the feet being placed close together, toes and heels. If then the eyes are closed, the patient sways, and may even tend to fall. In health slight unsteadiness is thus produced, varying in degree in different persons, but never amounting to even a suggestion of a fall. The effect of closure of the eyes is greatest when sensation in the soles of the feet is defective, but does not depend on this loss; it may be marked when sensation on the soles of the feet is perfect. The early defect in coördination may be discovered by the patient when he walks in the dark, or, not uncommonly, when he shuts his eyes in the process of washing the face. In a further degree of incoördination there is inability to stand with the feet together even when the eyes are open, and the patient is only steady when the feet are wide apart. If the feet are bare, the difficulty is greater, because muscular action has to replace the rigid base of the boot. The irregular contraction of the muscles is shown by the conspicuous movement of the tendons on the back of the feet. The patient may oscillate from toes to heels before he comes to rest. As the defect progresses, uncertainty is felt in walking even with full visual guidance, especially on uneven ground, or on a very smooth surface. A slight visible alteration in gait is then appreciable, the feet are not placed on the ground quite as in health, or there is distinct difficulty in maintaining equilibrium when the patient turns quickly. He has to put a foot down suddenly to keep from falling. The alteration is greater when the eyes are closed, and is especially conspicuous on an attempt to walk backward—a test that I have seen employed by Sir William Jenner many years ago. As the incoördination increases, the change in gait becomes greater, but varies much in its precise characters, probably according to the muscles that are most affected. Often the feet are raised too high, thrown forward too far, brought down too suddenly, and the whole sole comes in contact with the ground at once. There may be a tendency for one foot to be brought in front of the other, or, in avoiding this, the feet may be moved too far outward. Often the foot becomes inverted when it touches the ground. Efforts to correct error in movement have themselves to be corrected. In other cases the defect in maintaining equilibrium is greater than the disorder of movement; the patient sways about in the manner of one who has cerebellar disease. As the defect progresses, the patient is only able to walk by steadying himself with a stick, or by taking hold of the arm of another person, or of adjacent objects. At first a very slight degree of this help is sufficient; guidance rather than support is needed. Afterward, however, considerable support is necessary, and ultimately the patient may be unable to stand even with help. When he attempts to rise, the legs move hurriedly forward and backward, and if the upright posture is at last achieved the legs slip forward, and only strong support saves the patient from a fall. The ataxy is manifest also in other movements of the legs. If the patient, when lying, tries to touch an object with his foot, the leg is moved irregularly, goes beyond the place, and then is brought too far back, and only at last does it come in contact with the object, often with unintended force. This incoördination, like that in standing, is much greater if the eyes are closed.

The arms may present similar incoördination, although they may escape, even when the affection of the legs is extreme. Sometimes both legs and arm are deranged together; very rarely the arms suffer before the legs. The commencing defect is first revealed by delicate movements, such as writing.

When slight it may be conspicuous if the patient tries, with closed eyes, to touch some object, such as his own nose, or, having abducted his arms, tries to bring the forefingers together. As it increases, all movements become irregular; it is impossible for the patient to button his coat, or to pick up a small object from the table; the fingers twist about in the attempt, and the grasp is not sustained; first one finger is felt to relax and then another. If the patient attempts to hold out his hand in a fixed posture it is seen that the same irregularity obtains; instead of a uniform balanced contraction the muscles contract and relax involuntarily, and slow, unintended movements of the fingers result (Fig. 106), sometimes closely resembling those of athetosis. The same spontaneous movements may also be observed in the legs. They cease at once when the muscular effort is relinquished. Occasionally the muscles of the trunk present a similar ataxy. Thus one patient could sit steadily on a chair when his eyes were open, but if he closed them would at once fall off. In such a case, if the legs are but little affected, there may be (as I have seen) scarcely any incoördination of movement of the legs, but extreme unsteadiness and reeling on an attempt to stand or walk. The movements of the head, face, tongue, and eyes always escape derangement.

Even with extreme incoördination, motor power may be unimpaired. Occasionally some group of muscles, as the flexors of the ankle, become weak for a little time and then strong again, just as may the eyeball muscles, as we shall presently see. In some cases motor power in the limbs remains unimpaired to the end; more often, when the ataxy has become great, some muscular weakness supervenes, with or without wasting of the weak muscles. There is a distinct group of cases in which weakness and ataxy come on together, but these are considered separately ("Ataxic Paraplegia").

Sensory symptoms are prominent in most cases. They consist of subjective sensations, chiefly of pain and loss of sensibility. Spontaneous pains are present in some degree in nine-tenths of the cases of tabes. The most frequent and characteristic are the sudden and lancinating pains called "lightning pains." They occur chiefly in the legs, but may be felt in the trunk, arms, and even in the head. They are usually paroxysmal; attacks of such pains last for some hours or for a day or two, varying in seat, but often felt in the same part throughout an attack. Sometimes they are felt in a limited area of the skin, sometimes they seem more deeply seated, sometimes they dart down the limb. They may correspond to a nerve trunk, but more frequently have no relation to the nerves. Although the pains are usually acute and "stabbing," they are sometimes of different character. Thus in one patient the sensation was as if both legs were, for a moment, on fire. When referred to the skin, this often becomes tender, so that even the contact of the bedclothes cannot be borne. When pains continue at one spot for some days, ecchymoses have been known to form there. In one case, after pains had been felt at one part of the scalp for a time, the growth of the hair was changed; each hair bent and broke short off, over an area the size of half-a-crown; after the pains ceased the growth of the hair became normal. A sharp pain may be accompanied by a sudden reflex spasmodic movement of the legs, or by inhibitory weakness. Thus, in one case, a sudden pain would often make the patient fall on his knees. The pains may be so severe as to prevent sleep for several days.

Other kinds of severe pain, less brief than the lightning pains, occur in some cases, and are described by various epithets, as "burning," "tearing," "gnawing." Burning pain in the toes distressed one patient. Much more common, and occurring with other pains, are slighter dull pains like those of rheumatism, for which they are often mistaken, an error that is facilitated by the circumstance that any of the pains of tabes may be influenced by weather, being especially increased by damp cold. A painful sense of constriction is felt by some sufferers in the legs, groins, genitals, or trunk; it is less common than in myelitis, but when it does occur it is sometimes very intense; the patient may feel as if the whole trunk were tightly enveloped in a cuirass of brass. In the hands pains are common, but are usually slight in degree, and are occasionally felt chiefly in the ulnar area. Visceral pains may also occur, usually paroxysmal, and referred to the stomach, bladder, rectum, etc. These will be again considered in connection with the visceral symptoms of tabes. Pains are not only the most common, they are often the earliest symptom of the disease. Nevertheless they are sometimes slight, and occasionally are altogether absent. Perhaps they sometimes occur alone; I have seen two patients who suffered characteristic lightning pains, but presented no other symptoms of tabes; the tabetic nature of the pains is therefore uncertain.

Sensations other than pain are also common. They are various in character, described as "tingling," "pins and needles," "creeping," and very often as if the skin, especially of the soles, was covered by some soft substance. Sensations of cold or of heat are also common; the feet may feel as if the legs were always in cold water; a sensation of cold about the testicles is occasionally complained of. The sense of heat may be so intense as to amount to pain, as already mentioned. Similar cutaneous sensations are also common in the hands. Increased sensitiveness of the skin, chiefly to pain, may also exist, especially on the soles of the feet, and often accompanies lessened sensibility to touch.

A diminution of sensibility is also present in most cases of the developed disease. It may involve all forms of sensibility, or one much more than the others, or some forms may be much impaired and others normal. It is equally common for either pain or touch to be affected alone; for instance, a prick may cause no sensation of pain, although the least touch of the finger is everywhere perceived. A firm touch (*i. e.*, slight pressure) may be felt when a slight touch is unperceived. When a touch is felt, the power of localizing it may be impaired. Temperature sensations are rarely affected without other forms of sensibility, but they may be impaired with pain when tactile sensibility is normal. On the other hand, there may be extensive loss to pain without any defect of the temperature sense. A prick may then cause only a sensation of heat, and extreme degrees of heat or cold cause pain less readily than normal. The temperature loss may be partial—either cold or heat may be unperceived while the other is recognized. When touch is slightly impaired there may be an inability to discriminate differences in the degree of pressure on the skin.

Accompanying the diminution or increase of sensitiveness there are sometimes curious changes in the sensation produced. One of these is a delay in the perception of pain, which may amount to several seconds. The greatest delay I have met with amounted to seven seconds, but one of fifteen seconds has been described (Eulenberg). The contact of a point may be felt at once, the pain only after a considerable delay. Often there is an after-pain lasting sometimes for a quarter or half a minute or longer, and the maximum sensation may not be attained until some seconds after the pain is first felt. Thus, in a case in which there was a delay of seven seconds, the maximum sensation was only felt twenty-five seconds after the prick (Obersteiner). A second increase of pain has been noted (Naunyn). I have known a sort of rhythmical recurrence of sensation; the pain was not felt for several seconds, and then the sensation was repeated several times in successive darts of pain. A delay greater than normal may also attend the sensation of heat. The localizing power is sometimes strangely perverted. A touch or prick on one leg may be referred to the other, sometimes with singular uniformity in position (allocheiria). A prick in one spot may be felt in many places (polyæsthesia) on the same or (as I have seen) on both legs. It is said that the reaction of the sensory nerves to electricity may be changed in the same way as that of the motor nerves is changed when they are degenerated. Instead of the earliest sensation occurring on closure of the circuit, at the negative pole (kathode), as in health, it may be felt at the positive pole (anode). (Mendelssohn.)

The region in which sensibility is impaired varies much in different cases. The loss occurs most frequently, and is usually greatest, in the lower part of the legs and feet. It may be limited to the soles, and usually then ceases at the outer sharp edge of the foot, but extends for an inch or so up the inner side. Touch or pain may be lost on the sole only, and even, strange to say, the form of sensibility that is lost on the sole may alone persist in the legs. Although the sole is usually most affected, sensation may be perfect on the sole when lost elsewhere. One leg is often more affected than the other. The loss may extend to the trunk or even the neck. In the arms it is usually greatest on the hands, and often commences on the palmar aspect of the fingers. Sensation may be lost on the trunk and not on the limbs, an important fact, because the anæsthesia may then readily be overlooked. On the head a loss is rare, but has been noted on the cheeks.

The loss of sensibility is not confined to the skin, but affects the deeper tissues also in many cases. The contraction of the muscles on electrical stimulation may be unfelt, and the muscles may be insensitive to pressure or forcible extension. It is probable that the sensibility of the joints, fibrous tissues, and tendons is also involved. The position of the limbs in passive as well as in active movements may not be perceived. In slight cases this sense of posture may not be lost, but in severe cases it may be so absolutely abolished that one of my patients, who often fell out of bed, had always to look to see where his legs had gone to before he attempted to rise. The anæsthesia may extend to the viscera when the trunk is involved. In consequence of the loss of sensation grave injuries to the limbs, such as burns, may be unperceived, and visceral disease may be unattended with the customary pain. I have known a severe attack of pleurisy to be absolutely painless.

Cutaneous anæsthesia, although it is thus a common, and often an early, symptom, is not invariable. Every test may fail to reveal any loss, and this integrity of sensation may exist in patients who present considerable incoördination, and in those who suffer severe pains, as well as in those who have had no pains. But it is very rare to have no sensory loss when incoördination is considerable.

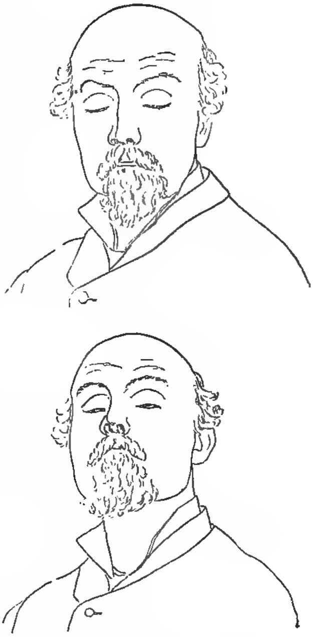
Reflex action from the skin is usually impaired in proportion to the loss of cutaneous sensibility, and especially to the loss of tactile sensibility. It may be normal when pain is unfelt. But reflex action may be lessened when there is no loss of sensation. The plantar reflex is most frequently impaired, and the progress upward of the disease may be attended by a progressive loss of the gluteal, cremasteric, and abdominal reflexes. On the other hand, in the early stage of the disease, there is sometimes a very marked excess of all cutaneous reflex action, and this may reach a degree rarely met with in any other affection. In one case a touch on the sole would make the foot fly up, and if the naked trunk was stroked, so violent a start was caused that the patient on one occasion rolled off a sofa. In this patient considerable ataxy coexisted with the excess, but the reflex over-action is usually an early symptom, and passes away as the ataxy becomes well developed.

The sexual act is, in part, a superficial reflex action, and increased sexual excitement sometimes coexists with the excess of reflex action. In the case just mentioned there was intense satyriasis. In the same manner the sexual power usually fails when the superficial reflex actions subserved by the corresponding region of the cord are abolished—the cremaster and the lower abdominal reflexes, but to this rule exceptions occur. Loss of sexual power is an exceedingly common and often an early symptom of the disease. It may precede any impairment of sensory or reflex functions. On the other hand, exceptions are met with in which sexual power persists to a late period of the disease, even until the patient has lost the ability to walk alone.

If the condition of cutaneous reflex action is somewhat inconstant, this is not the case with the reflex process on which the myotatic irritability depends. A loss of the knee jerk, as Westphal first pointed out, is one of the earliest and most constant symptoms of tabes. With it disappears, also, all other indications of this irritability. In cases with great excess of cutaneous reflex action, the knee jerk is lost as in other cases, and the contrast between the two forms of reflex action is very striking. It is exceedingly rare for the knee jerk to be obtained in any case of true tabes. Very rarely an early case may be met with in which it is not lost, although diminished, and commonly unequal on the two sides. In one or two cases I have found it slight on one side and lost on the other when distinct ataxy was present, and have observed its speedy entire disappearance. In one case, in which the jerk was lost in one leg only, lightning pains were confined to this leg. The loss often precedes for many years the development of incoördination. But the muscles preserve their irritability to percussion, the contraction thus produced being limited to the part struck. In cases of ataxic paraplegia, which are often confounded with true tabes, the knee jerk is not lost, and is usually excessive, and these, for the most part, constitute the cases sometimes described of locomotor ataxy without loss of this phenomenon.

Eye Symptoms.—The functions of the internal muscles of the eye are often affected in tabes. In no less than four-fifths of the cases the reflex action of the iris to light is lost. In most of these the contraction on accommodation is preserved—a characteristic first noted by Argyll-Robertson. Often, also, (as Erb has shown) the dilatation that occurs on painful stimulation of the skin of the neck, etc., can no longer be thus produced. Sometimes the ciliary muscle is also paralyzed, causing loss of accommodation. Very rarely there is loss of accommodation and of the associated contraction of the iris, without loss of the light reflex. The pupils are often very small ("spinal myosis"), especially when there is loss of the reflex dilatation from the skin, a loss which seems to be accompanied by atony of the radiating fibres supplied by the sympathetic. The pupils are not always small; they may be of medium size or beyond medium size, and then I have usually found that the skin reflex can be obtained, although the light reflex cannot. They are often not perfectly circular, and are sometimes unequal. Both eyes are usually affected in the same manner; occasionally the reflex action is lost in one and only lessened in the other. On the other hand, the action of the iris is sometimes perfectly natural, even in advanced cases.

FIG. 106.



Double tabetic ptosis.*

Paralysis of the external ocular muscles is also common in tabes, and occurs in several forms: (1) Transient weakness, lasting a few days or weeks and then passing away. (2) Permanent paralysis, complete or incomplete, of a single nerve or part of a nerve. Either form may occur at any stage, but the first is most common in the early, and the second in the later, stages of the disease. The transient palsy renders diplopia a common symptom in the early period; any muscle may be affected, but the external rectus is that which most frequently suffers. The persistent palsy may affect one or more muscles; often the levator is involved, causing what has been termed "tabetic ptosis" (Fig. 106). Sometimes the whole third nerve is paralyzed. (3) There may be a combined palsy suggesting a cerebral origin. Rarely there is loss of the movement of convergence associated with loss of accommodation, although the internal recti may act in the movement toward one side. (4) Many or all the muscles of both eyes may gradually become paralyzed, "external ophthalmoplegia," in consequence of disease of their centres.* This is very rarely met with in tabes, although it seems to bear a similar relation to syphilis.

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* From a Salpêtrière photograph, for which I am indebted to M. Charcot.

Atrophy of the optic nerve occurs in tabes, and this disease furnishes a large proportion of the cases of simple atrophy. Its frequency is difficult to ascertain, but is certainly less than is often supposed. It does not occur in more than one case in ten. It is generally an early symptom, usually commencing before incoördination is developed, and in a large number of cases ataxy never comes on, the spinal malady becoming stationary when the nerve suffers. It rarely begins after the gait is considerably disordered.

The failure of sight usually commences with a peripheral limitation of the field and loss of color vision, but sometimes central acuity fails early. When this remains good, the peripheral loss may reach a considerable degree before it is suspected by the patient. The visible pallor of the optic disk is not always proportioned to the failure of sight. The change in tint depends on wasting of the capillaries, and this is not always proportioned to the wasting of the nerve fibres. Ultimately the disk is grayish in tint, and the retinal vessels are but little narrowed.† Occasionally there are slight signs of congestion in the early stage of the atrophy, and then some tissue of gelatinous aspect may develop in the disk, and the vessels may be narrowed.

The course of the atrophy is usually slowly progressive, and, in most cases, it ends in total or almost total blindness. It may, however, cease to progress, and even undergo slight improvement. The onset is not always gradual. Sometimes sight fails considerably in the course of a few days without ophthalmoscopic changes to account for it. I have met with one case in which temporal hemiopia coming on rapidly suggested disease at the optic chiasma. The atrophy is often more advanced in one eye than in the other, and very rarely one eye only suffers. Flashes of light are occasionally experienced in the course of the atrophy, comparable to the lightning pains in the limbs, but they are not common. Headache is occasionally troublesome in cases of tabetic atrophy, and cephalic lightning pains are sometimes severe. Organic cerebral disease may be thus suggested.

Deafness, having the characters of nerve deafness, is met with in some cases, sudden or gradual in onset, transient or lasting. The persistent deafness has been attributed to an atrophy of the auditory nerve, analogous to that of the optic nerve, but certainly in many cases without sufficient reason.

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* Buzzard, "Clin. Lect. on Diseases of the Nervous System," 1882, p. 180.

† Further details will be found in "Medical Ophthalmoscopy," 2d ed., p. 163.

It is very difficult to distinguish an affection of the nerve from one of the labyrinth independent of the nerve. Even in a case of bilateral deafness coming on in the course of tabes, in which the patient could hear only a loud voice, and deep notes better than high ones, in which atrophy has been diagnosed, Lucae found calcareous masses in each of the labyrinths, and the auditory nerves were quite normal. But if there is a progressive limitation of the range of hearing, analogous to the peripheral limitation of the field of vision, we are justified in assuming nerve atrophy. I have seen two cases of this kind. In each there was also optic nerve atrophy, and the other symptoms of tabes were distinct. In one only notes between E^2 and D could be heard. * In the other the patient, when first examined, was absolutely deaf to notes above E^2 and below G_1. A few months later the restriction had confined the range of hearing to the octave in the treble clef, between E^2 and E^1, even $E\flat^1$ being inaudible. Thus the loss occurred chiefly from below upward. Ultimately no note could be heard. Attacks of vertigo are frequent in the cases attended by deafness, and seem to depend on the disturbance of the labyrinth or auditory nerve, and to be thus connected only indirectly with the primary disease.

The functions of the other cranial nerves are often affected. Pains may be felt in the region of the fifth nerve. Unilateral atrophy of the tongue has been noted in one case. In the larynx, besides the spasm to be presently described, paralysis of the vocal cords has been met with in rare cases, both as an isolated symptom and also after attacks of spasm have occurred for a long time. Both posterior cricoarytænoids have been paralyzed (Oppenheim), and in one case paralysis of one posticus occurred very early in the disease (Remak). A sort of ataxic irregularity in the movement of the cords has been described by Uhthoff.

Sphincters.—The functions of the bladder are frequently deranged; there may be either sluggish micturition or a tendency to incontinence, and often the bladder is not perfectly emptied. These symptoms frequently occur in the disease, and it may be indeed only for these that the patient seeks medical advice. Occasionally there is a curious reflex susceptibility on the part of the bladder; thus the urine always escaped from one patient when his hands were placed in cold water. The retention may become absolute, or there may be overflow incontinence; there is rarely paralytic incontinence. More commonly the affection of the bladder remains slight in degree, although even this may have serious effects. If the bladder for a long time is imperfectly emptied, cystitis is apt to occur, and secondary renal disease is a not uncommon cause of death when the difficulty of micturition has been insignificant. The sphincter ani is often also weak, so that a loose stool can be retained with difficulty, but its paralysis rarely reaches a high degree. Constipation is extremely common.

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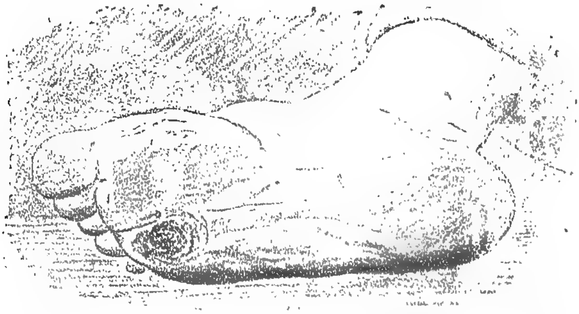
* The seven notes above the middle C of the bass clef are designated by simple letters, those of each higher and lower octave are indicated by small figures placed above and below the letter. Thus the middle C of the treble clef is C^2.

Vasomotor and trophic disturbances constitute a singular group of symptoms to which much attention has lately been drawn. Transient œdema of the limbs may occur without albuminuria, and is sometimes an early symptom. Local sweating has been noted, confined, for instance, to the palms and soles, or to one side of the head. The ecchymoses and altered growth of hair, in connection with attacks of pain, have been already mentioned. Pigment may disappear from the skin and hair in isolated spots. Herpes of the skin is not uncommon, and usually occurs in association with attacks of pain. I have twice known the penis to be the seat of herpetic eruptions. The epidermis of the sole becomes thickened; blisters readily form beneath it and may lead to indolent ulcers. The curious affection known as "perforating ulcer of the foot" has been found to be almost confined to cases of tabes. Troublesome ulceration of the toes, especially about the nails, is also occasionally met with, and may necessitate amputation. The growth of the nails of the feet, and sometimes of those of the hands, may be changed. They may be thickened and the surface furrowed and uneven.

FIG. 107.

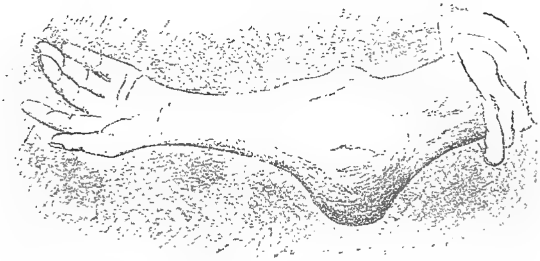


Perforating ulcer of the foot in tabes. (*From a Salpêtrière photograph.*)

The nails may even fall off and be slowly renewed. The teeth sometimes decay quickly, and may drop out. I have seen one patient, who had previously lost many of his nails, in whom all the teeth of the upper jaw fell out in the course of three days, without any pain or decay of the teeth. Wasting of the muscles may occur in the later stage of the disease, usually greatest toward the extremity of the limbs, and sometimes so great as to resemble that of progressive muscular atrophy.

Changes in the nutrition of the joints and bones have attracted much notice since attention was directed to them by the careful investigations of Charcot. They are not very common, but are sufficiently frequent and well marked to place their relation to the disease beyond doubt. The bones may become brittle, and may break readily in so-called "spontaneous" fractures. The process of union is attended with the formation of a large amount of callus. Sometimes there is ossification of the tissues adjacent to injured bones. In the joints the changes consist of erosion of the cartilage, wasting of the heads of the bones, ossification of the ligaments, and sometimes irregular bony growths. The movement of the joints may be thus too free or too limited. The wasting of the head of the femur renders dislocation of the hip easy. A remarkable instance of these osseous changes has been recorded by Charcot; a tabetic woman suffered successively, in the course of a few years, dislocation of both hips, fractures of the left femur, of both bones of each forearm, and dislocation of one shoulder; after death old fractures were found in both scapulæ and the left clavicle. With these changes, and often when they are slight in degree, there is effusion within the joints and occasionally œdema outside them. The larger joints are those most commonly diseased—knee, hip, ankle, elbow—but the small joints of the fingers have been known to suffer (Westphal). The lesions are sometimes excited by injury, and the extraordinary changes that may follow traumatic influence is alone a conclusive indication that abnormal trophic influences are at work. Fig. 108 represents the arm of a man who fell and struck his elbow, fracturing the olecranon and condyle. It was followed by extreme painless swelling, and bony masses could be felt, gradually increasing in size. After death the fractures of the bone were found, and masses of new bone had formed in the capsule of the joint.

FIG. 108.



Locomotor ataxy, painless swelling of elbow joint after a fall on it. One condyle of the humerus and olecranon were found, after death, to have been broken off. Several separate pieces of new bone had formed in the capsule of the distended joint.

The origin of the changes in some joints, as the knee, is more complex. This joint depends for its functional capacity partly on its ligaments, and partly on the support of the muscular tendons that pass by it, in one of which the patella is inserted. The sudden variations in the contraction of the muscles both render this support variable and determine sudden strains on the unsupported ligaments. Under this strain they yield, and when the patient stands the joint may become retroflexed as in Fig. 109. The strain on the ligaments and capsule leads to effusion, and may excite the more serious trophic changes already described. Changes in the tarsal bones and articulations may cause the foot to become flat, with a projection inward or backward of the tarso-metatarsal articulation, and of the tarsal bones. The condition has been called the "tabetic foot," by Charcot and Féré.*

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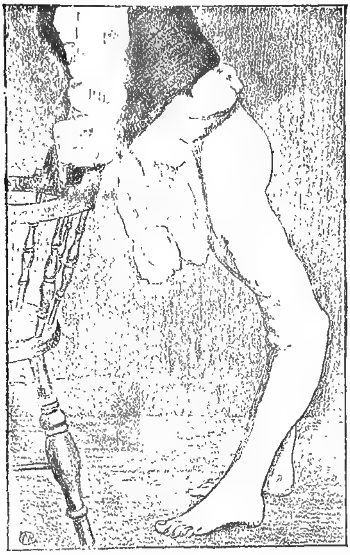
* The trophic changes in bone and joint have been regarded by some as simple chronic osteo-arthritis, without causal relation to the nerve disease in their subjects. All evidence

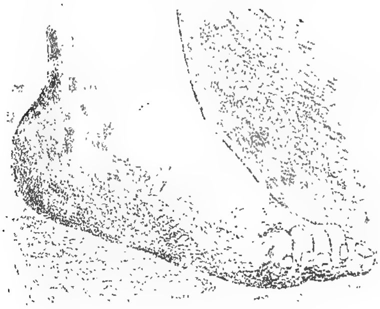
Visceral symptoms of peculiar character occur in many cases of tabes.* They consist almost entirely of paroxysmal disturbance of function, usually attended with great pain, and have been termed, by the French *crises*, with qualifying adjectives according to the seat of the symptom. There is a tendency to over-elaborate this terminology. The most frequent seat of such disturbance is the stomach, and these attacks are called *gastric crises.* They consist of paroxysms of severe gastric pain, felt in the epigastrium and often passing through to the back. The pain is accompanied by vomiting, with or without nausea. The vomiting is often incessant, and is first of food, then of clear liquid, which may be very abundant; ultimately bile is vomited, and sometimes blood. Retardation or irregularity of the action of the heart has sometimes attended such attack, and, rarely, pyrexia has been noted. I have known frequent hiccough to accompany the vomiting. Such an attack lasts for some hours or days, and then subsides, to recur in a few weeks. During the intervals the functions of the stomach may be performed in a perfectly normal manner. Although pain and vomiting usually occur together, some patients have attacks of pain without vomiting, and in others there is vomiting without pain. Rarely there is nausea alone. A temperate man, in the early stage of tabes, suffered during three years from frequent attacks in which, for several days, he had intense nausea each morning, passing away in the afternoon, and succeeded, in the evening, by an inordinate craving for food. He never vomited unless he made himself do so, in the vain hope of thus

FIG. 109.



Locomotor ataxy; retroflexion of knee joints.

FIG. 110.



Tabetic Foot.
(*From a photograph by Mr. Marriott, B. Sc.*)

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seems to me opposed to this view. The arguments that can be adduced in support of it will be found in some of the speeches delivered at a discussion at the Clinical Society, Nov. and Dec., 1885.

* The occurrence of attacks of vomiting, sometimes early in the disease, was pointed out by Topinard, but thought by him to be a mere complication. (Topinard, "De l'ataxie locomotrice," Paris, 1864, p. 273.)

obtaining relief. After two years attacks of laryngeal spasm were added to the attacks of nausea.

The chief *intestinal* disturbance is constipation, but paroxysmal diarrhœa has been supposed to be of vasomotor origin, and connected with the disease (Pierret). Paroxysms of rectal pain (*rectal crises*) are not uncommon, and are often accompanied by distressing tenesmus, sometimes by a sensation as if there was a foreign body in the rectum. In rare cases, paroxysms of pain have precisely the character of attacks of renal colic (*nephralgic crises*), or are felt in the neck of the bladder or along the urethra or at the meatus (*vesical or urethral crises*). These may be attended by an intense desire to micturate, although the bladder may be empty and only a few drops of urine may be expelled. In one patient such pain was repeated several times an hour, and each attack lasted for several days. Blood may be passed after an attack. Paroxysms of sexual excitement in women have been somewhat unnecessarily termed "*clitoris crises*."

"*Laryngeal crises*" are, perhaps, after those of the stomach, the most common of these symptoms. They vary much in character. The most common form is a true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping cough or of laryngeal stridulus. Pressure on the superior laryngeal nerve at its entrance into the larynx, or on the trachea, or the introduction of a sound, will sometimes induce an attack. The spasm may last for a quarter of an hour or for some hours, but rarely continues so long as the gastric crises. In one recorded case the spasm spread to the pharynx, making swallowing impossible; a very violent attack extended to the muscles of respiration and the patient died asphyxiated. Death from these attacks is, however, extremely rare. Paroxysms of rough cough have been termed "*bronchial crises;*" in one case such attacks ceased when the patient began to suffer from gastric crises. These crises are often early symptoms, and they may continue for many years. I have seen a patient, still in the first stage, who gave a history of gastric crises during the preceding eighteen years.

Comparable to these paroxysmal visceral disturbances, although very different in character and more alarming in aspect, are cerebral symptoms, which are, fortunately, very rare. They resemble the attacks that are common in general paralysis of the insane, and consist of transient apoplectiform seizures, hemiplegia, or convulsions general or one-sided. They may occur early in the course of the disease. The alarm they occasion is not without foundation, since death has been known to occur during an attack of apoplectic semblance. Paroxysms of vertigo sometimes seem to be of similar nature, but they more often depend on derangement of the auditory nerve.

Course and Termination.—It is convenient to divide the course of the disease into three stages: (1) In which there is no alteration in gait, the chief indications of the malady being the loss of the knee jerk and the pains, often associated with loss of the light reflex of the iris, and unsteadiness on standing with the feet together and eyes shut. (2) That in which there is distinct affection of gait, slight or considerable, but in which the patient is still able to walk, alone or with the aid of one or two sticks. (3) In which walking is possible only with the aid of another person, or it is impossible for the patient to walk or to stand. The first stage is not always present. Incoördination may be the earliest symptom, and I have known, in one or two cases, characteristic ataxy to be developed before the knee jerk was lost.

The course of the symptoms is extremely variable. The epithet "progressive" given to the malady by Duchenne expresses a characteristic of a large proportion of the cases in which ataxy is developed, and he knew of no other cases of the disease. The incoördination tends to increase, slowly or rapidly, until it reaches a considerable degree, and the sensory loss shows a like tendency. The pains fortunately do not always progress with the other symptoms. They are often most severe in the early period of the disease, and may either lessen or continue unchanged as the loss of sensation increases. But the power of recognizing the first stage of the disease, which we owe, especially, to Westphal's discovery of the loss of the knee jerk, has enlarged, and in enlarging has to some extent modified, our conception of the general tendency of the disease. It is exceedingly common for the first stage to remain stationary for a long time, for ten, twenty, and even, in one case, for twenty-five years, if the duration of the lightning pains may be accepted as proof of its existence. When all cases are taken together and patients are subjected to careful treatment, I do not think that the disease shows a progressive tendency in more than one-half of the cases.

The various symptoms do not always progress *pari passu*. Sensibility may fail although incoördination does not increase. Conversely, the ataxy may become greater although sensation remains the same, and even when it shows a distinct improvement. Thus a patient had slight ataxy, loss to pain on thighs, legs, and trunk, and no loss to touch or temperature. At the end of a year and a half pain was perceived on the thighs, and touch and temperature were still normal, although the ataxy had increased considerably. So marked indeed is the contrast between the course of different symptoms in some cases that it suggests a certain alternative tendency. If optic nerve atrophy develops, the spinal symptoms, in most cases, remain stationary. So, too, of visceral crises. I have known gastric crises to cease, when the incoordination began to develop. In one patient all the symptoms of tabes passed away, the knee jerk returned, optic nerve atrophy ceased to advance, but the patient became insane.

When the disease progresses, the rate of its advance varies much. Sometimes it is slow and uniform, and the ataxy is considered only at the end of several years. On the other hand it may be rapid, so that in a few months the patient is scarcely able to walk. More commonly the progress is varied; periods in which the disease is almost stationary alternate with others in which the symptoms increase rapidly. The exacerbations may seem spontaneous, or may be distinctly excited by some prejudicial influences,—a chill, a fall, or some excess, alcoholic or sexual. They are sometimes very acute; a marked change may occur in the course of a few days, or even in a few hours. Thus I have known a patient to pass, in the course of twenty-four hours, from a condition in which he could walk fairly well to one in which he could scarcely stand. Muscular power may lessen in such a sudden exacerbation, or the increase may be confined to the true symptoms of the disease. In the former case it is probable that there is a myelitic or neuritic complication; in the latter there seems to be merely an acute process developed in the nerve elements in which the chronic disease was in progress. It is often accompanied by acute sensory disturbance. One patient felt for weeks as if his feet were of enormous size, as large as the room he was in. The conditions thus developed may pass away again, leaving the patient on a little lower level than before, or they may persist without any considerable recovery.

There is nothing in the nature of the disease, in most cases, to produce death. The only direct effect of the malady, which has ended life, is laryngeal spasm or paralysis. Even the gastric symptoms, prostrating as they are, are never fatal. Patients who have reached the third stage have lived, bedridden, for twenty years. Death often results from intercurrent maladies, some of which are more dangerous to tabetics than to others, because they may develop painlessly and attain a serious degree before their existence is suspected. But many patients die from indirect results of the disease. Kidney complications are the most common; they often develop most insidiously, and then, as it were, explode. The patient may be seized with acute febrile symptoms, the exact nature of which may be unsuspected until the urine is examined. I have known a patient, still in the first stage, die thus after a few days' illness. Bedsores and pyæmia occasionally cause death, although less frequently than in some other diseases of the spinal cord.

Complications.—Occurring, as most cases do, in syphilitic subjects, tabes is sometimes complicated by true syphilitic lesions of the brain or cord. Some of the cranial nerve palsies that are observed in the early stage of the disease are of this nature.

Acute or subacute myelitis may occur during the course of the disease as well as before its development. In such cases rapid loss of power, with other symptoms, indicates the occurrence of an acute process involving other elements of the cord than those on which the special symptoms of tabes depend. Such myelitis is probably also, in some cases, of syphilitic origin. Sometimes it is apparently simple; it may develop after exposure to some exciting cause, as in the case already mentioned, in which, after the patient has suffered for four years from gastric crises and pain, complete paraplegia developed a few days after he had sat for some time on wet grass. Such a complication must not be confounded with the simple acute exacerbation in the special symptoms already described.

Other system diseases of the cord may develop as complications of tabes. The lateral columns may degenerate with the posterior, giving rise to "ataxic paraplegia," but this is rather a distinct affection than a complication of true tabes, and will be so described. Weakness may, however, supervene slowly in the course of locomotor ataxy, without muscular atrophy, and with a persistence of the special symptoms of the disease, from a later extension of the morbid process to the lateral columns. Another occasional complication is progressive muscular atrophy. We have already seen that local atrophy of muscles is not very rare in the later stages of tabes, but in the cases now under consideration, muscular atrophy develops as in its primary form, and runs an independent course, such as it pursues when it exists alone. Thus a gentleman, who had had hemiplegia from syphilitic vascular disease, developed the first stage of tabes—pains, loss of knee jerk, loss of the iris reflex, and slight unsteadiness. He had two courses of treatment at Aix-la-Chapelle, and at the end of the second, by which he was much prostrated, the muscles of the hands began to waste. The atrophy spread, and in the course of two years almost all the muscles of the arms, shoulders, and back became extremely wasted. In rare cases the legs present the symptoms of tabes, while in the arms there is a jerky incoördination of disseminated sclerosis, and the coexistence of the two diseases has been demonstrated post-mortem.

Another very important and frequent complication of tabes is general paralysis of the insane. The two diseases have many alliances. It is probable that syphilis predisposes to general paralysis as well as to tabes. Reflex iridoplegia is common in both diseases. The two maladies are often combined, and the symptoms of one or the other may preponderate. Thus many general paralytics present symptoms of tabes,

Fig. 111.

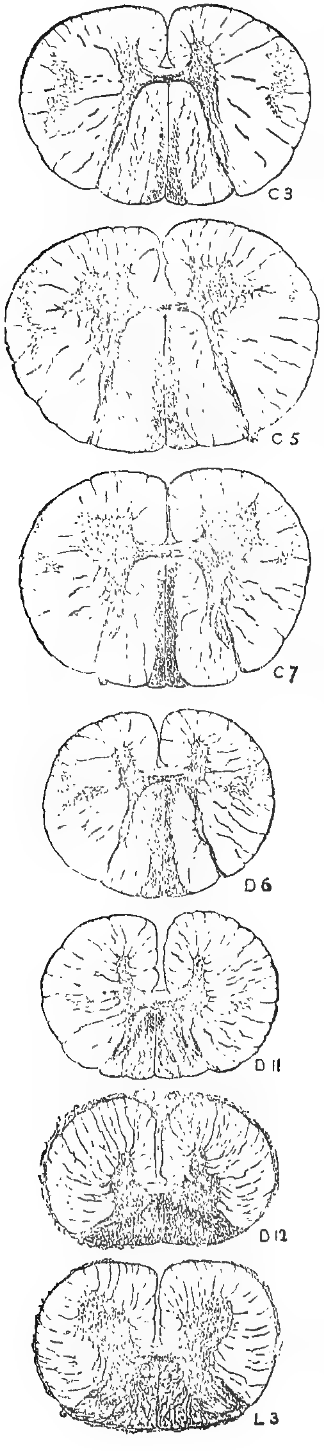


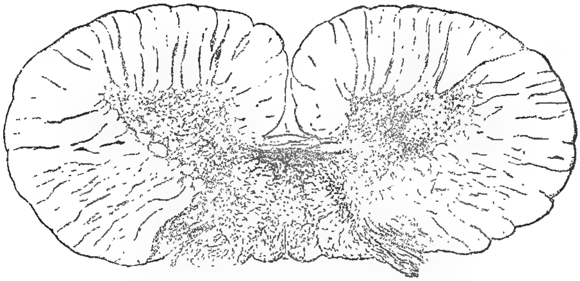
Fig. 111.—Locomotor ataxy involving the legs only. Sclerosis of the whole posterior columns in the lumbar region, gradually becoming limited, in the lower dorsal region, to the root zone and posterior median column. The latter is only affected in the upper half of the cord (ascending degeneration). There is also ascending degeneration in the antero-lateral ascending tract. This is rather greater on the left side, while the post-median degeneration is greater on the right, in harmony with the theory put forward on p. 132, that the latter contains fibres which do not decussate.

and its characteristic lesion is found after death. On the other hand, cases of tabes may present slight symptoms of general paralysis, perhaps only slight optimism and mental weakness, which may remain subordinate or may increase to a pronounced and preponderant degree. It may be difficult to say in which category a case should be placed. It is sometimes said that a case may commence as ataxy, and may change to general paralysis, but a more correct expression of the facts is the coexistence of the two affections, and the dominance of the symptoms of one or the other.

Of complications of tabes outside the nervous system, valvular heart disease is the most important, and usually develops without any of the usual antecedents of heart disease, and at an age at which degeneration is unlikely. Aortic regurgitation is especially frequent. It is not improbable that the cardiac and neural diseases own a common cause—syphilis, and that the valvular lesion is due to the syphilitic process that is known sometimes to attack the cardiac and especially the aortic valves.

Pathological Anatomy.—In most cases the spinal cord presents changes visible to the naked eye. The posterior columns have a gray translucent appearance, which is due to the loss of the white myelin of the nerve fibres, and to an increase of the connective tissue ("gray degeneration"). The whole of the posterior columns may be thus changed, or only parts of them, the distribution of the visible alteration being that of the histological changes revealed by the microscope and presently to be described. In the cord hardened by bichromate of potash or chromic acid, the difference of tint in the diseased areas is even more distinct than it is in the fresh organ. The posterior columns are also smaller than normal, because the bulk of the connective tissue is less than that of the normal fibres; hence the shape of the cord is slightly changed.

Fig. 112.



Tabes with ataxy of the arms as well as the legs. Section of cord in cervical region, showing sclerosis of the post.-med. column and root zone of post.-ext. col. Degeneration of left ant. cornu.

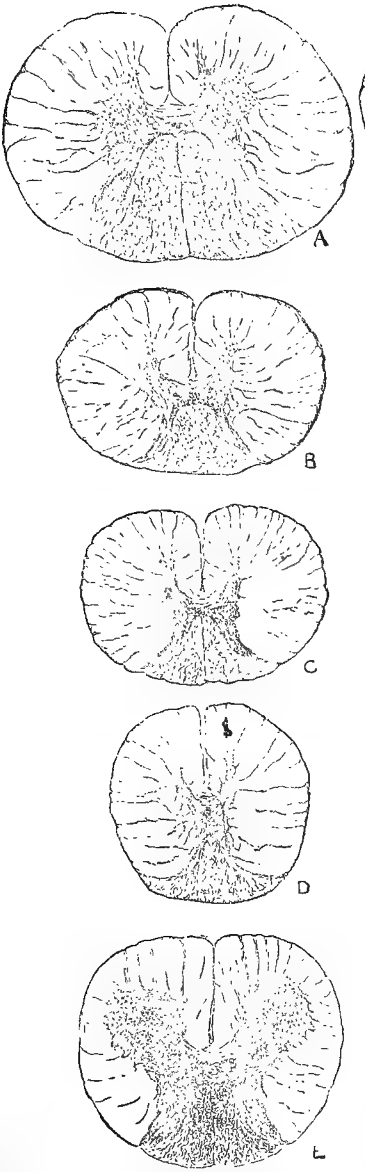
In a section of the cord stained with carmine or other agent, and examined under the microscope, the affected areas are conspicuous by the deep staining of the connective tissue. The position of the excess of this tissue, of the "sclerosis," as it is termed, indicates the place in which the nerve fibres have degenerated. In the most frequent condition, in which the legs only are affected, the sclerosis occupies the whole of the columns in the lumbar region (Fig. 111), but it is often slight in the anterior parts of the postero-external columns, which may even be free from sclerosis, and in the middle of these columns many normal nerve fibres may be mingled with tracts of connective tissue. The sclerosis is most dense in the part adjacent to the posterior cornu, through which the posterior root fibres run, and near the surface of the cord. Above the lumbar enlargement the affection of the postero-external columns gradually ceases, but the degeneration is intense in the postero-median columns, and has the distribution of an ascending degeneration, as it in fact is, receding, in the upper cervical region, from the commissure. (Compare Fig. 111 with Fig. 67, p. 131.) In other cases the external band of sclerosis, adjacent to the cornu, extends up through the dorsal cord and even through the cervical enlargement, blending with the median tract at the periphery (Fig. 112). The median degeneration then extends up to the commissure throughout the cervical region, just as does the secondary degeneration that results from a cervical lesion (see Fig. 69, p. 131). But in the cervical enlargement the unaffected area in the anterior part of the postero-external column is usually much larger than in the lumbar enlargement.

In rare cases, of severe degree and long duration, the posterior columns are occupied by connective tissue in their whole extent from one end of the cord to the other. A few fibres of the posterior roots and a few vertical fibres near the commissure may alone be recognizable. An instance of such complete sclerosis is shown in Fig. 113.

On the other hand, in slight cases, in which the disease is still in the first, or in the commencement of the second stage, the sclerosis is moderate in degree even in the regions of the cord most affected. The separation of the sclerosed areas in the postero-external and postero-median columns may be distinct throughout the cord, except in the lumbar enlargement, where the median degeneration expands into the external column. The sclerosis of the postero-external column is then usually limited to the root zone, and varies in width, according to the width of the area of the column through which the root fibres pass. In the dorsal region there is usually a narrow band of sclerosis close to the posterior cornu; in the lumbar region it reaches almost to the median septum. The anterior part of the post.-external column is often free from disease. In some instances, however, a very slight degree of sclerosis extends from the most affected tracts through the rest of the columns and is generally greatest in their posterior half. This condition existed in the very instructive case shown in Fig. 114. The intensification of this diffuse sclerosis in the root zone and median columns is very distinct, although the affection of the latter is unusually slight. There is, moreover, in the dorsal region, sclerosis of another part of the postero-external column—the comma-shaped tract of short fibres, which degenerates downward for a few inches below a transverse lesion of the cord (see p. 133 and Figs. 69 and 98, F). It is most distinct at D, 8, but can be traced up to the cervical region, where its form is changed.

The antero-lateral columns may be perfectly normal, even when the dis-

Fig. 113.



Locomotor ataxy: extreme incoördination and anæsthesia in both arms and legs; the posterior columns are sclerosed throughout the cord in their entire extent. A, upper cervical, B, C, D, dorsal, E, lumbar regions. In D there is also some degeneration of the intermediate gray substance.

Fig. 114.

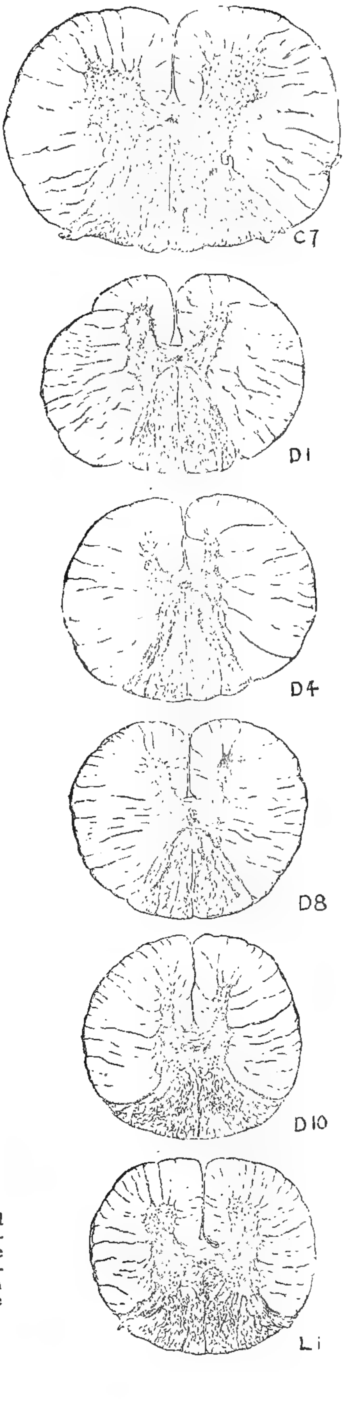


Fig. 114.—Loco. ataxy. Sclerosis of posterior columns. In the lumbar section the posterior portion of both p. m. c. and p. e. c. is densely sclerosed, but in front the disease is limited to the post.-med. col. At the tenth dorsal the sclerosis is also general, but is dense only in the root zone of the post.-ext. col. At the eighth and fourth dorsal the change is slight except in three areas, the root zone, the middle part of the post.-med. col., and the comma-shaped tract in the front of the post.-ext. col. At the first dorsal the comma-shaped tract is narrow, and in the lower cervical it merges in a band of sclerosis, which bounds the anterior part of the post.-med. col. There is a little diffuse sclerosis through the rest of the column, and in the first dorsal there is some degeneration of the anterior cornua. Symptoms of the first stage of tabes existed for four years (lightning pains, slight analgesia, loss of knee jerk, loss of iris reflex, gastric crises, optic nerve atrophy). A few weeks before death the legs gradually became paralyzed, and the arms ataxic; retention of urine caused acute kidney mischief, which was the immediate cause of death. No lesion was found after death to explain the subacute paraplegia.

ease of the posterior columns is great. Often, however, in old cases, there is a slight general increase in the connective tissue throughout the cord. Sometimes there is distinct sclerosis of other definite tracts. The ascending antero-lateral tract may be degenerated above the lower dorsal region, below which it seems not to exist; the fibres probably pass to it from the posterior commissure in the lower dorsal region. An example of this degeneration is shown in Fig. 111. The direct cerebellar tract is also occasionally diseased, but certainly less frequently than has been alleged. In rare cases there is a distinct degeneration of the pyramidal tracts. The peripheral layer of the antero-lateral column is occasionally the seat of sclerosis, the connective tissue extending in from the pia mater; this is usually also thickened, apparently from chronic inflammation, which will be again referred to.

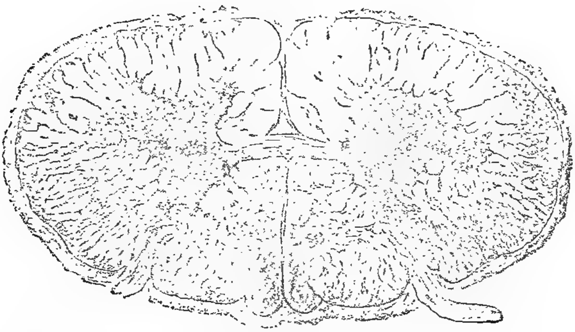
In slight cases it is difficult to distinguish any change in the gray matter of the cord, although it is probable that there is an atrophy of some of the nerve cells and fibres in the posterior horn. In more advanced cases such atrophy is constant, as Lockhart Clarke first showed. The posterior commissure may also be smaller than normal. The posterior vesicular column (of Clarke) shows distinct degeneration in many cases. Some atrophy of the plexus of fine fibres contained in these columns is said to be almost invariable (Lissauer). In other cases the change is greater, the cells and vertical fibres waste, and only a few shrunken cells scattered through a translucent nucleated tissue may remain. The atrophy of the cells is said to be associated with sclerosis of the direct cerebellar tract. When the cells are unaffected, although the intervening substance is diseased, the cerebellar tract may be normal, and the fibres passing to it from the front of the vesicular tract may be unaffected. The degeneration of the gray matter often extends into the intermediate region between the cornua (Fig. 113, B); the intermediate lateral tract of cells may be atrophied, and occasionally even the anterior cornu and its cells suffer in a similar way (Figs. 112, 114, D, I). This change is met with in the cases in which there is secondary muscular atrophy.

The sclerosis varies considerably in its histological characters. In slight cases, there is merely a moderate thickening of the trabeculæ and their branches. The larger tracts are more fibrous than normal, and more nuclei are seen than in health. Everywhere the new growth of connective tissue proceeds chiefly from the preëxisting tracts, trabeculæ, vessel walls, etc. When the change is considerable, the trabeculæ are very broad, and areas seem to consist wholly of connective tissue, fibrillated and nucleated. In old cases there is sometimes little appearance of fibrillation in the most affected region; cells and thickened vessels lie in granular or homogeneous tissue. From the parts most affected, the nerve fibres have wholly disappeared. In other parts, however, close examination reveals many fibres narrower than normal, and many points that may possibly be axis cylinders. The walls of the vessels are usually increased in thickness, sometimes to a very considerable degree. The coat next the endothelium may be thickened and contain many nuclei, but the chief thickening, in most cases, is in the outer coat and adventitia. The vessels are most affected near the posterior median septum, the posterior surface, and the gray matter. From the thickened wall trabeculæ pass off into the adjacent tissue. The pia mater is sometimes thickened over the posterior columns; the vessels in it may be similarly changed. The alteration in the membrane may extend over the neighboring part of the lateral column, and even around the cord (Fig. 115), and, as already stated, septa passing from it into the cord may be thickened in the circumferential zone. In cases of rapid course, products of degeneration, masses of myelin and granule corpuscles, may be found in the affected parts.

In rare cases other lesions are found in the cord, corresponding to the complications already described. The lateral columns may be sclerosed apart from any thickening of the membranes. Diffuse myelitis may be met with, and, occasionally, characteristic insular sclerosis has been found in the cervical region with posterior sclerosis in the lumbar.

The posterior nerve roots may appear normal in slight cases; it must be remembered that only a considerable degree of disease can be detected even by the microscope. Often their disease is conspicuous; when the cord is much affected they are invariably atrophied, gray and thin to the naked eye, and the microscope shows wasting of the nerve fibres and slight increase of connective tissue. The change may extend down to the ganglia, where it ceases. The ganglia themselves are almost always normal in structure, and the mixed nerve beyond them is free usually from degeneration. The anterior nerve roots are altogether normal, except in the rare cases in which the anterior cornua have suffered, and then some of their fibres may be degenerated. In this case these atrophied fibres may be traced also in the mixed nerve trunk beyond the ganglion.

FIG. 115.



Tabes. Chronic meningitis. Cervical region. Sclerosis of post.-median columns and slight diffuse sclerosis of the cord, least in ant. cols., dense in the right lateral column. Thickened pia mater, and damage to subjacent superficial layer of the cord.

The peripheral spinal nerves have been recently found to undergo atrophy in a large number of cases of tabes.* The change consists in a wasting of the nerve fibres, beginning in the white substance, which may be reduced to a very narrow layer. Ultimately the axis cylinders perish. There may be a slight increase in the interstitial tissue and nuclei, but the change seems to commence in the nerve fibres themselves. The degeneration is greatest in the cutaneous nerve filaments, and lessens in degree as the nerves are examined higher up. In nerves one or two millimetres thick it is still considerable, although there are more healthy fibres than in the peripheral twigs. In larger nerve trunks it gradually ceases, and near the spine the nerves are always healthy. The sensory fibres seem to be exclusively affected. The lesion has hitherto been found chiefly in the sensory nerves that supply the skin and joints, but that the sensory nerves of muscle undergo a similar change has been ascertained by Déjerine. The peripheral degeneration is found most commonly in the legs, but is met with also in the arms when these are the seat of symptoms. The change in the nerves bears no relation to that in the spinal cord in degree, or even in existence, for extreme alterations have been found in the nerves when the cord is quite normal. On the other hand, in some cases, the nerves have been examined and found healthy.

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* The first observation was made by Westphal, but the extent and significance of the

The optic nerves when atrophied present wasting of the nerve fibres, and very often a remarkable increase in the interstitial connective tissue, which forms thick tracts, of gelatinous aspect, between the bundles of nerve fibres. Degeneration of the ascending root of the fifth nerve has been found (first by Westphal in 1864) in cases in which symptoms were present in the area of its distribution. Sclerosis of the third nerve has also been found.

Pierret has described, as common in cases with vasomotor and visceral symptoms, a degeneration of the intermedio-lateral tract and adjacent nerve fibres in the upper part of the cord, traceable up the medulla oblongata in the region of the so-called "slender column," adjacent to the accessory, glossopharyngeal, and pneumogastric nuclei from which it is supposed that the chief visceral and vasomotor influences are exerted. The sympathetic has been examined in several cases, and found healthy, with one exception, in which the sympathetic on the left side of the neck was degenerated in association with left exophthalmos.

In the cases in which arthritic symptoms have been present during life, or fractures have occurred, conspicuous changes are found after death. There is occasionally an extraordinary wasting of the articular ends of the bones, which seems to begin by rapid erosion of the cartilages, extending quickly to the bone beyond, the extremity of which may be much reduced in size and altered in form. In the hip joint, for instance, in a case recorded by Charcot, the edges of the acetabulum had been removed, and the whole of the head of the femur and most of the trochanter had disappeared (Fig. 116).

Similar changes have been found in most of the larger joints. In the bones, the compact tissue has been found thinner and more porous than

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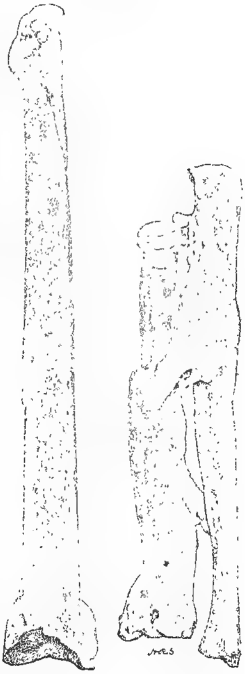
change in the nerves was pointed out by Pierret (1880), and his observation was soon after confirmed and extended by Déjerine and Pitres.

normal. At the seat of old fractures a large amount of bony callus is formed (Fig. 116), and sometimes new bone is found in the capsule of joints that have been injured.

PATHOLOGY.—The great fact of the pathology of tabes is that it is a neural degeneration in the sensory nervous system, peripheral and central. In both peripheral nerves and spinal cord, the incidence of the disease is almost exclusively on sensory structures, and the changes seem to begin in the nerve elements themselves. The cerebral system suffers in far less degree than the spinal, and in more irregular form, but in the occasional peripheral degeneration of the optic nerve and the central changes in the roots of the fifth we may trace the same law of distribution. It is less dominant, however, than in the spinal system, since some of the cranial nerve symptoms are exclusively motor. The motor character of the leading symptom, ataxy, is, as we shall see, only an apparent and not a real exception to the general sensory character of the malady.

FIG. 116.



Osseous lesions in tabes. (*After Charcot*) A. Atrophy of the head of the femur. B. Excessive formation of callus after so-called "spontaneous" fracture of the ulna and radius.

The degeneration, it has just been said, must be regarded as commencing in the nerve elements themselves; the overgrowth of connective tissue, which gives to the lesion its obtrusive character, is secondary. That this is true of the spinal cord lesion has, indeed, been doubted, chiefly on the ground of the thickening of the walls of the vessels seen in some cases, and the manner in which the increase of tissue seems to start from them. But the "system" character of a disease, the limitation of a widespread lesion in its early stage or slight degree to structures having a common function, is probably in all cases proof of its neural origin, *i. e.*, its origin in the nerve elements themselves. In tabes the evidence thus afforded is corroborated by the disease of the peripheral nerves, which unquestionably commences in the nerve fibres, and by the partial character of the sensory loss, which means the limitation of the chief lesion to fibres of one sensory function. There is every gradation, moreover, between the cases in which the vessels of the cord suffer much, and those in which they are unchanged, and in which there is nothing even to suggest an initial interstitial process. The change in the vessels may reach a high degree in the posterior median columns when their degeneration is secondary, and certainly commences in the nerve fibres; a similar thickening of the vessels has also been met with in some other instances of purely secondary degeneration. We must, moreover, remember that the overgrowth of the neuroglia is a pathological process of growth distinguishable from the degeneration of the fibres that excites it. It may vary in degree in different cases, and does so conspicuously in secondary degeneration of peripheral nerves, in which it may assume an inflammatory character independent of any propagated inflammation from the primary lesion. Moreover the growth of new is mainly from old tissue, and the chief new tracts are clearly determined in position by the preëxisting tissue. The neighborhood of vessels seems the place in which connective tissue grows most readily. It is from the trunk that the twigs of a pollard willow spring, but they would not grow there had not the branches been lopped off. Once excited, the energy of tissue growth may be in some degree independent of its cause. Some vascular disturbance may attend it, and thus a subinflammatory condition is probably sometimes developed, which may invade the pia mater, and through this may spread widely. Whether, in some cases, a constitutional state, disposing to tissue overgrowth and tissue inflammation (as syphilis perhaps does in certain stages), may determine the degree of the interstitial change, is a problem of much interest, which does not, however, affect the question which has been just discussed.

The relation of the disease to syphilis is as obscure in nature as it is certain in fact. There is nothing in the histological character of the lesion that can be compared with the morbid processes that are recognized as certainly syphilitic. There is nothing, therefore, to warrant the opinion that any morbid germs deposited in the cord or nerves lead to the development of the disease. It is probable that syphilis leaves behind it a predisposition to nerve degeneration, lowers, in certain nerve elements, the capacity for resisting morbid influences, leaves many patients in a condition analogous to that of one who inherits a predisposition to neural diseases. Thus we can understand the length of time that often intervenes between the syphilis and the first symptoms of tabes, the coöperation of other causes, and likewise the fact that other degenerative diseases, besides locomotor ataxy, sometimes occur in those who have had constitutional syphilis.

The pathological facts already known afford an adequate and easy explanation of many of the symptoms of the disease. There may be an interruption of the sensory path in one or both of two places, in the peripheral nerves, and in the posterior root fibres as they enter the cord, and an interruption of the fibres in either place will explain the loss of sensibility which is so frequently present. The pains may reasonably be ascribed to the molecular changes of the process of degeneration in the nerve fibres, either in the periphery or in the cord. The intensity of a sensation is no measure of that of the change that causes it. Whether the affection of the sensory cells of the cord takes part in the production of the pain we cannot tell; such a mechanism is not improbable, since it is certain that degeneration occurs in the posterior horns, and the affection of the antero-lateral ascending tract may be taken as evidence of the degeneration of the cells in which some sensory fibres from the skin terminate.* The loss of reflex action from the skin is explained by the interruption of the sensory path, and an increased irritability of the sensory nerves, the result of the commencing degenerative changes, sufficiently explains both the hyperæsthesia and the increase of reflex action sometimes observed (see p. 157). Whatever theory of the nature of the so-called "tendon reflex" action is held, the loss of the knee jerk must be explained by an interruption of the sensory path. On the theory I have advanced, the arrest of impressions from the sensory muscular nerves abolishes the muscle reflex action on which the local irritability depends. Many other facts of disease show that this irritability is easily lost, and that a very slight change in any part of the reflex arc, too slight to cause other symptoms, is sufficient to arrest the knee jerk.* Hence we can understand that this loss should be constant and early. It is not improbable, moreover, that these nerves suffer in special degree. The muscles may be insensitive to pain as of electrical stimulation. Pressure and extension, which in health are painful, sometimes cause no sensation, even when the skin is sensitive.

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* In disease of the nerve roots of the cauda equina there is always ascending degeneration of the posterior median columns, but not of the antero-lateral ascending tract. I have

The mechanism of the muscular incoördination, which is the obtrusive symptom of the disease, has been the subject of much discussion. Two fundamental facts, however, limit the problem. First, the ataxy cannot be primarily due to the loss of cutaneous sensibility. Disease of the conducting path in the cord may cause absolute anæsthesia of the skin without the least ataxy. Although this does not prove that interruption of the sensory path in the nerves, between the skin and the reflex centres, may not cause incoördination, this element seems to be excluded by the fact that there is no relation between the ataxy and the loss of feeling in the skin. There may be, in tabes, much ataxy without any cutaneous anæsthesia, and *vice versâ*. The second fact is that ataxy may exist in considerable degree when the lesion is solely one of the peripheral nerves, and the posterior columns of the cord are free from disease.† These two facts, taken together, seem to show that the ataxy may be produced by one mechanism, even operating alone, the disease of the sensory muscle nerves. If the loss of the knee jerk in tabes is to be taken, as I believe it may, as an indication of the disease of these nerves, the constancy of the loss shows the constancy of the presence of this element in some degree. As just stated, a very slight degree of disease may abolish the knee jerk; it is probable that a considerable degree is needed to cause incoördination.

In unilateral lesions of the spinal cord, as we have already seen (p. 166),

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recently had an opportunity of ascertaining this. Hence the root fibres from which the path is continued by this tract must end in nerve cells in the posterior cornua, and the degeneration of this tract in tabes is proof of the degeneration of these sensory cells. See the *Lancet*, June 19, 1886.

* *e. g.* its loss after diphtheria when there are no other symptoms.

† Apart from the evidence of this from cases of true tabes, a very instructive case has been recently recorded by Dr. Hughes Bennett, in which all the symptoms of tabes were present in a case of multiple tumors of the posterior nerve roots (*Clinical Soc. Transactions*, vol. xviii).

there may be loss of the sense of posture, with intact cutaneous sensibility, on the side of the lesion, and no loss of this sense on the opposite side, on which cutaneous sensibility is lost. In such a case marked ataxy has been observed on the side on which the sense of posture was lost, when motor power returned.* Whatever effect in causing ataxy is produced by disease of the path of muscular sensibility in the spinal cord must also be produced by interruption of the path between the muscles and the cord. The latter must also arrest whatever reflex action depends upon these muscle nerves,† and it is possible that such reflex action takes some part in the mechanism of muscular incoördination.

In this connection it is important to note that the fibres that pass up by the posterior median columns probably constitute the path from these nerves. The root fibres to the columns seem to pass up, as the path is proved to do, without decussating, and they may be diseased in intense degree when there is no loss of cutaneous sensibility. This tract is affected in almost all cases of tabes in which there is a lesion in the cord, and in all such cases in which there is ataxy. This fact suggests that the lesion of the root fibres is first and chiefly of those from the muscles.‡

The posterior median columns (extensively connected with the cerebellum) and the direct cerebellar tract probably conduct to the cerebellum the impressions from the sensory muscle nerves. If so, the disease of these tracts and nerves must interfere with the coördinating action of the cerebellum and cause some part of the loss of function which results from the disease of the cerebellum itself.

There is another mechanism which may coöperate in increasing ataxy. The vertical fibres of the postero-external column have only a short course, and some probably connect the posterior gray matter at adjacent levels. The affection of these fibres seems to be proved by the interesting fact already mentioned (Fig. 114) that the comma-shaped bundle of fibres in the front of the post.-external column may be specially degenerated. This lesion may impair the association of the sensory structures, and so cause a want of harmony in the central mechanism. The theory that the ataxy was due to this cause was advanced by Todd in 1847. It is not probable that, if effective, it has more than an intensifying influence, since ataxy may exist when the posterior columns are throughout unaffected.

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* Gilbert; see footnote, p. 166.

† In a case in which the incoördination was much greater in the right leg than in the left, and cutaneous sensibility was equally impaired in the two, a strong traction on the calf muscles, by forcible passive flexion of the foot, produced a distinct sensation in the extended muscles of the left leg, and no sensation in those of the right. In another case, in which the skin was sensitive to the slightest touch, the patient was unconscious of a vigorous extension of the toes produced by faradaic stimulation of their short extensor. This tract is most diseased on the side opposite to that on which there is most degeneration of the antero-lateral ascending tract, which probably conducts sensations of pain.

‡ The opinion that the ataxy depends on impairment of the muscular sense was put forward in 1855 by Dr. Russell Reynolds; see p. 286. Déjerine has also associated the ataxy with the degeneration of the peripheral sensory muscle nerves (*Archives de Physiologie*, 3d series, vol. iii, p. 231).

Although cutaneous anæsthesia cannot, alone, produce ataxy, it may reasonably be assumed to increase that which already exists. Sensations from the skin furnish important guidance to the motor cerebral centres, and are probably also concerned in such reflex muscular actions as that of standing. Similar guidance to the cerebral coördinating centres is also afforded by visual impressions, the loss of which distinctly augments the defect of coördination.

The varying characters of the ataxy in different cases probably depend on the local variations in the degree of change in the muscle nerves. If, as Flechsig believes, the direct cerebellar tract conducts impressions from the trunk muscles, its disease may produce the trunk ataxy described on p. 291.*

It is thus probable that coördination is chiefly an automatic process, depending partly perhaps on muscle reflex actions, and on the connection of neighboring sensory structures in the spinal cord, but chiefly on the function of the cerebellum itself, and the connection of the muscles with it, and that the interruption of this connection is the chief element in the incoördination of locomotor ataxy. It is probable, also, that the automatic processes are in part under cerebral control, guided by sensory impressions which do enter the sphere of consciousness, and that the derangement of this control will intensify incoördination, though incapable of producing it. We do not at present know to what extent, in any given case, the symptoms are due to the cord disease or to the peripheral nerve lesion. Apparently pains, ataxy, and anæsthesia may be due to either. The question can only be decided by the comparison of symptoms and pathological changes in a large number of cases. That anæsthesia may be due to the peripheral changes is proved by the observed correspondence of the two in distribution (Déjerine).

The trophic changes in the skin, bones, and joints are probably due to the process of degeneration in the peripheral nerves. The degeneration has been found in all cases of the kind in which it has been looked for, and found also in the nerves of diseased joints.

The pains in the region of the fifth nerve are explained by the lesions in its root, especially in its ascending root, which, coming up from the medulla, is homologous with the posterior spinal roots. This nerve, indeed, as Pierret has pointed out, represents the sensory roots of almost all the motor cranial nerves. The degeneration of the optic nerve is fairly comparable with that of the peripheral spinal nerves. Considering the special character of the optic nerve the absence of degeneration of the retina does not seem to destroy the analogy between the two. Regarding the pathology of the visceral crises we know but little. The changes observed by Pierret in the neighborhood of the centre for the pneumogastric may be indications of the cause of the disturbances that occur within the range of this nerve.

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* As already mentioned, the fibres in the posterior vesicular columns may be degenerated when the cells suffer but little, and the fibres from this column to the direct cerebellar tract, and this tract itself, are unaffected. Hence the function subserved by this tract may be lost, from the peripheral degeneration of the fibres to the vesicular column, when the tract itself is not diseased.

The transient motor symptoms in the limbs and in the eyeball muscles are apparently of functional origin. They must be distinguished from the lasting palsies which are probably due to degenerative processes. Sclerosis of the third nerve has been observed, and the loss of function of the internal ocular muscles must be ascribed to degeneration of the related centres in the nucleus of the third nerve. In the account of the diseases of this nerve they are more fully described.

Diagnosis.—The diagnosis of tabes rests on the combination of symptoms already described. In the early stage, the loss of the knee jerk, together with pains, or unsteadiness on standing with the feet together and the eyes closed, justifies a diagnosis of the commencing affection, provided we can exclude diabetes, diphtheritic palsy, and also a lesion of the anterior cornua or nerve roots by the absence of wasting of the muscles and change in their irritability. The diagnostic value of the loss of the knee jerk can hardly be overrated. It is probably never absent in health. If there is doubt as to its loss, the precautions recommended on p. 36 should be adopted. When it is lost and reflex action is in excess, a true reflex movement may sometimes simulate the jerk; the distinction is that the tap sometimes causes a movement and sometimes does not; that an interval, brief but appreciable, elapses before the movement occurs; and that a similar movement is caused by a prick on the skin. In cases in which the knee jerk is present, the diagnosis of tabes is only justified by distinct and characteristic incoördination. In the few cases of this kind that have come under my own observation, the knee jerk has been either unequal on the two sides, or has been lost on one. Such abnormality is probably the rule in these cases, and the diagnosis is thus facilitated. In a case with lightning pains, but neither incoördination nor loss of knee jerk, a suspicion of tabes would only be justified by the presence of some other symptom, such as retention or incontinence of urine, loss of sexual power, or loss of the iris reflex.

The last-named sign is of great diagnostic importance. Its significance is that a degenerative process is at work in the nervous system, and it suggests, therefore, that other symptoms are also due to degeneration. But since the iris reflex is not always lost in tabes, the negative significance of a normal reflex is far less than is the positive significance of its loss. The practical value of this symptom can hardly be overrated. It puts the observer, so to speak, on the track of nerve degeneration.

There are certain diseases with which tabes is especially liable to be confounded. One of these is multiple alcoholic neuritis. The ordinary form of this disease is readily distinguished by the symmetrical paralysis which is its chief manifestation. In the rare variety of alcoholic nerve degeneration which closely resembles locomotor ataxy in its symptoms, "alcoholic pseudotabes" (p. 116), the diagnosis may be very difficult. The disease, moreover, resembles tabes pathologically, since the lesion is a "parenchymatous neuritis," subacute degeneration, beginning in the nerve fibres. It often affects, however, in some degree the motor as well as the sensory nerves, and there is then some weakness in the distal portions of the limbs. An altered electrical reaction may be found in the muscles, and there is generally marked muscular tenderness, scarcely ever present in tabes. The pupils act normally, but this is not an absolute distinction, since they may be unaffected in tabes. A history of alcoholism also helps the diagnosis. By attention to all these points, a diagnosis can generally be made without much difficulty. In rare cases, without motor symptoms, it may be impossible (see p. 117).

When the symptoms and lesion of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is rather one of the preponderance of the symptoms of one or the other malady than of absolute distinction between them. In most cases, however, in which this combination exists, the symptoms of general paralysis become more pronounced as time goes on, and the spinal symptoms, which at first were the most conspicuous, pass into the background.

All common forms of paraplegia are distinguished by the early loss of power, and by the persistence of the knee jerk, often emphasized by its excess and by the occurrence of a clonus in the rectus or the calf muscles. If weakness supervenes in tabes, it is usually late in the course of the disease, and other symptoms have been and are well marked. If the knee jerk has been once lost in this disease, it is not reproduced by secondary lesions of the cord which ordinarily increase the knee jerk. The same distinctions usually suffice for the diagnosis from the combined form of paralysis and incoördination that I have termed "ataxic paraplegia." In this there is always an excessive knee jerk and a foot clonus, pains and anæsthesia are absent, and spasm supersedes ataxy. The diagnosis between the two maladies can be better discussed when this disease has been described. In other forms of combined disease of the cord, those, for instance, in which the symptoms of tabes are associated with indications of multiple sclerosis or of progressive muscular atrophy, the diagnosis depends on the recognition of union rather than on the distinction of difference.

Acute lesions of the cord, probably situated in the postero-external column, may produce incoördination and pain, but the suddenness of the onset, the limitation of the symptoms, and their tendency to subside, usually suffice for the distinction. Symptoms from this cause are far more common in the arms than in the legs. Ataxy in one limb may be produced by a tumor growing in the posterior column, but other symptoms soon indicate the tendency of the disease to invade the other elements of the cord. All these cases present the characteristics of a focal lesion with random consequences, rather than of a system disease with limitation to special function.

In cases of diphtheritic paralysis in which the nature of the sore throat was not recognized, the loss of the knee jerk may cause tabes to be suspected, but weakness is usually distinct from the first. The danger of a mistake is greater in the rare cases in which incoördination is the chief symptom; and the persistence of ataxy may cause an impression that the malady is really tabes, even if its relation to diphtheria is recognized. The paralysis of accommodation and of the palate which almost always precedes the ataxy, and often passes away as the latter develops, afford sufficient proof of the nature of the spinal symptoms. Pains are absent or trifling and there is usually some weakness in addition to the ataxy. True tabes never follows diphtheria, and the mere sequence, therefore, affords proof of the nature of the case, and assurance that the symptoms will not be permanent, however long they persist.

In the cases in which the dorsal region of the cord is chiefly affected the nature of the disease is often not recognized. The severity of the pains in the trunk may lead to a suspicion of disease of the bones; but the two are distinguished by the special symptoms of each disease and also by the absence in tabes of the increase of pain when the patient moves, characteristic of bone disease, and by the fact that the tabetic pains are wide in distribution and variable in seat, while the pain of bone disease is local and constant. Such cases are sometimes also mistaken for neuralgia of the intercostal nerves, but even if there are no pains in the legs, the knee jerk is lost. A careful examination will generally reveal some loss of sensibility in the regions in which the pains are felt; and the light reflex of the iris, so commonly lost in tabes, is present in all functional diseases with which tabetic pains may be confounded.

The only organic intracranial disease the symptoms of which present any likeness to those of tabes is tumor of the cerebellum. In some cases of tabes the unsteadiness bears considerable resemblance to that of cerebellar origin. Loss of the knee jerk is strongly in favor of tabes; although, in some cases of cerebellar tumor, it is said to be lost, it is much more commonly increased. Anæsthesia and lightning pains are conclusive evidence of the spinal disorder, while in cerebellar tumor severe occipital headache is almost invariable, and considerable optic neuritis is extremely common. Optic nerve atrophy is only secondary to neuritis, and loss of the reflex action of the iris to loss of sight.

The various visceral crises are often mistaken for primary disorders of the deranged organs. Recurring attacks of the character described should always arouse suspicion of their possible tabetic nature, and an examination of the knee jerk will usually decide the point. Errors in diagnosis are usually due to ignorance of the occurrence of these visceral symptoms.

PROGNOSIS.—The prognosis in tabes corresponds with the facts stated as to the course of the disease. It is not, as was once thought, uniformly bad. Arrest is frequent, considerable improvement is not rare, but perfect recovery scarcely ever takes place. The earlier the stage of the disease, the better is the prognosis. In the first stage the chances are in favor of arrest. When ataxy is developed, the prospect of arrest is less than in the first stage, but is still considerable. One patient came under my care fourteen years ago with distinct incoördination, which is no greater now than it was then. Occasionally, even in the typical form of the disease, the amount of improvement is very great. One patient was scarcely able to walk across the room, in consequence of the incoördination, when he came under treatment, and at the end of six months his gait was scarcely distinguishable from that of a healthy person. When the third stage is reached the possibility of improvement is far less, but is not quite absent. A woman suffering from pure tabes, when admitted to hospital, had been unable even to stand for six months, on account of the extreme ataxy, but she improved so much that in a few months she was able to walk about the room without help, and has continued, now for six years, to do her household work without difficulty, and even to walk some miles with the aid of a stick. But in the cases that present most improvement all the symptoms of the disease scarcely ever disappear. I have only known in one case the knee jerk to return, and in this patient the disease had not passed the first stage, although the diagnosis was certain. It is difficult to find any trustworthy indications to guide the prognosis in an individual case. The fact of preceding syphilis influences the prognosis only in one condition, when the symptoms develop rapidly and the patient is seen soon after their onset, and especially when they occur within two years of infection. In some cases of this kind anti-syphilitic treatment does great good. Even in these, improvement cannot be confidently anticipated. I have seen some cases of this character in which no benefit resulted from the treatment. In most cases the only trustworthy guide to prognosis is the observed tendency of the individual case.

Of special symptoms, the pains are often the most distressing, and unfortunately they are the most obstinate symptom of the disease. They may persist in undiminished severity when other symptoms lessen, and on the other hand they may become trifling when the disease increases. Diminution of the pain is a good sign if other symptoms are stationary, but not if they increase. The optic nerve atrophy is usually progressive, but not so uniformly as is often asserted. In a few cases I have known its progress to be permanently arrested. Strange to say, the occurrence of optic nerve atrophy makes the prognosis as to the spinal symptoms better; in most cases of atrophy the spinal lesion remains in the first stage.

TREATMENT.—It is important, in every case, that all influences likely to depress the nervous system should be avoided. Excessive mental work and anxiety are most prejudicial. Physical fatigue is also harmful. In severe and acute cases, absolute rest for a week or two is often useful at the onset of treatment. If the patient walks about, exercise should always stop short of fatigue. Care should be taken to avoid the risk of falls; a severe concussion of the spine will sometimes excite to fresh activity a stationary disease. Exposure to cold is also injurious; a severe exposure may cause acute mischief. If practicable, it is well that the winter should be spent in a warm and dry climate. A sea voyage is frequently useful, combining, as it does, a maximum of fresh air with a minimum of exertion. If, however, there is much ataxy, the risk of falls from the motion of the ship must be taken into consideration; it is less than might be anticipated. A sea voyage is especially useful in cases that are already stationary; such patients often return with a diminution in all their symptoms. When the disease is advancing, the influence of a voyage is less frequently distinct. The digestive organs should be kept in careful order; an attack of indigestion or constipation is often attended by lightning pains, which cease when an aperient has acted. Food should therefore be light in quality and easily digestible. The change from an active to an inactive life, which tabes often causes, may lead to the development of gout in an individual predisposed to it, either by inheritance or by his previous mode of life, and the tendency must, as far as possible, be obviated by regulations of diet and by frequent aperients. Smoking should be either avoided or limited to a small amount. Alcoholic excess is especially harmful; it sometimes excites a very severe exacerbation of the symptoms, and this is not surprising, since, as we have seen, chronic alcoholism may cause a disease resembling tabes in its symptoms and pathology. Sexual excess is also most injurious. Many patients are preserved from this danger by the disease itself, but it is not always so. Excess seems to have a special influence on the optic nerve atrophy. Several very painful instances of this have come under my notice. A man in the early stage of tabes, with slight atrophy, some peripheral limitation of the fields, but little impairment of acuity of vision, started on a voyage from Australia to England. The day before he started he married, and the marriage had its common sequel. When he reached this country he was quite blind.

Drugs have certainly some influence on the disease, but it is rarely sufficient to check the morbid tendency when this is very active. Moreover, the influence of drugs is variable; that which seems to do much good in one case has no influence on another, and this is true also of the same case at different periods. Hence the therapeutic statements that have been made by various observers are somewhat discordant. The first question in the treatment of any disease that is related to syphilis, is the influence of anti-syphilitic agents. In tabes such influence is distinct only in rare cases, chiefly under the conditions mentioned in the section on prognosis. In those circumstances, full doses of iodide of potassium should be given, and, if the interval since the primary disease is short, mercury may be rubbed in until the gums are slightly affected. In cases of slow development, several years after primary syphilis, iodide seldom does good. Still, in rare cases, improvement follows its administration, and therefore (as well as to clear the therapeutic ground) it is desirable to let the patient take iodide of potassium, if he has not already done so, in doses not exceeding thirty grains a day, for six or eight weeks. Very small doses of mercury, such as $\frac{1}{16}$ grain of the red iodide, may also be given for a longer time, and may often be conveniently combined with other drugs. It seems sometimes to have a tonic influence, and perhaps may lessen the secondary subinflammatory process in the cord and pia mater, which, as we have seen, sometimes constitutes part of the morbid process. But in such late cases, and in all the late nerve degenerations that follow syphilis, energetic mercurial treatment only does harm. Its depressing influence often increases the degenerative tendency, and hastens the progress of the disease. I have seen several such cases in which an energetic course of mercurial treatment, at Aix-la-Chapelle for instance, had an effect which was simply disastrous.

Of drugs that have been recommended for tabes, those that are most useful, in the order of their value, according to my own experience, are arsenic, iron, quinine, nux vomica, nitrate of silver, Calabar bean, belladonna, ergot, phosphorus. Of these arsenic is certainly that which most frequently does distinct good. Most of the cases in which I have known the greatest improvement to occur have been taking it at the time. In several of these cases there was no other change in the conditions of life to which the improvement could be ascribed. The form in which arsenic is given matters little; ♏v or ♏x of the Liq. Arsenicalis may be given in mixture, or $\frac{1}{12}$ gr. of arseniate of soda in a pill. Iodide of arsenic has no special advantage, and is not so well borne. This or any other drug must be continued for at least two months before an opinion of its influence can be formed. Rapid improvement can never be expected in so chronic a disease. In connection with the influence of arsenic, it is interesting to note the well-known effect of the drug in improving the nutrition of the skin, and the frequency with which the cutaneous nerves are degenerated in tabes. Quinine, nux vomica, or iron may be conveniently combined with arsenic. Nitrate of silver is far less useful, but it may sometimes be given with advantage alternately with arsenic. It should not be given in larger quantity than half a grain a day, nor continued for a longer period at a time than two or three months. A grain a day has been said to cause fatal kidney disease, and I have seen a case in which a still smaller dose taken daily for a long time caused both wrist-drop and albuminuria.

Calabar bean has been recommended by Ringer as producing temporary improvement in many chronic spinal diseases. Its influence is not great, but it seems to do no harm. Ergot is rarely useful in chronic diseases of the cord, and it is difficult to understand the estimation in which it has been held in the treatment of tabes. It may perhaps have some influence on the dilated vessels that are to be seen in many cases, and may therefore be given in the cases in which spinal pain suggests congestion of the membranes, or in which an acute exacerbation of the symptoms, or their extension beyond the special tabetic limits, makes it probable that there is a secondary subinflammatory process. Some years ago I gave gold a careful trial, but without obtaining any satisfactory results.

Counter-irritation to the spine, by blisters or the actual cautery, is sometimes useful, chiefly in cases just mentioned, in which the disease develops rapidly, or the symptoms rapidly increase, or in which there is spinal pain or tenderness. It is not probable that it influences the process of degeneration, but it may lessen any secondary inflammation.

Of special symptoms that require treatment the pains are most important. Unfortunately they are often most obstinate, and their variability renders it very difficult to estimate the influence of treatment upon them. Severe paroxysms may yield only to hypodermic injections of morphia, but this should be reserved for the most intense attacks, since its use is undesirable for a recurring symptom which may continue for years. Among other sedatives, Indian hemp is often effective; gr. ¼ or ½ should be given every three or four hours during a paroxysm. Other sedatives are of little use. Hypodermic injections of cocain are uncertain in their influence on tabetic pains, but sometimes relieve the local pain that is accompanied with hyperæsthesia. The only external application that is of service is chloroform sprinkled on spongio-piline (or lint and oiled silk), but this relieves only the superficial pain. Occasionally the pains are lessened by a warm bath. Counter-irritation to the spine, as by repeated sinapisms, sometimes gives some relief, but probably only when the pain has a spinal origin. Visceral symptoms rarely yield to special treatment; when severe morphia alone mitigates their intensity. Slight gastric crises may be relieved by simultaneous sinapisms to the epigastrium and neck. Laryngeal spasm is usually at once relieved by nitrite of amyl; and the local application of cocain is also useful. For weakness of the bladder, nux vomica, or strychnia is the most useful. The latter may be given by hypodermic injection, as recommended in the chapter on muscular atrophy. Nocturnal passage of urine is often stopped by belladonna.

If blisters or sores form on the feet they should receive careful local treatment until they are well. If neglected they may become very troublesome. Tabetic patients should be cautioned never to cut a corn; perforating ulcer is often set up by a corn being cut too deeply. The epidermis should be softened with alkali and rubbed off with pumice stone.

Attention to the bladder is one of the most important elements in the treatment of tabes, as in that of all diseases of the spinal cord, chronic and acute. If there is any reason to believe that the bladder is imperfectly emptied, a cathether should be passed, and if residual urine is found, the bladder should be emptied perfectly and washed out every day or every other day. No hesitation need be felt in the use of the catheter in these cases. Scores of patients die every year from want of the catheter because it is thought that all is right if they pass urine freely. The residual accumulation or the slight cystitis sets up chronic pyelo-nephritis, which develops insidiously, and is unsuspected until mysterious febrile disturbance occurs, chronic or acute, and leads to death. It is probable that the onset of the final acute disturbance is sometimes determined by the passage of a catheter, just as this will cause a rigor or transient hæmaturia in a healthy person, and then the case is called "catheter fever." I have seen many melancholy instances of death from this cause which might have been prevented, and I have never seen any case in which the early and frequent use of the catheter did harm. It is especially important to insist on the danger that the practitioner may be misled by the patient, who thinks that because he passes urine he necessarily empties his bladder.

Electricity has little influence on the chief symptoms of tabes. The voltaic current is powerless over either the pains or the ataxy, whether it be applied to the spine or the limbs. Faradisation of the skin by the wire brush has been recommended for the defect of cutaneous sensibility, but it has little, if any, influence. When the bladder or its sphincter is weak, faradisation from the hypogastrium to the perineum may be employed, and occasionally seems to do some good. In secondary wasting of the muscles, an attempt may be made to maintain their nutrition by stimulating them with whatever current they respond to, but the central cause usually renders local treatment ineffective.

There remains for consideration one therapeutic procedure—nerve stretching, an operation that has usually been performed on the sciatic nerve. In the first case in which it was employed (by Langenbuch), the procedure was followed by remarkable and mysterious improvement, not only in the pains, but also in the incoördination. The patient subsequently died under chloroform, administered in order that the nerves of the arms might be stretched. The spinal cord was found by Westphal to be healthy. Although the nerves were not examined, there can be little doubt that it was one of the cases in which the nerves alone are diseased. The operation has since been performed in a large number of cases. In some the pains have been lessened for a time; in a few there has been some temporary improvement in the incoördination. In many cases the procedure has had no influence on the symptoms. If ever justified it is only as a last resort, in a case in which the pains are very intense, and are especially felt in the region of the sciatic, but it is not justifiable in any case to hold out an expectation of more than possible, and perhaps transient, relief to the one symptom. It must be remembered, moreover, that the operation is not devoid of danger of evil results; there is the risk incidental to the necessary anæsthetic, and the operation has also caused death through the agencies of erysipelas and spinal hemorrhage. Its modus operandi is not easy to explain, and the theories that have been advanced to account for its influence are so inadequate as to be scarcely worth reproduction. Its common inutility is more easily intelligible. It would seem now to be passing into merited disuse.

PRIMARY SPASTIC PARAPLEGIA.

PRIMARY LATERAL SCLEROSIS.

The morbid state thus designated is one that has been, and still is, the subject of much discussion. Its relations are complex, and a somewhat lengthy pathological introduction is necessary.

We have seen that, in every kind of transverse lesion of the spinal cord, provided this is situated above the lumbar enlargement, the paralysis of the legs is soon accompanied by excess of myotatic irritability (increased knee jerk, foot clonus), and that the muscle reflex action, on which this irritability (and muscular tone) seems to depend, gradually increases to tonic spasm, so that a condition develops to which the term "spastic paraplegia" is applied. A condition, quite similar to that which is thus secondary to a transverse lesion, often develops gradually, without any indication of a primary focal disease, and without any sensory symptoms to indicate that the mischief extends beyond the purely motor elements of the cord. We have also seen that these symptoms indicate disease of the upper segment of the motor path, the cortico-spinal segment, which extends from the motor cortex, through the pyramidal tracts, and ends in the gray matter of the cord, doubtless by a subdivision and ramification of the nerve fibres in the fibrillary network of the spongy substance. From the gradual onset and limitation of the symptoms in these cases, it has been assumed that the disease consists of a primary sclerosis of the pyramidal tracts, *i. e.*, in a degeneration of the fibres of this upper segment. Since these tracts run chiefly in the lateral columns, the disease has been termed "lateral sclerosis." The clinical features presented by these cases, and their probable significance, were first pointed out by Erb.* Degeneration of the pyramidal tracts had indeed been previously observed, by Türck (1856) and Charcot (1865). Erb's inference as to the nature of these cases was supported by the independent (and indeed previous) researches of Charcot on cases of muscular atrophy; he showed that in such cases muscular rigidity coincides with degeneration of the pyramidal tracts. Pathologists have since been searching for confirmation of the hypothesis—for evidence that the symptoms, in their pure form, without muscular atrophy, depend on degeneration limited to the pyramidal tracts. Such degeneration, in slight degree, associated with slight symptoms, has been found in cases of general paralysis of the insane (by Westphal and others), but in all other cases that have been examined, either other parts of the white substance have been degenerated, or the disease has involved also the anterior cornua.† In some cases, as one published by Dreschfeld (of which a figure is given at another page) the change in the anterior cornua has been slight, so that the required conditions are nearly but not quite fulfilled.

Although absolute demonstration has not yet been furnished, the indirect evidence of the correctness of Erb's hypothesis is very strong, the only question being whether, in the cases in which there is no disease of the motor nerve cells of the anterior cornu, the degeneration is so limited to the pyramidal tracts as to constitute a system disease in the strict sense of the word. It is not surprising that demonstration of the nature of the pure cases is not forthcoming, since, as we shall see, the disease has very little tendency to shorten life.

Cases that present the same group of symptoms are very various in nature. We have seen (p. 154) that, in each segment of the motor path, the same symptoms are produced by disease of any part of the segment. In the upper segment, with which we are now concerned, the symptoms are the same, whether the disease is in the cortex of the brain, the internal capsule, the pyramids of the medulla, or the pyramidal tracts of the cord; and they must also be the same if the disease is limited to the termi-

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* In 1875 *Berlin klin. Wochenschr.*, No. 26; 1877, *Virchow's Archiv*, Bd. 70.

† In a case recorded by Stofella no other part than the pyramidal tracts could be seen diseased on naked-eye observation, but no microscopical examination was made.

nation of the segment in the gray matter of the cord. In cerebral hemiplegia the state of the arm may closely resemble that present when plastic paraplegia involves the arm. The leg in hemiplegia presents a less close resemblance to its condition in spinal disease, because the leg is innervated from both cerebral hemispheres, and the supplementary influence of the hemisphere of the same side lessens the effect of the disease of the hemisphere of the opposite side. But if there is disease of the leg centres in both hemispheres, the state of the legs may be identical with that resulting from disease of the spinal cord. Such bilateral disease often results from injury during birth,—meningeal hemorrhage over the upper part of the central convolutions. This resulting condition is termed "congenital spastic paraplegia."*

On the other hand, it is extremely probable that degeneration is sometimes limited to the termination of the segment in the gray matter. This is probably susceptible of isolated disease, just as the corresponding termination of the lower segment is paralyzed alone by curara. In such disease the white columns would be found normal. It is highly probable that this was the pathological condition in one recorded case, in which the symptoms of spastic paraplegia existed during life, and no anatomical change was discovered after death. The detection of the disease of the terminal portion of the segment in the gray matter is extremely difficult, perhaps impossible, because the structure must consist of nerve fibrillæ that interlace with others that are unaffected. In other cases, some degeneration has been found in the lateral pyramidal tract in the lumbar region, slighter, however, than the intensity of the symptoms suggested, and gradually lessening or even ceasing higher up the cord. Such a condition is comparable to the degeneration of the lower portion of the second segment of the motor tract in some cases of peripheral (degenerative) neuritis, in which there is a degeneration of motor nerve fibres, greatest in their extremities, extending for a variable distance up the nerves, but lessening long before the spine is reached.

Both clinical and pathological evidence shows that the morbid state often occurs also as part of a more extensive degeneration. Degeneration may occur in both lateral and posterior columns, giving rise to combined paralysis and ataxy, "ataxic paraplegia," sufficiently definite in its clinical characters and course to make its distinction convenient, and it is therefore separately described. The following account of the clinical features of spastic paraplegia is founded on cases in which the motor paralysis and spasm existed alone, with no definite sensory loss, in which they came on gradually, with nothing in the state of the patient or the history of his symptoms to suggest either a focal lesion or an acute process.

CAUSES.—An inherited neuropathic tendency is to be traced occasionally in this as in other chronic spinal diseases. Thus in one case there was a history of insanity in uncle, aunt, and two cousins. The disease affects both sexes in almost equal frequency, presenting in this a contrast to posterior sclerosis. The period of life at which it most frequently begins is between twenty and forty; about three-quarters of the cases begin in these two decades, and about an equal number in each. Cases frequently commence, however, in the second decade of life, chiefly in its latter portion. After forty they become much less common; the latest age at which I have known a typical case to commence is sixty-one.

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* Attention was called to these infantile cases by v. Heine as early as 1840, by Little, in his work on "Deformities," in 1853, and in the *Obstetrical Transactions* for 1862. See McNutt, *Am. Journal of Medical Science*, January, 1885.

The disease sometimes follows syphilis in a way to suggest a causal relation, even when all cases are excluded in which there is any reason to suspect a focal lesion. In one case the symptoms commenced six months after the primary disease.* But this antecedent is not frequent, and in this respect also the disease contrasts with posterior sclerosis. Proximate causes are to be traced only in a minority of the cases. The most frequent is concussion of the spine, such as a fall on the back. Some time, often two or three years, elapses between the fall and the first pronounced symptoms of the disease, and hence focal lesions due to the fall can be excluded. Next in frequency is repeated exposure to wet cold. Very rarely the symptoms have slowly followed some acute illness. In several cases the disease has succeeded prostration after childbirth or abortion, or has commenced during lactation. In one case an attack of arthritis in the knee and ankle of one leg was the immediate antecedent, and this leg was the first to become weak. In another case the symptoms followed subacute arthritis of both knee joints, apparently rheumatic in nature. The possibility that a joint inflammation may be of spinal origin must be borne in mind in considering the significance of such cases, but it is on the whole probable that a primary joint affection is an occasional cause of the spinal disease.† Acute arthritis, certainly of spinal origin, has only been observed in severe myelitis. The cause of the congenital form is always injury to the brain during birth, in most cases meningeal hemorrhage, perhaps sometimes compression of the cortex.

Symptoms.—Weakness of the legs, of very gradual development, is the first symptom. The patient finds that he gets tired more readily than before, and that the legs feel heavy. It is very common for one leg to become weak before the other. The progress of the weakness is very variable, but it is slow in all characteristic cases. In many instances the patient is still able to walk a mile or two, even after the disease has lasted for several years, slowly increasing. On the other hand, walking power may be almost lost at the end of six months. It is doubtful whether more acute cases should be classed in this category. When the patient seeks advice, the degree of weakness is thus very variable. It will generally be found that there is very distinct loss of power in the flexors, often greatest in the flexors of the hip, but considerable also in those of the knee and ankle. The knee jerk is excessive and quick; the rectus contraction can be obtained, as the patient lies, by tapping the depressed patella, and a rectus clonus is often obtainable by sudden depression of the patella. The foot clonus is usually also obtained with readiness. In rare cases, in which the upper part of the legs suffers most, there may be a rectus clonus, but only slight indications of a foot clonus, two or three movements quickly ceasing. The tendency to spasm is at first noticeable as slight stiffness of the legs on first rising in the morning, but it gradually increases in degree as power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to the pelvis, so that, as the patient lies, if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee cannot be passively flexed by any force that can be applied to it, until the spasm has become less. When flexed the limb is comparatively supple, but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity." The spasm is, roughly speaking, proportioned to the loss of power, and its extensor character may enable the patient to stand, the legs being fixed and rigid, when muscular power is quite insufficient to support the body, were it unaided by the spasm. In a still greater degree, a voluntary effort may only excite general spasm of the limb instead of causing a definite movement. Occasionally there are also paroxysms of brief flexor spasm, causing the legs to draw up. This occurs chiefly when the patient is in bed, and especially during sleep. When the spasm is great, a paroxysm may pass into violent clonic spasm. This was termed "spinal epilepsy" by Brown-Séquard, but it has, of course, nothing to do with epilepsy. It is merely the same clonus that can be produced by passive extension, excited by the tension of the spontaneous spasm. A paroxysm can often be arrested by a strong sensory impression, such as a prick or a pinch, and also, as Brown-Séquard has shown, by a forcible flexion of the great toe, which probably acts in a similar manner. Reflex action from the skin is also usually excessive, and the stimulus excites an attack of spasm.

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* The cord lesion was found to be sclerosis, but the direct cerebellar tract was also degenerated (Minkowski).

† See "Arthritic Muscular Atrophy."

The gait of the sufferers from spastic paraplegia is very characteristic. The legs seem to drag behind the patient, and, in walking, each is hauled forward as a rigid whole, the toes catching against the ground, and, when the ball of the foot rests on the ground, the limb may shake from the clonus developed by the extension of the calf muscles. When the patient sits, a similar trepidation occurs until the patient pushes the leg forward so that the heel rests also on the floor. The muscles of the leg are usually large and well nourished. They often seem, indeed, to be hypertrophied, and perhaps are really so; the contraction of spasm constitutes a physiological stimulus to growth just as does voluntary effort. But they are not always large; they are sometimes moderately wasted. Rarely they are much wasted; such cases are considered in the chapter on muscular atrophy. The electric irritability is usually perfectly normal, but in the muscles that present the slight wasting just mentioned, there may be a trifling diminution of irritability, to faradaism and voltaism alike. When the patient ceases, or almost ceases to walk, contracture sometimes occurs in the calf muscles. An active contracture is indeed common, preventing at first passive flexion of the ankle beyond a right angle, but if the pressure is maintained, the muscles yield and full flexion is possible. But in some cases there is a fixed contracture that cannot be overcome. This rarely develops while the patient is walking about, but is common when rest in bed permits habitual extension of the foot on the leg. It occurs especially when there is slight muscular wasting. Flexor contraction at the hip or knee may occur also in the same cases, under the influence of posture, but is on the whole rare, the tendency to it being counteracted by the extensor spasm.

The arms are often unaffected, but they suffer in some cases, and present the same progressive weakness and excess of myotatic irritability. Vigorous contractions are elicited by a tap on the tendon of a muscle, or on the bone to which the tendon is attached, provided the muscle is extended. A clonus can often be obtained in the flexors of the fingers. There is less paroxysmal spasm than in the leg, but much rigidity of the limb, from active muscular contracture, flexing the elbow and flexing the fingers at all the joints, as in the "late rigidity" of hemiplegia. The difference in the form of spasm in the arm and leg is no doubt connected with existence of the reflex extensor mechanism, in the lumbar centres, concerned in the act of standing. The muscles of the arm are often as well nourished as those of the leg. The affection of the two arms is very rarely equal, and it is common for one arm to be almost normal when the other is much affected, and both legs are equally involved. Sometimes, however, the arm and leg on one side are paralyzed, and the limbs on the other side very slightly. In comparison with the palsied members those on the other side may seem normal, and the patient often believes that they are, but examination almost always reveals slight weakness and an abnormal degree of myotatic irritability.

The muscles of the trunk may also suffer. A subjective "feeling of weakness" in the back is often complained of early, but seems to be as much a sensory as a motor symptom. Painful spasm of the abdominal muscles may occur in severe cases, and I have even known attacks of tetanoid rigidity of the back to occur on attempts to move.

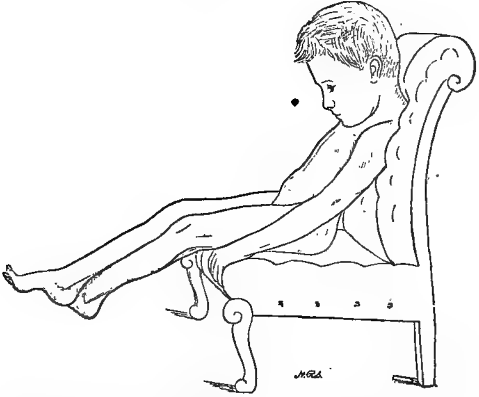
It is not common, in typical cases, for the symptoms to extend into the region of the cranial nerves, but in rare instances difficulty of swallowing and of articulation has existed, due to a similar palsy of the bulbar nerves. Excessive myotatic irritability has also been observed in the muscles of mastication, so that a tap on the chin causes a vigorous elevation of the depressed jaw (Beevor; see p. 160).

Sensory symptoms are often entirely absent, with the exception of slight dull pains in the legs, or more frequently in the back. The sensation of weakness in the back, already mentioned, occasionally reaches an intense degree. Rheumatoid pains are troublesome in some cases; sharp pain is rare, except in untypical cases to be presently mentioned. In such cases, also, there may be very slight defect of sensibility, such, for instance, as blunting of tactile sensibility on the finger tips. Subjective sensations of "numbness," tingling, formication, are more common. They may exist for years without the development of any other anæsthesia, in spite of the progress of the motor symptoms.

The sphincters are often affected, and sometimes very early in the course of the disease. Sexual power may be lost, or may remain, even when the spastic paraplegia has reached a high degree. The nutrition of the skin and joints undergoes no change.

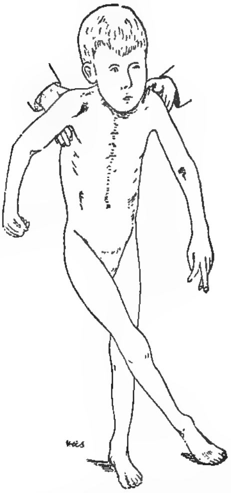
Ocular symptoms are extremely rare. The action of the pupils is usually perfect, and I have only once seen optic nerve atrophy in an uncomplicated case.

FIG. 117.



Congenital spastic paralysis (cerebral). Extensor spasm in the legs excited by a sensory impression.*

FIG. 118.



Infantile spastic paralysis of cerebral origin: crossed-legged progression. (*Drawn by Dr. Spencer, from a photograph.*)

The infantile form may resemble very closely that which occurs in adults. There is the same extensor spasm and increase of all forms of reflex action. As the child sits on the knee or a chair any sensory stimulus will make the legs shoot out in spasm (Fig. 117). But the spasm does not reach the extreme degree often attained in the common form. The excess of the knee jerk is always distinct, but a foot clonus is not often to be obtained, perhaps because the muscle reflex mechanism related to the calf muscles has not received the functional development that must result from the ever-recurring sequence of tension and contraction involved in walking. The active contracture in the calf muscles, which most cases present, is a serious hindrance to walking even when the muscular power is sufficient. It is long before the attempt to walk overcomes the contracture. In most cases, however, the child ultimately gains the power of walking, although much later than normal, and it often presents some peculiarity of gait, sometimes a tendency to "cross-legged progression," in which one foot gets over or in front of the other (Fig. 118), or a swinging oscillation of the body occurs which may persist to adult life. The growth of the legs is hindered.

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* Drawn by Dr. H. R. Spencer, from a photograph by Mr. Hyde Marriott.

The arms do not present tonic spasm such as is seen in adult cases. There may be a choreic disorder of movement, spontaneous irregular movements with incoördination. In the cases that can fairly be called "spastic paraplegia," the arm symptoms are slight. When considerable, the condition is usually termed "double athetosis," and its characters are described in the account of diseases of the brain.

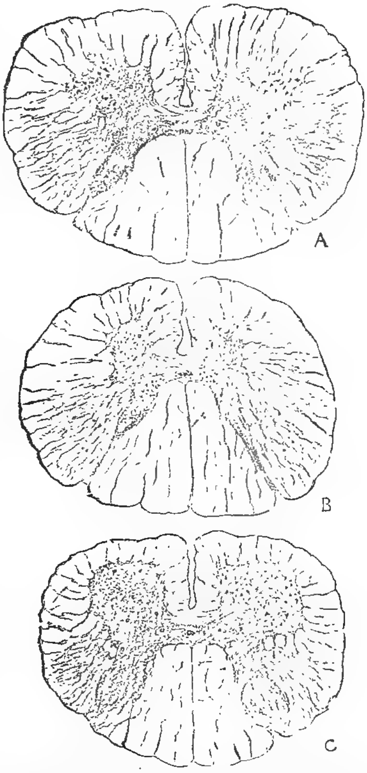
Transitional forms are met with which constitute gradations between primary spastic paraplegia and other varieties of sclerosis of the spinal cord. Indications of posterior sclerosis may coexist, and to the motor symptoms there is added incoördination, sometimes with other sensory symptoms. These cases of "ataxic paraplegia" are described in the next section. Cases may begin as pure tabes, and indications of lateral sclerosis may be superadded. On the other hand, muscular wasting in the arms, considerable in degree and progressive in character, may be associated with indications of lateral sclerosis in the legs, cases to which the name "amyotrophic lateral sclerosis" has been given by Charcot. Very rarely considerable muscular wasting succeeds, in the same part, the symptoms we have been considering.

Lateral sclerosis of the spinal cord (like posterior sclerosis) often forms part of the morbid process that constitutes general paralysis of the insane. In some cases of this character the mental symptoms are extremely slight, and the case may have the aspect of a primary spinal disease, with slight mental failure complicating it.

The course of characteristic cases of primary spastic paraplegia is usually very chronic, and the malady may, at any stage, cease to advance. In one case slight symptoms have apparently been stationary now for twenty years. Often, however, the disease reaches a considerable degree before it becomes stationary. It is perhaps the least dangerous to life of any chronic spinal disease. Even secondary kidney trouble scarcely ever occurs; perhaps the excessive reflex action may save the bladder from injurious over-distention. It is when other elements of the cord suffer that dangerous complications ensue.

Pathological Anatomy.—In the fact just stated we probably have an explanation of the silence of morbid anatomy on the subject of uncomplicated lateral sclerosis. "Nec silet mors," the apt motto of the Pathological Society, is true chiefly of disease that kills. Complete degeneration of the pyramidal tracts, anterior as well as lateral, is met with in the cases in which the anterior ganglion cells and motor nerves are also diseased, although, in some instances, the amount of this disease is small, and limited to the cervical region. An instance of such degeneration is shown in Fig. 119. The degeneration in the lumbar enlargement is usually limited to the lateral tract, the anterior having disappeared higher up the cord. It is bounded externally, in the dorsal and cervical regions, by the normal direct cerebellar tract, and internally by the narrow "lateral limiting zone," which intervenes between the pyramidal tract and the intermediate gray matter. There is the usual increase of connective tissue and wasting of nerve fibres. In many cases granule cells are abundant in the affected area. They are always present in cases of short duration and indicate the stage rather than the nature of the process. The degeneration probably begins in all cases in the nerve elements themselves. In most cases many nerve fibres can still be seen, scattered through the sclerosed area; most of them are fibres of the direct cerebellar tract which lie among the pyramidal fibres (see p. 134). The degeneration of the tract has been traced through the medulla, pons, and cerebral hemispheres, to the motor cortex, in which indications of degeneration have also been found. This degeneration through the brain was traced in one case in which the disease of the anterior cornua was slight, the wasting was limited to the hand muscles, and the spastic paralysis began in the legs and then invaded the arms.† Such a case is almost a pure degeneration of the whole of the first segment of the motor path. The degeneration of the pyramidal tracts is well shown in Fig. 120, from a case of progressive muscular atrophy. The anterior and lateral tracts are entirely degenerated, and the sclerosis stops abruptly at the outer margin of the lateral, but in front extends forward, probably in the short fibres that connect the anterior cornu at different levels. In other cases, in which the anterior ganglion cells were normal, the sclerosis has not been limited to the pyramidal tracts; the anterior columns have been also sclerosed in the lumbar region of the cord.‡

FIG. 119.



Sclerosis of the lateral and (in the cervical region anterior) pyramidal tracts, with slight degeneration of the anterior cornua. A, Cervical; B, dorsal; C, lumbar sections.*

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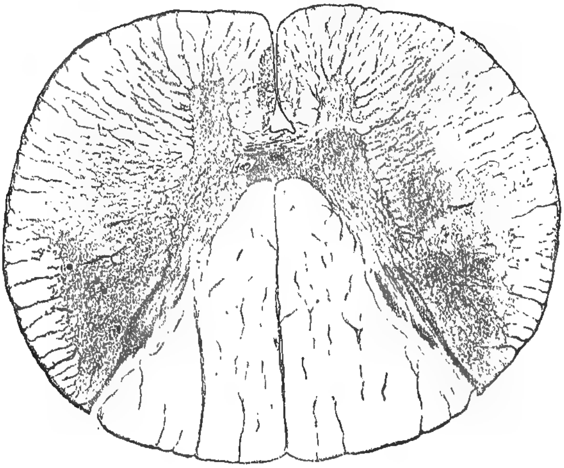
* I am indebted for these sections to Dr. Dreschfeld, who has published the case (*British Med. Journal*, Jan. 29th, 1881). † Kojewnikoff, *Arch. de Neurologie*, *1883*, No. 18.

‡ As in a case recorded as one of spastic paraplegia by Hopkins (*Brain*, Oct. 1883), but this was an example of combined lateral and posterior sclerosis (ataxic paraplegia). An illustration of the changes in this case is given in the next section.

The direct cerebellar tract (and posterior vesicular columns) have been also found degenerated with the pyramidal tract, and an annular sclerosis of the periphery of the antero-lateral column has been found in several cases.* More frequent still is a combination with degeneration of the posterior columns, described more fully in the next section. In most of these combined cases, the degeneration has lessened in the upper part of the cord. In one case, at least, the white columns of the cord have been found healthy. The probable significance of this fact, and the general conclusion from the anatomical evidence, has been already stated in the introductory paragraph. One point, however, that deserves mention is the occasional occurrence of lateral sclerosis in association with insular sclerosis. In some cases of this character the degeneration of the pyramidal tracts is purely secondary, the result of the damage to the pyramidal fibres by an islet of sclerosis situated in some part of their course. But it does not appear that this explanation can be given of all cases. In some instances the sclerosis of the pyramidal tract appears to be independent and coincident. A similar association of insular and posterior sclerosis has also been observed.

FIG. 120.



Sclerosis of the pyramidal tracts, lateral and anterior: dorsal region.
(*From a case of muscular atrophy.*)

The probable mechanism of the symptoms that give the dominant characters to the malady, have been considered in the general account of the symptoms of disease of the spinal cord. The most important fact is that the degeneration, whatever its upward extent, always involves the lowest part of the pyramidal segment, in consequence of its descending tendency. Hence the intra-cornual termination must always be involved. This, it is assumed, is the structure that controls the muscle-reflex centre, and the

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* Direct cerebellar and pyramidal tracts in a case by Minkowski, in which the disease rapidly succeeded syphilis; annular sclerosis by Westphal (see "Ataxic Paraplegia").

consequent loss of control explains the excess of myotatic irritability and the progressive spasm, progressive by a functional hypertrophy resulting from continuous over-action. When there is the rare consecutive wasting of muscles, we must conceive that the degeneration involves also the ganglion cells and lower segment of the motor path; when, as is the rule, there is coincident wasting of other muscles than those that are the seat of spasm, it would seem that some elements of this lower segment are the seat of a primary degeneration. The slighter muscular wasting without, or with only trifling, change in electrical irritability, is the expression of a slighter alteration in the nutrition of the cells and fibres, without actual destructive degeneration.

DIAGNOSIS.—The diagnosis rests on the combination of weakness, excess of myotatic irritability, and spasm,—on the gradual onset of these symptoms,—and also on the absence of indications of a focal lesion. An acute onset, occupying a few days or weeks, is *primâ facie* evidence of a lesion that takes the case out of the category of degenerative disease. In most acute cases there is other evidence of a focal lesion, extending, at some level, beyond the limits of the motor path. Some indications are initial impairment of sensation, or a girdle pain. The latter indicates irritation of the posterior root fibres at a certain level, and proves that at that level the disease extends beyond the limits of the pyramidal tracts. Spastic paraplegia is common after such lesions, but is secondary and not primary.

If any marked sensory symptoms exist in a case that presents the symptoms and course of primary lateral sclerosis, these are evidence that the sclerosis extends beyond the motor tracts, and the case is not one of pure lateral sclerosis. It is doubtful, however, what significance is to be attached to very slight sensory symptoms, such as slight subjective sensations of dull pain, formication, etc., when they exist alone. It is possible that they are due to functional disturbance in the sensory nerve elements and do not imply structural disease outside the motor area. They usually remain stationary, and are not followed by any more pronounced sensory symptoms. Considerable muscular atrophy in any part is commonly regarded as bringing the case under the designation of "amyotrophic lateral sclerosis," but some cases of the kind, in which the wasting is confined to a few muscles, resemble more closely the cases we are now considering than they do those with widespread and extreme muscular atrophy. These cases are further considered in the account of progressive muscular atrophy.

The disease is not uncommon in those who are at the age and of the sex at which hysteria prevails, and there is no form of cord disease that is so often mistaken for hysterical paraplegia. The mistake is facilitated by the perfect muscular nutrition in most cases of spastic paraplegia. When there is considerable spasm, the mistake ought not to occur, as the extensor character of the spasm, described above, is distinctive; nothing resembling it ever occurs in hysterical paraplegia. Hysterical contracture is fixed, and does not vary with posture as does the "clasp-knife rigidity" of spastic paraplegia. When the spasm is trifling or absent, as in slight and early cases, the diagnostic difficulty is much greater, and is increased by the fact that slight excess of myotatic irritability occurs in some cases of so-called hysterical paralysis. But this scarcely ever reaches the degree necessary to give rise to a true foot clonus or a rectus clonus. There may be a "spurious foot clonus," or a true clonus may be obtainable if there is hysterical contracture, but apart from such contracture a true foot clonus or a rectus clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis, in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I do not recall a single instance in which the opposite error resulted from undue regard to this symptom. When there is an excess of myotatic irritability in so-called hysterical paralysis it is a persistent symptom, and must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle-reflex centres. On the hypothesis that I have advanced, this control is exerted by the termination of the pyramidal fibres, *i. e.*, of the upper motor segment, by the structures, degeneration of which may probably cause spastic paraplegia. A case is actually on record, the case of lateral sclerosis observed by Charcot in 1865, in which an initial hysterical paraplegia, cured suddenly, and relapsing on emotion, passed into lateral sclerosis.

When the arm and leg suffer on one side only, the disease may be mistaken for cerebral hemiplegia. The resemblance may be very close. There is not, however, any affection of the face, which, although theoretically conceivable, is, as a matter of fact, always absent. The limbs on the other side are never normal, but present slight symptoms. The excess of myotatic irritability in the other arm has afforded, in all the cases I have seen, a distinct indication of the nature of the malady. It is never present in simple cerebral hemiplegia, although such excess is often present in the leg of the unparalyzed side.

The diagnosis of the congenital infantile form is only difficult when the observer is unaware of the occurrence of these cases. A slight degree of incoördination in the hands will usually be found if they are carefully watched while the patient takes hold of some object. The wide separation and irregular movement of the fingers is very characteristic. Chronic primary cord diseases are almost unknown in young children. Caries of the spine, causing paraplegia, is, in them, always recognizable, and the distinct onset of the paralysis, in a previously healthy child, is an absolute distinction from the cases of birth palsy. The same fact also distinguishes glioma of the pons, which may likewise cause spastic paralysis of arms and legs.

The disease with which the infantile form is most frequently confounded is pseudo-hypertrophic paralysis, to which a superficial resemblance may be caused by the gait of slight cases, the large muscles, and the contracture of the calves. Besides the other characteristic features of the muscular malady, the two are distinguished especially by the excessive knee jerk of spastic paraplegia, by the active and yielding nature of the contracture, and by the course of the disease; the impairment of locomotion gradually lessens in birth palsy while it increases in pseudo-hypertrophic paralysis.

Prognosis.—In primary lateral sclerosis the chance of recovery and the danger to life are both small. There is some prospect of arrest, and even of slight improvement, if the disease has not reached an advanced stage. When the spastic state is well developed and has lasted for some time it is very rare for marked improvement to occur. This is perhaps due rather to the tendency of the disease than to its degree, since a similar condition that is secondary to a focal lesion may undergo remarkable improvement. In slight cases there may be considerable gain of strength. In some cases which improve it is possible that the disease is limited to the terminal structure. In the infantile form, the prognosis is much better. Most children ultimately gain some power of walking, and in slight cases the ultimate impairment may be trifling.

Treatment.—So far as drugs are concerned, the treatment is the same as that of posterior sclerosis already described. The drugs most useful are the same, but, unfortunately, their influence is less frequently appreciable. Nux vomica and strychnine have, however, to be given with caution, and in very minute doses, as they have a tendency to increase the spasm. In severe cases this is a most distressing symptom, and often not amenable to any influence. Bromide sometimes lessens it slightly, but even large doses of bromide have but a trifling effect. Neither Indian hemp, belladonna, calabar bean, or piscidia erythrina has much effect. Absolute rest is sometimes of service, and occasionally seems to produce improvement, which all treatment failed to effect while the patient was walking about. Rubbing is also beneficial in some cases. Its influence on the spasm is often very distinct during the process, and a long course of rubbing has produced a permanent improvement in the spastic condition. Upward rubbing seems to have more influence than kneading the muscles. If there is contraction of the calf muscles, the foot should be pressed up while these muscles are rubbed. When there are facilities for it, the rubbing may advantageously be combined with sweating in the Turkish bath. In one case, of moderate degree, almost all the symptoms passed away after a long course of Turkish baths, arsenic being also given. The patient, who could at first walk scarcely half a mile, became able to walk several miles without fatigue. Electricity is useless in the pure disease. Faradaism, and all painful operations, are harmful, stimulating further the already excessive excitability of the reflex centres. The constant current to the muscles, or from the spine to the muscles or to the feet in water, has no influence in lessening the spasm or improving the strength, as far as I have been able to observe, and I have tried it thoroughly in many cases.

In the infantile form drugs are useless. Rubbing is desirable, and can be efficiently performed by the nurse or mother. Carefully planned gymnastic exercises are also useful. The tendo-Achillis is sometimes divided for the contracture of the calf muscles, but the operation is useless and ought never to be performed. Supports occasionally help the child to walk somewhat sooner than it would without their aid, and so may hasten improvement, but they should only be employed where there is sufficient power and control to enable them to be useful.

ATAXIC PARAPLEGIA.

COMBINED LATERAL AND POSTERIOR SCLEROSIS.

The term ataxic paraplegia seems the most accurate clinical designation for a disease of the spinal cord which presents a combination of the symptoms of paraplegia and ataxy, and consists in combined disease of the posterior and lateral columns. Although its clinical features present some varieties, and may approach those of each of its constituent forms of disease, yet in the majority of cases the symptoms are uniform, and sufficiently characteristic to justify the distinction of the disease and its separate description. A few pathological observations have been published.* The following account is based on these and on a series of cases that have come under my own observation, and of which the clinical characters were well marked.

Causes.—Neurotic heredity is to be traced only in a small proportion of the cases, about one-tenth. A history of syphilis is as rare as it is frequent in pure tabes. Males suffer much more frequently than females. The disease usually commences between thirty and forty, but I have known it to begin as early as nineteen and as late as fifty-two. Exposure to cold is to be traced occasionally as an exciting cause. In one young lady, the symptoms commenced after a season of balls, at which when heated she would habitually sit at open windows, and often sleep in clothes saturated with perspiration. A severe concussion of the spine has sometimes preceded the first symptoms by a few months. The disease may also follow great sexual excess. In many cases no cause can be traced, immediate or remote. Its general etiology thus resembles that of simple spastic paraplegia.

Symptoms.—The onset of the disease is usually slow and gradual, two or three years passing before walking power is much impaired; it is rarely subacute, so as to reach a considerable degree in two or three months. The legs are the first to suffer, and the symptoms may remain limited to them or may involve the arms also. There is a slow and simultaneous development of weakness and defect of coördination. The patient finds that he tires more readily than before, and also becomes unsteady on turning or walking in the dark, and these symptoms gradually increase. Examination then shows distinct loss of power in the legs. This may be slight and difficult to elicit, but if all the movements are tested it will be generally found that there is marked weakness of the flexors of the knee and hip, often much greater than that of the extensors. Frequently one leg is distinctly weaker than the other. The patient is unsteady when he stands with feet together, and he tends to fall if the eyes are then closed. If the feet are bare, the irregular action of the muscles is shown by the movement of the tendons on the dorsum of the foot, as in tabes. In most cases the incoördination is revealed also by the patient's gait, which is distinctly unsteady. There is rarely the high movement and sudden descent of the feet often seen in tabes, although I have observed it in a few cases. But the walk may closely resemble that of many cases of tabes ; the patient is unsteady, reels on turning, and has often to bring his foot suddenly to the ground to maintain his equilibrium. He may even have to steady himself with a stick, or to catch hold of some adjacent object, to save himself from falling. The ataxy is equally evident when he lies, and attempts, his eyes being closed, to touch some object with his foot.

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* By Prevost, *Arch. de Physiologie*, T. iv; Pierret, *ib.;* Babesieu, *Virchow's Archiv*, Bd. 76; Kahler and Pick, *Arch. f. Psychiatrie*, Bd. viii; and Westphal, *ib.*, Bd. viii and x. Westphal's observations are the most important.

The sensory and reflex symptoms present a marked contrast to those of tabes. Lightning pains are almost always absent ; I have only met with them in one case, and in this they were a transient symptom. Sometimes there is a slight dull pain in the legs, felt especially on fatigue. Dull pain in the sacral region or in the spine is not uncommon, and is often an early symptom. The sacral pain, indeed, is sufficiently frequent to deserve special note. A girdle pain is met with only in rare cases. As a rule, there is no loss of sensation either on the legs or trunk. Hyperæsthesia is equally rare. Reflex action from the sole may be normal or increased ; less commonly it is diminished. The cremasteric and abdominal reflexes are sometimes lost. The most striking difference from tabes, however, is in the condition of myotatic irritability, which, in the vast majority of cases, is greatly increased. The knee jerk is quick and extensive ; it can be obtained from above (see p. 36), and there is generally a distinct rectus clonus. The foot clonus is also commonly to be obtained. This myotatic excess usually persists throughout the whole course of the disease, whereas, in ordinary tabes, however slight the lesion, the knee jerk is all but invariably lost.

The arms may be normal, or may present symptoms similar to those in the legs, incoördination, weakness, and marked excess of the myotatic irritability. The muscles in both arms and legs are usually well nourished.

Sexual power is often lost early in the disease. The sphincters are frequently impaired, sometimes early, but the impairment rarely reaches a considerable degree.

The iris usually acts to light, but I have seen loss of the light reflex in two or three cases and have once known accommodation to be lost, the action to light being normal. Optic nerve atrophy occurs only in rare cases, less frequently than in tabes ; I have seen one case in which there were indications of a retro-ocular axial neuritis. The external ocular muscles are usually normal, and there is no nystagmus.

Slight impairment of articulation is not uncommon ; sometimes there are irregular tremulous movements of the face resembling those of general paralysis, and this in cases in which there is no mental change. Westphal has observed marked ataxy of the facial muscles. As a rule, the mental state is either normal or there is merely slight failure of memory. Undue frequency of the pulse is occasionally present, but is less common than in tabes.

As the disease increases, the muscular power becomes more and more impaired, usually without much increase in the incoördination, which sinks into the background as the paralysis increases, and the patient has to have recourse to supports which at the same time afford him guidance. The increased myotatic irritability continues; stiffness and rigidity develop as its consequence. Thus the aspect of the patient comes to be that of spastic paraplegia; the feet drag as the patient walks, shake from clonus when he stops, and the legs are hauled forward with visible effort at each step. Indications of unsteadiness may still be observed in isolated movements. The arms, if previously free, may begin to suffer, but sometimes they escape altogether. The motor weakness may go on to complete paralysis. This is quickly reached in some cases, but so slow is the usual progress of the disease that one patient in whom the disease has at no time been absolutely stationary, is still able to stand, whose case I have had under observation for eight years. In spite of the progress of the weakness, sensation remains in most cases unimpaired, and the reflex action of the iris, unless early lost, continues perfect throughout.

The symptoms present, in some cases, variations from this type. Sensation on the legs may be impaired and the knee jerk may be lost. These cases are intermediate between the form now under consideration and pure tabes. They are, however, very rare, and do not constitute more than five per cent. of the cases in which ataxy and weakness come on together.

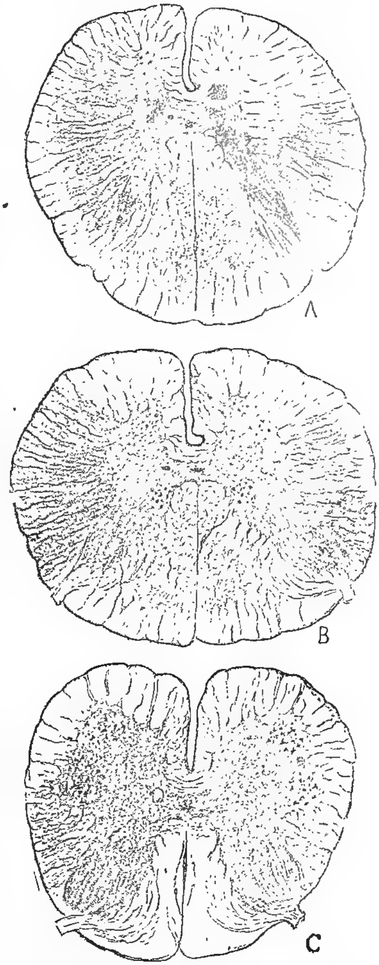
The disease has little tendency to cause death. Indeed, the fatal cases have, for the most part, been untypical, and do not convey an accurate idea of the characters of the disease. The chief danger to life is from the accidents common to all chronic spinal affections,—kidney disease and bedsores.

Among complications the most important are mental changes resembling those of general paralysis of the insane, of which, indeed, this combined sclerosis may form part. Slight muscular atrophy sometimes occurs. Arthritis of doubtful significance has been once observed. A patient of Westphal's presented derangement of the sympathetic, and died from peculiar spasm of the muscles of respiration. Visceral crises, however, are practically unknown.

Pathological Anatomy.—In all cases the spinal cord has presented sclerosis of both posterior and lateral columns; but the precise extent and degree of the degeneration are subject to considerable variations. As a general rule, the sclerosis of the posterior columns differs from that of tabes in two particulars. First, it is not more intense, and often it is less intense, in the lumbar than in the dorsal region of the cord. Sometimes, indeed, as in the case shown in Fig. 121, in the middle and lower parts of the lumbar region, the posterior columns may be free from sclerosis, although it is considerable in the dorsal region and at the junction of this with the lumbar enlargement (B). The second difference is that the sclerosis has rarely that special intensity in the root zone of the postero-external column which characterizes the lesion of tabes. In rare cases the whole posterior column in the lumbar region is diseased. The part of the external column near the commissure and near the neck of the posterior horn usually remains free. Sometimes the degeneration does not extend up to the posterior surface of the cord, as in the case figured, in which it affects chiefly the middle three-fifths of the posterior columns. When the degeneration is considerable, the posterior median columns, in the upper part of the cord, may present the usual ascending degeneration of secondary origin. When the lesion is slight in degree in the lower half of the cord, there may be only a slight diffuse degeneration of the columns in the cervical region, similar to that elsewhere, and not the more intense limited affection of the median part, which occurs when there is a considerable lesion of the column lower down, and characterizes the secondary form.

The degeneration in the lateral columns is also variable in extent and position, and is not often "systemic" in character, *i.e.*, does not correspond to a single system of fibres. In one or two cases, indeed, the whole pyramidal tract, and this alone, has been degenerated,

FIG. 121.



Ataxic paraplegia, combined lateral and posterior sclerosis. A, upper dorsal; B, last dorsal; C, mid-lumbar. The posterior columns are free from sclerosis in C, except in their anterior parts; in B the disease involves the middle and anterior parts of both post.-med. and post.-ext. columns except in the neighborhood of the neck of the horn; in A the sclerosis is slighter and is confined to the middle three-fifths of these columns. The sclerosis of the lateral columns in C is limited, on the right, to the pyramidal tract, on the left it extends in front of this; in B it is very dense in the whole lateral column, involving not only the pyramidal tract but the limiting layer, and part of the "mixed zone;" in A it is similar in extent but slighter in degree. There is some increase of tissue throughout the anterior columns, and a focus of sclerosis near the anterior fissure, on the right in A and B, on both sides in C.*

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* I am indebted to Mr. J. Hopkins for the opportunity of drawing these sections. The patient was a man aged twenty-one, in whom weakness of the legs commenced at twenty, after a wetting, improved, and then slowly increased. He was admitted a year after the onset, with considerable weakness of the legs, a reeling, unsteady gait, increased knee jerk, foot clonus, but with no anæsthesia, wasting of muscles, pains, or affection of the arms. The symptoms slowly increased, paraplegia became absolute, and the spasm very intense, sometimes flexor and sometimes extensor. There was a doubtful impairment of sensibility to touch on the legs, but no loss to pain. The sphincters became affected, bedsores formed, and the patient died two years after the onset. Fuller details will be found in *Brain*, October, 1883, p. 383.

but more often the sclerosis, while intense in the pyramidal tract, also extends in front of this into the mixed zone of the lateral columns (Fig. 121, A, B). (A similar extension, it may be noted, is met with in the sclerosis that attends degeneration of the anterior cornua; compare Fig. 119.) The lateral limiting layer, between the pyramidal tract and the gray matter, may be also invaded. The direct cerebellar tract often escapes, as in Fig. 121, A (B is below its level of origin). But in some cases there is a zone of sclerosis round the periphery of the cord, extending deeply into the lateral column in the position of the pyramidal tract. The latter has been found diseased, in some degree, in all cases, and in several the extent of its degeneration has been greater in the lower part of the cord than in the upper. As in most other degenerative diseases of the cord, there may be some increase of connective tissue in the unaffected columns, and it may be so intense at some spots as to constitute areas of distinct sclerosis, such as are represented near the anterior median fissure in Fig. 121. As a rule no morbid change has been recognized in the gray matter or in the membranes. In one case only was there slight meningitis. Disease of the ascending root of the fifth was found in a case in which there were corresponding symptoms. The peripheral nerves seem not to have been examined in any case. The muscles have been found normal.

PATHOLOGY.—The double lesion in the posterior and lateral columns supplies an explanation of the two sets of symptoms which characterize the disease. The incoördination is doubtless due chiefly to the disease of the posterior columns of the cord. There is an obvious analogy between the symptoms and lesion in this disease and in true tabes. But there are also differences between the two diseases in this particular. We have seen that in tabes the ataxy is probably due chiefly to the disease of the sensory muscle nerves. In ataxic paraplegia, to judge by the general integrity of sensation in the legs, and by the preservation of myotatic irritability, the sensory nerves escape, and among them those of the muscles. In correspondence with this integrity of sensation, the root zone in the lumbar region scarcely ever presents dense sclerosis, and is often free from degeneration. Hence one element in the production of incoördination in tabes is absent in ataxic paraplegia. But we have seen that, in the former disease, other mechanisms are influential in causing this symptom, and these are doubtless effective in the malady we are now considering. The considerable disease of the posterior columns in the dorsal region, and the occasional degeneration of the direct cerebellar tract, may arrest conduction upward from the muscles to the cerebellum. As we have seen, it is probable that the direct cerebellar tract conducts impressions from the lower trunk muscles, and that it is also probable that most of the fibres of the posterior median columns have a similar function, conducting from the muscles of the legs impressions which reach the cerebellum through the connection with it of the post-pyramidal nucleus of the medulla. If the impressions thus conducted guide cerebellar coördination, we can understand that ataxy may result from the interruption of this path. That interruption of some conducting path in the cord will produce incoördination is proved by the occurrence of this symptom in cases of unilateral lesion of the cord, on the side of the lesion (see p. 166, note). Thus the chief difference between the two diseases, so far as concerns the effect of the lesion which causes the incoördination, is that in tabes it arrests the muscle-reflex action. The sclerosis in ataxic paraplegia occupies also the region of the cord in which the short vertical fibres run, connecting the posterior gray matter at different levels, and it is possible that the damage to these fibres may contribute to the ataxy.

The only recognizable lesion that can be regarded as the cause of the paralysis is the degeneration of the pyramidal tract. This is, indeed, so variable in degree in different cases, and in some is so small in proportion to the degree of palsy, that it has been questioned whether this lateral degeneration is the sole cause of the paralysis. But no other lesion has been found; the excess of myotatic irritability and ultimate spasm are conclusive evidence that the cause of the palsy is in the upper segment of the motor path, *i. e.*, above the ganglion cells of the anterior cornu (see pp. 130 and 154), and when the paralysis has been greater in one leg than the other, the disease of the lateral column has been found to be greater on the corresponding side. It is possible, however, that the degeneration is often more extensive in the termination of the upper segment in the gray matter, than in the (pyramidal) fibres of this segment, and that the occasional discrepancy between the amount of degeneration of the lateral column and the amount of paralysis may be susceptible of the same explanation as in pure spastic paraplegia (see p. 325). In most cases that have been examined, the visible disease was greater in the lower than in the upper part of the cord, *i. e.*, greater in the lower than in the upper part of the segment, increasing from above downward. This suggests that the maximum lesion is at the *lowest* part—in the terminal ramification within the gray matter. Degeneration here would not necessarily cause any secondary change in the white columns, *i. e.*, in the part of the segment above its termination, while it would explain all the paralytic and spastic symptoms of the disease.

The differences between this disease and tabes depend therefore on the escape of the sensory nerves, both in their peripheral course and in the posterior column of the cord. It is on the integrity of the reflex arc that the preservation of myotatic irritability depends, not on the presence of a lesion in the cord tending to increase this reflex action. If the knee jerk is lost as in tabes, it is not restored by degeneration of the pyramidal tracts, whether beginning in the cord or descending from the brain. Hence this difference from tabes depends partly on the escape of the peripheral nerves, partly on the difference in the distribution of the sclerosis in the posterior columns, by which the root fibres are damaged in one case and escape in the other.* It is not surprising, however, that intermediate cases should be met with, in which these differences are not present; in which the sclerosis extends to the root zone, and lightning pains, and some loss of sensation, show that the fibres of the posterior roots from the skin are involved in some degree. Even in these cases, however, the knee jerk is not lost; if it is absent there is no spasm and the cases are then of a different character; they are cases of tabes with secondary lateral sclerosis. It is in the remarkable facts that, as a general rule, if the knee jerk is not lost early it is not lost at all, and that if it is lost early, spasm does not come on, that we have the justification for the separation of these cases from tabes.*

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* We have seen (p. 145) that the fibres that subserve reflex action cannot be the same as those that ascend in the posterior median column, and which, in this disease, are often damaged higher up the cord.

It has been doubted whether the disease is to be regarded as a system degeneration, in the strict sense of the word. The lesion of the lateral columns certainly, as Westphal has insisted, does not correspond strictly to the pyramidal tracts, and yet its cervical extent forbids us regarding it as focal in nature. The disease is certainly allied to system diseases, and its claim to be considered such may perhaps be strengthened by future observations as to the elements which compose the more extensive degeneration,† but its claim to separate distinction is at present, as we have seen, clinical rather than pathological, is based on the common uniformity of the range of symptoms, alike in different cases, and in the same case throughout its course.

Diagnosis.—The diseases from which ataxic paraplegia has to be distinguished differ according to the stage of the disease at which the diagnostic problem presents itself. The dominant symptom in the early period is the incoördination; in the later period, the spastic palsy. Hence the disease in the early stage is liable to be confounded with pure locomotor ataxy. In discussing the relation of the two diseases we have already considered their differences. Of these the most important is the condition of the knee jerk, which is lost in the one, excessive in the other. In the rare cases of early tabes in which it is not lost, it is never increased. The presence of a clonus is also exclusive of pure tabes; and so also in the extensor spasm, of which we may often observe some indications, while power is still only slightly impaired. Other differences may confirm the diagnosis, but are never necessary to make it, and are indeed not uniform enough to be alone conclusive. In the state of sensation on the limbs, in the occurrence of, or freedom from, pains, and in the action of the iris, the two diseases present a contrast in the vast majority of cases, although not invariably. From primary spastic paraplegia the diagnosis depends on the presence of incoordination, or in a history of it if voluntary power has become too slight to be susceptible of derangement. Ataxic paraplegia is spastic paraplegia, plus incoördination. If the ataxy is considerable, as it often is, the distinction is easy enough, for experience shows that it is chiefly with tabes that such cases are confounded. The so-called "hereditary ataxy" presents a close resemblance to ataxic paraplegia, and is indeed, as we shall see, intermediate between this disease and true tabes. It is distinguished by its occurrence in several members of the same family, by the common loss of the knee jerk, and by the presence of nystagmus and of some impairment of articulation.

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* If cases occur in which an ultimate extension of the sclerosis of the posterior column abolishes the knee jerk and the spasm, it is quite certain that such cases are too rare to invalidate the grounds of distinction. The possible combinations of morbid processes are only limited by arithmetic and incompatibility, but this does not stultify our distinctions.

† For instance, it may be found that the degeneration in front of the pyramidal tract is of an association system of short vertical fibres, and that the annular sclerosis is of the direct cerebellar and antero-lateral ascending tracts.

The greatest diagnostic difficulty is presented by cases which have a subacute onset, and the distinction from a local myelitis involving posterior and lateral columns may be very difficult. It depends chiefly on the progressive tendency of the symptoms, which contrast with the regressive tendency of myelitis. It is possible that when there is a degenerative tendency, a focal myelitis may set up a progressive degeneration in the columns concerned, as it certainly may set up a degeneration limited to the posterior columns.

A tumor in the middle lobe of the cerebellum may cause unsteadiness, closely resembling that of some cases of ataxic paraplegia, and it may also cause weakness of the legs with increased knee jerk, from the pressure on the pyramidal fibres as they pass though the pons. In such cases, indeed, we probably have the two elements of the disease, produced although in a different manner by a lesion of different position. We have a degeneration of the lateral columns which is secondary instead of primary, and we have disease of the coördinating centre instead of interruption of the path to it. But the weakness in the legs is never great in cerebellar tumor, and special symptoms of this are never absent. Occipital headache, vomiting, and optic neuritis are present in most cases. In a case in which the pons is compressed, some cranial nerves are usually also damaged.

Prognosis.—So far as the prospect of recovery is concerned, the prognosis in ataxic paraplegia is unfavorable. In a few cases I have seen considerable improvement; and arrest is occasionally obtained, but in the majority of cases the progressive tendency of the disease baffles all attempts to check it. The downward progress of most cases is very slow, far slower than might be inferred from the history of recorded fatal cases, and the danger to life is small.

Treatment.—The treatment of ataxic paraplegia is the same as that of the allied diseases, and this need not be here repeated. The remedies most useful in tabes also most frequently do good in this disease. In the later period, in which spasm has attained considerable intensity, the treatment necessary is that of the condition described in the preceding chapter. I have known a long course of Turkish baths, with thorough rubbing, in more than one case to be of marked service, and distinctly contribute to the improvement that occurred. There are no symptoms peculiar to ataxic paraplegia that require special treatment.

"HEREDITARY ATAXY."

FRIEDREICH'S DISEASE, HEREDITARY ATAXIC PARAPLEGIA.

The so-called hereditary ataxy is a form of ataxy, or rather of ataxic paraplegia, usually depending on combined posterior and lateral sclerosis, which occurs in families, and differs also from the common forms of tabes and of ataxic paraplegia, in the early age at which it always commences, and in the presence of certain additional symptoms. It is often termed "Friedreich's disease," because this physician first described the characteristic features of the malady and the lesion in the posterior columns.* About sixty-five cases have been hitherto recorded.†

The dependence of the disease on a congenital tendency is clearly shown by its usual occurrence in families. But direct inheritance has been traced in only a few instances. In one, the mother of the family affected, and *her* mother, both suffered from the disease. That a general neuropathic inheritance is also sometimes effective is shown by cases in which there is a history of other neurotic diseases, such as insanity, in collaterals or ancestors. Alcoholism in parents has been supposed to be influential, but the evidence of this is not strong. Consanguinity of parents has existed in some instances, and has doubtless intensified the morbid tendency in these cases.

The family tendency of the disease is shown by the affection, in most instances, of brothers and sisters. The sixty-five cases were distributed in nineteen families, giving an average of rather more than three to each. The number affected in one generation has varied from two to eight. In the case in which the mother and grandmother suffered, an uncle and seven children were also affected, making ten in one family. Scarcely any isolated cases have been recorded; whether this is because they do not occur, or because the nature of the disease has not been recognized, is uncertain. The two sexes present nearly equal liability; males slightly preponderate (thirty-five males to thirty females). In some families the two sexes have suffered equally, but in others the disease has shown a marked tendency to affect one sex. Thus in one family of nineteen, the two males suffered and the seventeen females escaped. In another instance, recorded by Musso, a brother and sister (the offspring of a melancholic mother) married healthy individuals; the brother had three daughters affected out of seven living children; and of the sister's children three sons were diseased. Another curious circumstance is that three of the brother's and four of the sister's children were born dead.

The age at which the first symptoms are recognized has varied between four and twenty-four years.‡ The seventh and eighth years of life are those

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*Friedreich's first account was given to a medical society in 1861, and published in *Virchow's Archiv* (Bd. 26 and 27), in 1863. Further cases were published by him in 1876 (*ib.*, Bd. 68 and 70).

† The most complete list of these (57 cases) has been given by Dr. Everett Smith, *Boston Med. and Surg. Journal*, Oct. 15th, 1885.

‡ In the family recorded by Everett Smith, the father presented symptoms of ataxic paraplegia at the age of sixty-six, coming on gradually after an attack of rheumatism produced

in which disease most often begins, and next comes the period of puberty, from twelve to sixteen. It shows a slight tendency to begin earlier in males than in females, and often a marked tendency to commence about the same period in the same family. Immediate causes can rarely be traced.

SYMPTOMS.—The first and chief manifestation of the disease is a gradual impairment of coördination, first in the legs and afterward in the arms. Initial pains scarcely ever occur. The ataxy is shown by unsteadiness in standing and walking, at first slight, but slowly increasing, until the feet have to be placed wide apart in standing, and the patient reels in walking like one under the influence of alcohol. The feet are not often raised too high, unless when an unusually long step is taken. Closure of the eyes causes a considerable increase in the unsteadiness in some cases; in others it has but little influence. The impairment of movement in the arms is of a similar character, but usually commences some time after that of the legs. There is irregularity in the voluntary movement of the arms and fingers, and the ataxy has often a distinctly jerky character. In most cases the power of the muscles is at first unimpaired, their nutrition is good, but the myotatic irritability is lost. In exceptional cases, as in one recorded by Seligmuller, it has been preserved and increased. In most cases the knee jerk has been found absent as soon as the patient came under observation; in one case it disappeared after the other symptoms had set in.

As the disease progresses, some jerky irregularity develops in the movements of the neck and head, so that the head presents slight, unsteady movements, sometimes like an irregular tremor, when it is supported by the muscles. Speech is also impaired; syllables are elided, and there may be, with this, an occasional hesitation. There is no twitching of lips, but occasional jerky movements have been noted in the tongue. The affection of speech is not often an early symptom. It may not be noticed until three, five, or ten years after the onset of the other symptoms. In most cases (but not in all) there is nystagmus when the eyes are moved laterally, due clearly to jerky unsteadiness in the action of the muscles, corresponding in the two eyes. Sometimes the movement is slower than in most forms of nystagmus, and it is rarely present when the eyes are at rest, directed straight forward. This symptom also usually comes on after those in the limbs. Paralysis of the ocular muscles does not occur, nor does optic nerve atrophy.

Sensory symptoms are very variable. Lightning pains and any severe pains are extremely rare, but slight dull or rheumatoid pains in the legs are not uncommon. Sensibility has been quite normal in many cases; in others there has been slight anæsthesia in the legs, early in some instances, late in others. Sensibility to pain and temperature is very rarely impaired. Sensation may, however, be unaffected even when the ataxy and weakness have become extreme. The sense of posture of the limbs has been found normal

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by exposure to cold. In the age of the sufferer, this case stands alone in the history of the disease, and is not included in the figures given. The case may be a mere coincidence, or may show that a latent predisposition may persist through life.

in several cases in which it has been examined. The electro-sensibility of the muscles is said to be sometimes lessened. Increased sensitiveness to pain is occasionally met with. Reflex action from the sole is usually preserved, but may be lost when there is anæsthesia. The cremaster reflex is often lost. On account of the age of the patients little is known of the condition of sexual power, but it is certainly often lost. Menstruation becomes irregular and ceases. The sphincters, as a rule, are unaffected. There is no tendency to trophic changes in the skin or joints.

Although muscular power is commonly normal at first, and may be normal even when the ataxy is considerable, it usually becomes impaired as the disease progresses, and sometimes weakness comes on, with the ataxy, at the onset of the disease. The loss of power is always greatest in the legs and may be confined to them. The flexors suffer more than the extensors. It may be ultimately great, although it rarely amounts to absolute paralysis. Slight wasting of the muscles may occur in the later stages of the disease, but is only attended with trifling depression of the electrical contractility. Lateral curvature of the spine, and talipes equinus, or equino-varus, of the foot, have been observed as later results of the muscular weakness, developing under the influence of posture, and contraction of the flexors of the knees has also resulted.

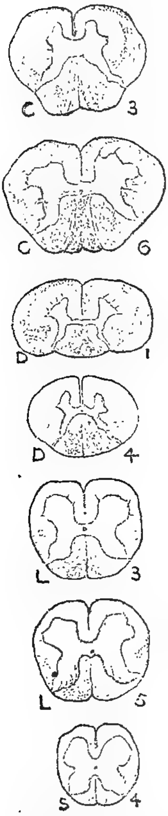
Visceral crises do not occur. Frequency of pulse has been noted, however, in many cases, and curious vasomotor symptoms (flushing, œdema, sweating, polyuria, and salivation) were present in one of Friedreich's cases. There is no mental change that can be regarded as part of the disease. Imbecility coexisted in one case (Power).

The rate of progress of the malady varies much, even in different members of the same family. One in whom the disease commenced last may be unable to walk, while another who suffered sooner may be still in the early stage of the disease. Now and then the disease is stationary for many years. The duration of the malady is correspondingly variable. It is always long, even more than thirty years, and in many instances it has not apparently shortened life. On the other hand, death may occur at the end of ten or twelve years. The end has generally come from intercurrent affections, rarely from exhaustion. An attack of acute myelitis ended life in one of the cases recorded by Everett Smith.

Pathological Anatomy.—The lesion in hereditary ataxy is that of ataxic paraplegia and of tabes combined. There is degeneration in the lateral column, often also in the anterior column, such as occurs in ataxic paraplegia, but the sclerosis of the posterior column is more intense than in that disease, and it is also more extensive, especially in the lumbar region. It is similar to the degeneration of tabes, and agrees with this, also, in that the posterior nerve roots are usually affected, whereas in ataxic paraplegia they almost invariably escape.

The distribution of the lesions shows a close correspondence in different cases, as will be seen by a comparison of the figures here given, which are from three different cases of the disease. The sclerosis of the posterior columns may be complete throughout the cord, with the exception of a narrow band near the neck of the cornu, which always remains but little damaged. In the lumbar region it is usually intense up to the head of the horn and the posterior surface of the cord. Occasionally, however, it is somewhat less intense in the lumbar region than it is higher up, extending, however, into the root zone. In the cervical region the sclerosis may also be general, or it may be greatest in the root zone and in the posterior median columns. The cervical root zone never escapes, as it often does in tabes.

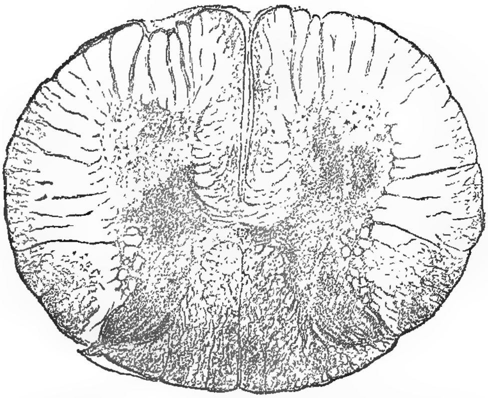
FIG. 122.



Hereditary ataxy; distribution of degeneration in the white columns, indicated by the dotted shading. (*After Friedreich.*)

The degeneration of the lateral columns always involves the pyramidal tracts, and is most intense in their position. It is not, however, limited to them. It usually extends outward to the periphery of the cord, where the pyramidal tract does not reach the surface, thus involving the direct cerebellar tract, and it also extends forward at the surface, so as to constitute a band of annular sclerosis, which varies in thickness in different parts of the cord and in different cases. In the inner part of the anterior column there

FIG. 123.



Hereditary ataxy; section of spinal cord at first lumbar segment. Sclerosis of the whole of the posterior columns except a narrow zone adjacent to the neck of the horn. Degeneration also of the lateral pyramidal tracts, and, in front of this, slight degeneration in the superficial layer of the lateral column. Adjacent to the anterior median fissure there is also a zone of sclerosis of the anterior pyramidal tract which extends, in this cord, into the lumbar region.*

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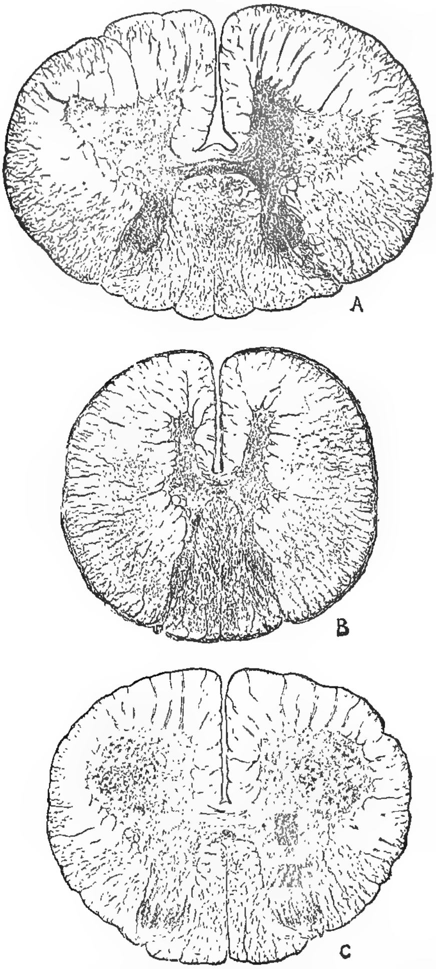
* I am indebted for this section to Dr. Everett Smith, who has published the case in the *Boston Med. and Surg. Journal*, Oct. 15th, 1885. The patient was one of three sisters affected with the disease, the brothers being healthy. The father suffered from ataxic paraplegia late in life, as mentioned on p. 344. In the case from which the drawing is made, the incoördination began in the legs at the age of nineteen, and in the arms soon afterward, and was quickly followed by loss of power, which gradually increased to almost universal paralysis, with muscular contractures (talipes equino-varus, etc.), and considerable loss of sensation. There was nystagmus, affection of articulation, and some mental dullness. Death occurred at the age of forty.

may be a distinct area of degeneration in the position of the anterior pyramidal tract. This is seen in Fig. 123, in which this tract extends unusually low in the cord, and is distinctly degenerated in the upper part of the lumbar enlargement. Atrophy of the posterior vesicular column has been found associated with that of the direct cerebellar tract. Slight abnormal appearances have been seen in the nerve cells of the anterior cornua, in some cases, but, as a rule, these are normal, and the gray matter presents no other disease. It is very likely, however, that future observations will reveal the occurrence of changes in the nerve cells of the posterior horns, such as are met with in tabes.

Fig. 124.



Hereditary ataxy. A, cervical; B, dorsal; C, lumbar regions of the cord. The posterior columns are sclerosed in their whole extent, except in the vicinity of the neck of the horn in the cervical and lumbar regions, the degeneration being rather less intense in the lumbar than in the other parts. In the antero-lateral columns there is an annular zone of sclerosis in the periphery of the cord, widening in the region of the pyramidal tract. In front of this tract the zone is widest in the dorsal region, and trifling in degree in the lumbar. The gray matter is but little affected.[†]

The pia mater over the posterior columns has been found thickened. The posterior nerve roots usually present some degeneration of their fibres; this may be partial (Fig. 125, B), or may amount to total destruction of the fibres (as at C). The peripheral nerves in the limbs have been found normal.[*]

Some general shrinking and induration of the pons and medulla were found by Friedreich, with atrophy of the cells of the post-pyramidal nucleus, and some degeneration of the restiform bodies, but none of the anterior pyramids. Corpora amylacea were present in the hypoglossal nerves.

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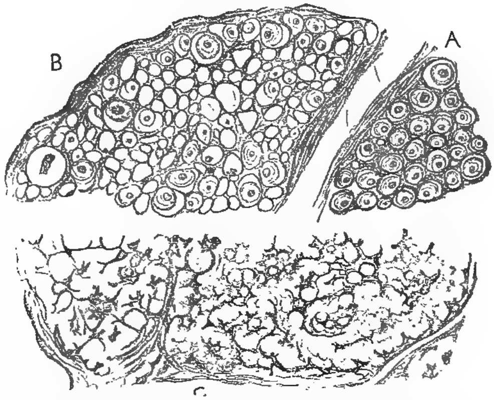
[*] Friedreich, *Virchow's Archiv*, Bd. 70, p. 145.

[†] For the sections from which the drawing is made I am indebted to Dr. Pitt (who prepared them) and Dr. Moxon, under whose care the patient died in Guy's Hospital. The case (full details of which will be found in the current volume of the *Guy's Hosp. Reports*) is one of a series of five cases (four brothers and a sister) which I reported some years ago to the Clinical Society (*Clin. Soc. Trans.*, vol. xiv, 1881, p. 1).

Pathology.—The disease apparently occupies a clinical and pathological position between the combined sclerosis described as ataxic paraplegia and simple tabes, resembling the former in the common affection of the lateral columns and the consequent weakness, but differing from it and resembling tabes in the degree of affection of the posterior columns in the lumbar regions and the affection of the posterior roots; with these the loss of the knee jerk and the affection of sensation are apparently connected. Isolated cases presenting the same combination of spinal symptoms are occasionally met with. The precise origin of the affection of articulation and of the nystagmus has not yet been traced.

Fig. 125.



Hereditary ataxy; degeneration of posterior nerve roots (from the same case as Fig. 124). A, normal anterior root, for comparison. B, posterior root, partially degenerated; a few normal nerve fibres are seen, but most of the spaces which should contain nerve fibres are empty. C, posterior root, totally degenerated, probably by a more acute process than that of B, since there is some destruction of the septa and increase of amorphous connective tissue. The products of degeneration are rendered invisible by the mode of preparation. (Compare C with the similar changes in a totally degenerated peripheral nerve shown in Fig. 51, B, p. 111.)

From the age at which the disease commences, long before the period at which ordinary degenerations occur, we must look for its ultimate cause to a congenital tendency of development, by which the affected elements have a briefer period of vital endurance than the other tissues of the organism. Mobius has suggested that there is an actual arrest of development of these structures, but it is doubtful whether this assumption is warranted by the facts.

Diagnosis.—In most cases the incoördination is sufficiently predominant to suggest that the disease is a form of ataxy, and the unsteadiness of the head, the affection of articulation, the nystagmus, and the age at onset, suffice for the distinction from ordinary tabes. The common form of ataxic paraplegia bears a close resemblance to this disease, a resemblance that is more than superficial, although the excessive knee jerk and foot clonus, almost always present in ataxic paraplegia, are usually absent in hereditary ataxy, and nystagmus is absent in the former. The difference in the state of myotatic irritability is, as we have seen, not absolute. Cases of combined lateral and posterior sclerosis occur, of tabetic type, in which the knee jerk is lost, and in at least one case of hereditary ataxy the knee jerk has been excessive. The age of onset and family multiplicity may decide the question, but isolated cases occur, commencing soon after puberty, scarcely distinguishable from the hereditary disease. Such a case has been recorded by Kahler and Pick, which has been regarded by some, and not by others, as an example of this disease.

Disseminated sclerosis presents incoördination, nystagmus, and impaired articulation, but the ataxy of the arms differs in the wide range and violent character of the disordering jerks which characterize this disease; in the legs, moreover, simple unsteadiness is very rare. The affection of speech differs from that of hereditary ataxy in being simply "staccato," with undue separation of syllables, which are rarely run together. The cases are always isolated.

Cerebellar tumor and hereditary ataxy can hardly be confounded, in spite of the fact that the unsteadiness in walking is very similar in the two diseases; the common affection of the arms in the one, and the conspicuous head symptoms (severe pain, optic neuritis, etc.) of the other, sufficiently distinguish them.

PROGNOSIS.—The prognosis in every case is very serious, since the disease is essentially progressive, but life may be prolonged for many years, and in slight cases the malady may interfere comparatively little with the patient's occupation. Thus I have known a man, with very marked symptoms, follow his business as a tradesman for many years. The only guide to individual prognosis is the observed rate of progress.

TREATMENT.—As in other diseases that depend on a congenital tendency, treatment is almost powerless. The measures recommended for ordinary locomotor ataxy (apart from those suggested by the syphilitic relation of tabes) are those most suitable to the hereditary form. Arsenic and nitrate of silver especially deserve a trial; now and then they seem to check the progress of the disease for a longer or shorter time, but its progressive tendency ultimately reasserts itself.

CHRONIC SPINAL MUSCULAR ATROPHY.

PROGRESSIVE MUSCULAR ATROPHY; WASTING PALSY; AMYOTROPHIC LATERAL SCLEROSIS; CHRONIC POLIO-MYELITIS.

The disease which has long been known by the names "progressive muscular atrophy" and "wasting palsy" is characterized by slow wasting of the muscles, beginning in some one part, and usually spreading and increasing, until it is wide in extent and extreme in degree. The changes in the muscles depend on changes in the spinal cord, a slow degeneration of the ganglion cells of the anterior cornua, accompanied by a similar degeneration in the motor nerve fibres which spring from the cells. With this degeneration of the cells and peripheral fibres, there is usually also associated a degeneration of the pyramidal tracts in the cord.

Chronic muscular atrophy was separated from primary paralysis by Sir Charles Bell, and was afterward described more fully by Aran and Cruveilhier. Bell and Cruveilhier regarded it as a spinal disease. Aran (who first called it "progressive muscular atrophy") thought that it was primarily a disease of the muscles, a view that was afterward held by Duchenne, and subsequently, also, by Friedreich. This opinion was supplemented by a theory that the wasting was due to a disturbance of the sympathetic. Lockhart Clarke first discovered the disease of the gray substance of the spinal cord, and Charcot specially associated the atrophy with the wasting of the ganglion cells. When the constancy of the spinal lesion was demonstrated, it was thought that every form of chronic muscular atrophy was of spinal origin, but this view has proved erroneous, and it has been shown that there are forms of muscular atrophy which are purely local and idiopathic. The morbid process is not confined to the spinal cord. Atrophy of the nerve cells from which the motor bulbar nerves arise often accompanies that of the spinal cells, and "progressive bulbar paralysis" is then associated with "progressive muscular atrophy." Moreover, the degeneration of the pyramidal fibres can often be traced through the brain.

Two varieties of the disease have been distinguished by Charcot, who has been followed in this by most subsequent writers. In the one variety the disease is manifested only by the wasting of the muscles; in the other there are, in other parts than those much atrophied, the indications of degeneration of the pyramidal tracts of the cord. In the latter cases, Charcot regarded the degeneration of the pyramidal tracts as the primary lesion, and the affection of the cornua as secondary. He therefore termed the cases of the first class "protopathic," and those of the second class "deuteropathic," and gave to the latter the name "amyotrophic lateral sclerosis." In Germany, however, and especially by Leyden, doubts have been expressed as to the validity of the distinction between the two classes of cases, and as to the sequence of the lesions in the second class. These doubts are not without foundation. The subject will be discussed when the pathology of the disease is considered, and reasons will then be given for the course here adopted of considering the two varieties together, as essentially one disease.

Causes.—The disease is more frequent in males than in females, the proportion being about three to one. It is chiefly a disease of adult life, commencing usually between twenty-five and forty-five. I have known it to begin at fourteen and as late as seventy, but most cases of muscular atrophy in early life are idiopathic, and not spinal. Heredity is to be traced only in less than half the cases, and generally as an indirect neuropathic disposition. Rarely there is direct inheritance of the disease. Among instances that I have met with are a lady whose mother died from a similar atrophy, and another case (from which Fig. 135 is taken) in which a brother had died from some chronic disease of the cord, attended with wasting. When many members of a family suffer from muscular atrophy, the malady is nearly always idiopathic and not spinal. The affection occurs in all classes of society, and it is doubtful whether workers with the muscles furnish a larger proportion of the cases than can be accounted for by their greater exposure to the certain exciting causes.

Of these more direct causes one of the most frequent is mental distress and anxiety, and this is especially met with in females. A severe fright has been thought to produce it. Another cause is exposure to wet cold, which is a cause, also, of many other chronic spinal diseases. Sometimes the exposure has been habitual; sometimes a single exposure has been the cause, and some neuralgic pains, indicative of the deleterious influence on the nervous system, have followed the exposure and connected it with the later wasting. The pain has been sometimes in the part afterward wasted, sometimes in some other more common seat of neuralgia. Thus one patient, after remaining in wet clothes for twelve hours, suffered for six weeks from severe neuralgia in the left side of the face, and then the muscles of the left shoulder began to waste. Although excessive use of individual muscles may cause them to waste, it is doubtful whether this influence produces genera muscular atrophy. Injury to the cord, such as results from concussion of the spine, is a rare cause. It more often causes disseminated myelitis, which may be manifested by muscular wasting combined with other symptoms. In a few instances I have known progressive atrophy, of typical characters, to slowly follow a concussion, as if this had set up a perversion of the nutrition of the nerve elements. Still more rarely a fall, injuring one limb, has been followed by muscular atrophy commencing in this limb and becoming general. Thus a woman fell downstairs, and pitched on her left hand and wrist; she had pain in the arm for a long time, and two years after the fall this arm began to waste and the atrophy ultimately became general. The relation might be passed as an accidental coincidence, were it not that in other central diseases, such, for instance, as paralysis agitans, the symptoms may commence in an injured limb.

The disease sometimes succeeds syphilis, and no other cause may be traceable. As with other degenerative diseases, an interval of years elapses between the primary disease and its nervous sequel. The cases that I have seen after syphilis have been typical in course and evidently degenerative in nature. It is probable that syphilis has some share in the causation of these cases, because the same relation obtains in the case of other diseases of the same class, and especially in that of locomotor ataxy. Occasionally, syphilis and a neurotic heredity can both be traced. In rare cases the disease has followed acute specific diseases, especially measles, as in a remarkable group of cases, in one family, recorded by Ormerod.* General muscular atrophy may result from lead poisoning, but this form is not, as a rule, progressive in character when its cause has ceased to act. In many cases of progressive muscular atrophy no cause for the disease can be traced.

SYMPTOMS.—The definite symptoms of the disease are often preceded by aching pain in the part afterward wasted, rarely severe. Sometimes there is such pain in the spine or elsewhere, especially, as already stated, in the cases that are due to exposure to cold. Rarely some sensation other than pain precedes the local symptoms. Thus in one case a sense of coldness preceded the atrophy in each limb that was attacked. Weakness and wasting usually come on together; sometimes one and sometimes the other first attracts the attention of the patient. In the upper part of the arm the loss of power is usually first noticed, and in such covered parts the wasting may become considerable before it is discovered. In the hand the wasting is often first noticed, but sometimes it is the impairment of some delicate action, such as writing, that draws attention to the part. The affected muscles lose their proper shape, and there is flattening, or even a depression where there should be a prominence. If a patient is fat, however, the wasting may cause at first very slight alteration in external aspect.

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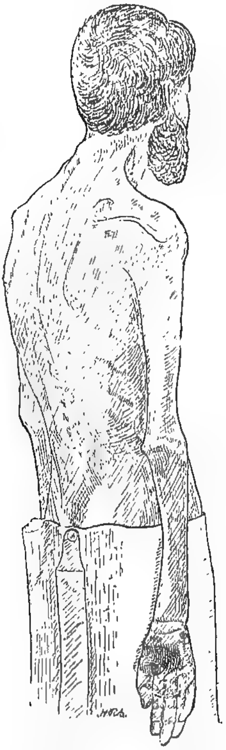
* *Brain, 1884*, p. 334.

The disease commences in the arms in nine-tenths of the cases, and as frequently in one arm as in the other. It begins with almost equal frequency in the hand and in the shoulder muscles. From the part first affected the disease spreads to other parts of the same limb. Before it has attained a considerable degree in one limb it usually shows itself in the corresponding limb on the other side; often in the muscles corresponding to those in which it commenced, sometimes in those affected second in order of time. As the muscles waste, the voluntary power is lessened, and paralysis results of various character and degree, corresponding to the atrophy. In the hand, the thenar muscles and interossei are usually the first to suffer, sometimes one, sometimes the other, but both are soon involved in most cases. The thenar eminence becomes flattened; the base of the first metacarpal bone becomes distinct and even prominent. Of the interossei, the atrophy of the abductor indicis is especially conspicuous; the normal prominence gives place to a hollow beside the metacarpal bone when the thumb is abducted. Depressions form between the metacarpal bones on the back of the hand, and also between the flexor tendons in the palm in consequence of the wasting of the lumbricales. The forearm muscles may be next involved, the flexors usually before the extensors, and with the flexors the supinators may suffer or they may escape until the biceps is involved. Occasionally the disease begins in the forearm, and then especially in the extensor muscles. The several parts of the long flexors or extensors may suffer unequally. Of the extensors, those toward the ulnar side usually suffer most. The extensors of the phalanges of the thumb are usually more affected than that of its metacarpal bone. Of the shoulder muscles, the deltoid is generally the first to manifest the disease, and in some cases which begin in the hand the deltoid suffers before the forearm muscles. The rounded contour of the shoulder becomes changed, and the head of the humerus can be recognized beneath the acromion. The wasting of the deltoid is soon followed by that of the other muscles of the upper arm and of the scapula. The triceps usually suffers less and later than the biceps, but sometimes the reverse is the case. The supra- and infra-spinati are often affected with the deltoid.

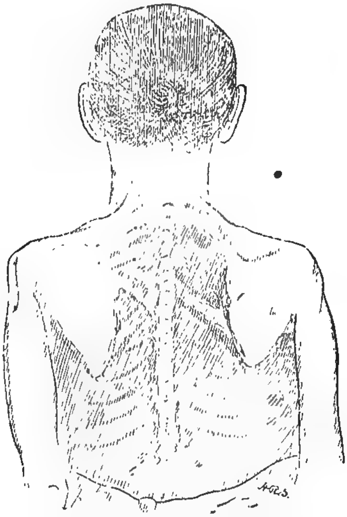
In most cases the wasting early involves the muscles of the back, and it sometimes begins in them. The middle and lower parts of the trapezius usually suffer first; the rhomboids and erectors of the spine at a later date. The affection of the trapezius is readily recognized if the patient tries to put the shoulder back. The highest part of the trapezius presents a remarkable indisposition to atrophy; it often remains intact to the last and then may contrast with the wasting below it, standing out, on each side, like a cord passing from the occiput to the shoulder. Hence Duchenne termed this part the *ultimum moriens.* I have, however, seen two otherwise typical cases (one beginning in the deltoids, the other in the hands) in which the highest parts of the trapezei suffered before the middle parts. The levator anguli scapulæ also generally escapes even when all the muscles about it are wasted. The serratus, latissimus, and pectoralis major are usually affected later. They may escape wholly or in part; isolated bundles of the pectoralis may waste, the intervening parts escaping, or either the upper or lower

FIG. 126.



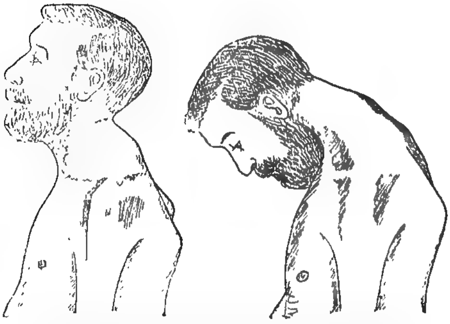
Progressive muscular atrophy. Wasting of the muscles of the back and arms; in the forearm scarcely any muscular tissue can be recognized, and in the hand all the muscular prominences have vanished. (*Drawn by Dr. H. R. Spencer.*)

FIG. 127.



Progressive muscular atrophy. Wasting of right trapezius complete (the rhomboids remaining), of the left trapezius partial. On each side the upper part of the trapezius is wasted, and the contour of the neck is correspondingly changed. Both deltoids are also atrophied. (*Drawn by Dr. Spencer.*)

FIG. 128.



Progressive muscular atrophy. Weakness of the muscles of the neck. (A) Habitual posture of the head inclined backward. (B) Position into which it falls if the patient attempts to keep it in the normal balance. (*Drawn by Dr. Spencer.*)

part of the muscle may atrophy alone. According to the affection of the muscles that are attached to the scapula, the position of the bone changes, and it becomes rotated under the influence of the muscles that are unaffected and unopposed. The muscles that extend the head on the spine often suffer in considerable degree, and from this there results a difficulty in the carriage of the head (Fig. 128). It is habitually inclined a little backward, so as to balance it on the spine with but little muscular exertion; if moved forward, or if the occiput is touched, it falls so that the chin touches the chest. It can be brought back into its former position only with difficulty; the patient has to incline the trunk backward, so as to bring the head nearly into the vertical position, and then, with a sudden contraction of the sterno-mastoids, and a jerk, the head goes back into its former posture. The increased innervation of the weak extensors of the head often causes a synergic over-action of the frontalis muscles, which are normally associated with the extensors (so that the eyebrows are raised when the head is put back to look upward). In such cases the wasting of the muscles at the back of the neck causes the skin to lie in transverse folds when the neck is extended. The patient may be unable, when lying in bed, to move the head from side to side. The sterno-mastoids also are often wasted; either the sternal or clavicular part may be most affected. In striking contrast to the general wasting of the neck is the condition of the platysma myoides, which always escapes, and may become hypertrophied in a vain attempt at compensation.

The muscles of respiration suffer in the majority of cases, and their impairment constitutes a grave source of danger to life. The intercostals rarely escape altogether; the diaphragm is involved in about one-third of the cases, and when it is attacked quickly loses all power, so that the respiration is carried on only by the intercostals and superior thoracic muscles. On the other hand, the intercostals may suffer much, and the diaphragm remain free; respiration is then purely abdominal and the walls of the thorax may be almost motionless in breathing, or there may be merely a slight movement of elevation of the upper ribs. The chest becomes flattened in front, and narrow from before back, from the influence of atmospheric pressure on the ribs, unopposed by the intercostals. Either the upper or lower intercostals may suffer most, with a resulting difference in the relative degree of movement of the upper or lower parts of the chest. In cases in which the diaphragm is paralyzed, the upper chest muscles often suffer more than the lower; there is then a compensatory over-action of the lower part, and the increased movement of the lower ribs, carrying forward the abdominal wall, may suggest that the diaphragm is acting when it is not. A careful examination will always prevent the error. The muscles of the abdominal wall occasionally waste, but far less frequently than those of the thorax.

Wasting in the legs is much less common than in the arms, and if it occurs is usually slighter in degree, but occasionally the disease first manifests itself in the legs, and is more intense in them than elsewhere. The glutei, extensors of the knee, and the muscles in the front and on the outer side of the lower leg, are those that are most affected. We shall presently see, however, that the legs are often paralyzed when they are not wasted, and sometimes they are the seat of wasting which differs in certain features from that which is the special characteristic of the disease.

The face almost always escapes the general wasting, and its normal appearance may present a striking contrast to the rest of the body. In many cases, however, the lips are paralyzed as part of the bulbar palsy that so often accompanies the spinal disease. A remarkable case in which the face shared the atrophy of the limbs, and the tongue escaped, has been recorded by Langer.*

As the wasting progresses, the appearance of the parts in which it is most advanced becomes extremely changed, All trace of muscle may disappear from parts of limbs or even from an entire limb, and there is usually a wasting of the adipose tissue as well as of the muscle, so that the bone seems to be covered only by fascia and skin. The transverse processes of the vertebræ may be felt in the hollow beside the spine, and the bony prominences about the shoulder may be almost as conspicuous as in a skeleton, so that beneath the acromion there may be a groove, into which the finger can be placed, in consequence of the descent of the head of the humerus from the glenoid cavity. The unequal affection of antagonistic muscles leads to various contractions and deformities. In the hand, especially, distortion is apt to occur; from the paralysis of the interossei and the contraction of the longer flexor and extensor muscles, the "claw-like hand" develops in extreme degree (Figs. 17 and 18, p. 52). If all the muscles of a part are equally and simultaneously affected, no deformity results unless an unsupported part yields to the influence of gravitation. Lordosis is very common in cases in which the trunk and hip muscles are involved. Its precise mechanism is not always clear, but it is certainly often the indirect result of the weakness of the extensors of the hip joint, in consequence of which the pelvis is unduly inclined forward, and carries with it the lower lumbar vertebræ; the upper part of the trunk has then to be carried far backward, to bring the centre of gravity within the base of support. The evidence of this mechanism is that the lordosis entirely disappears when the patient sits, and the pelvis, resting on the ischial tuberosities, is no longer inclined. This was the case, for example, in a woman with extreme lordosis from progressive muscular atrophy, in whom, when standing, a plumb line from the seventh cervical spine fell three inches from the sacrum and an inch and a half from the buttock. Sitting, her spine was perfectly straight.

The electrical irritability of the wasted muscles presents changes which vary in character in different cases. When the wasting is slow, there is usually a diminution in both faradaic and voltaic irritability, similar in character to each current. The irritability fails with the muscular nutrition, and when the wasting is great only a slight contraction can be obtained, even with a strong current. When the wasting is extreme, irritability at last becomes extinct, but the voltaic irritability of the muscular fibres persists longer than the faradaic irritability, and a strong voltaic current will often cause a slight contraction long after faradaism, in any strength that can be borne, fails to stimulate the muscle. In such cases, the quality of the voltaic irritability may be normal, but often there is a change in the order of reactions. ACC (anodal closure contraction) occurs as readily, or more readily, than KCC. Tetanic contraction during the passage of the current is produced with undue readiness, compared with the closure contractions, and opening contractions are often caused by currents but little stronger than those that cause closure contractions. Thus there is the qualitative, without the quantitative character of the reaction of degeneration (see p. 67). When the wasting is rapid, and especially when the weakness comes on more rapidly than nutrition fails, there may be considerable diminution of faradaic irritability, while that to voltaism is but little lower than normal, and in such cases the qualitative change is often well marked. Lastly, when there is rapid and considerable paralysis developing in the course of a few days, either at the onset or during the course of the disease, the palsy being followed by rapid wasting, there may be quick extinction of faradaic with actual exaltation of voltaic irritability, and the reaction of degeneration may be present in all its characteristics.

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* Meeting of the Vienna Medical Society, March 17th, 1882.

The mechanical irritability of the muscles is considerably increased; a tap causes a local contraction of the fibres struck. Moreover, spontaneous flickering contractions of parts of the muscles are very common, now of one bundle, now of another, conspicuous to the eye, although scarcely felt by the patient. This "fibrillation," as it is termed, is so frequent as to be characteristic, but it is not invariable, nor is it confined to this disease. It may sometimes be observed in muscles that are not yet invaded by the wasting, but where it is observed, atrophy usually follows.

In the parts affected by the characteristic wasting all reflex action is abolished, clearly in consequence of the damage to the motor part of the reflex arc; the afferent portion of the arc is unaffected, for, as we shall see, there is no loss of sensation. The myotatic irritability ("tendon-reflex action") is also lost, and lost early. The knee jerk, for instance, disappears as soon as there is even a slight diminution in the bulk of the thigh muscles, in cases in which the legs are the seat of primary wasting. The muscles are flaccid and toneless; a condition that may be conveniently termed "atonic atrophy." To this common rule, however, exceptions are sometimes met with; there may be from the first rigidity of the affected muscles. When this is the case, the wasting does not go on to the entire destruction of the muscle; it is often trifling, but may be considerable. With this rigidity there is a preservation of myotatic irritability. We may term this "tonic atrophy." There is thus a marked contrast in the two conditions; we shall presently consider the difference more fully.*

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* It should be noted, however, that in the muscles that are the seat of flaccid wasting,

Sensory symptoms are usually slight and always subordinate. They are confined to pain, usually dull and rheumatoid in character, felt chiefly in the limbs in which the malady is most active. Such pains are common at the onset, as we have already seen, and they may recur from time to time during the course of the disease. Sometimes vague feelings of "numbness" or "deadness" are complained of, but cutaneous sensibility is never impaired. (When anæsthesia accompanies muscular wasting both symptoms are due either to chronic meningitis, damaging the nerve roots, or to disseminated focal myelitis.) Nor do the muscles lose such sensibility as they normally possess. Perception of posture persists, and the muscles are not only sensitive to pressure, but are often more sensitive than in health, not only to pressure but also to extension.

When the arms are the seat of such atrophy as has been described, the legs, if not also wasted, may be normal, but they are often paralyzed without being wasted. The loss of power comes on slowly and gradually, and is accompanied by an increase of myotatic irritability. The knee jerk is excessive, a clonus can readily be obtained, and the reflex over-action of the muscles may increase to actual rigidity and spasm, so that there is the condition of spastic paraplegia described in a preceding chapter. It is rare, however, for the spasm to reach the higher degree of intensity, and for the muscles to present the massive firmness that characterizes the simple form of spastic paraplegia. On the other hand there is often some diminution in the size of the muscles, and thus we have a gradation to the condition of tonic atrophy just described. In this condition, moreover, there is usually only a moderate diminution in electrical excitability, which does not go on to extinction. The one condition does not seem to pass into the other; the rigidity and myotatic excess do not give place to muscular relaxation; atonic atrophy does not succeed the tonic wasting. In very rare cases, of which I have seen a few instances, there is a similar condition in the hands when the shoulders are the seat of simple atonic atrophy. The forearms are then rigid, moderately wasted, with myotatic excess, while the muscles of the shoulders are extremely wasted and absolutely flaccid. In still more rare cases, the whole arms are thus affected, are thin and rigid, and in no part is there atonic atrophy.

In one remarkable case of this character the muscles of the trunk participated in the spasm. After coughing or yawning respiration would be checked for a few seconds by general spasm, and whenever the patient was raised from the bed, the arms, legs, back, and neck became stiff in tetanoid rigidity, the head being bent backward.

The extreme emaciation of the most affected parts shows that the adipose

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when the atrophy has attained an extreme degree, slight rigidity may develop, accompanied by considerable tenderness of the muscle. It is probable that this rigidity is idiopathic, and due to the structural changes in the muscles, presently to be described. This condition, which should be distinguished from the initial rigidity, is rare; the early relaxation is usually maintained to the last.

tissue wastes as well as the muscles. The atrophied limbs are usually cold and may be livid or pale, but there is no tendency to acute vasomotor disturbance in the ordinary form of the disease. I have known the skin of the face to become thin and smooth, so that, in one instance, the dark iris could be seen through the closed eyelids, but such a change is quite exceptional. In another patient there was a very remarkable form of local atrophy. Certain areas of muscles underwent wasting, the rest being normal; the affection commenced by a livid discoloration of the skin, and the wasting seemed to involve the skin, subcutaneous tissue and muscle, causing local depressions. Considerable tracts of the trapezii were thus affected, and smaller spots in the arms and legs. The patient was a single woman of thirty-five, and the disease did not show a strongly progressive tendency.

The functions of the sympathetic are not, as a rule, deranged. Dilatation or contraction of one pupil has been frequently observed, chiefly in association with atrophy of the muscles that are supplied from the lower part of the cervical region, and no doubt depends on the disease of the spinal cord, and not of the sympathetic itself. The reflex action of the iris is usually normal, and optic nerve atrophy never occurs. I have once met with a remarkable reflex fixation of the eyeballs in a case of advanced progressive muscular atrophy. If the patient, looking to one side, was suddenly told to look at an object on the other side, his head was instantly turned toward the second object, while the eyes remained fixed on the first, by a movement corresponding to that of the head, but in the opposite direction, and then, after a few seconds, they were slowly moved toward the second object. The phenomenon continued to the end of the patient's life.*

The visceral functions are usually little disturbed. Sexual power is often lost. The sphincters rarely suffer. Even when the wasting is general and extreme they may be unaffected, but occasionally they are involved, and they may even suffer early. In other cases, in which the legs become weak after the wasting sets in in the arms, the affection of the sphincters may coincide with that of the legs. In the composition of the urine slight changes have been found, but not constantly. Urea has been increased in some cases, lessened in others.† The quantity of lime has been found to be increased (Frömman), that of kreatin diminished, even to one-tenth of the normal (Rosenthal, Langer). The lungs may suffer from the impairment of breathing power, when the intercostals and diaphragm are weakened.

Varieties.—The chief varieties of the disease depend on the relative distribution of the three conditions: (1) atonic atrophy, becoming extreme, (2) muscular weakness with spasm, but without wasting, or with only slight wasting, and (3) tonic atrophy, rarely extreme in degree, with myotatic excess. The commonest condition is to have atonic atrophy in the arms and upper part of the trunk, with simple weakness and spasm in the legs. Atonic atrophy in both arms and legs is less common, and the least common is tonic atrophy alone, in the arms, or universal. The last is, indeed, extremely rare. It is important to note, however, that these conditions may coexist in every degree and combination; between universal atonic atrophy on the one hand, and universal spastic paralysis without wasting on the other, there is every gradation. The latter does not come into the category now under consideration, but similar cases are met with in which there is atrophy of a few muscles, as, for instance, of the hands only, which complete the series.

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* As I pointed out in an account of this curious condition (*Brain*, vol. i), it is interesting evidence of a normal reflex mechanism in the fixation of the eyes, which was, as it were, isolated by disease, which lessened voluntary control over it.

† In a patient who weighed 7 st. 13 lbs., and whose urine varied between 760 and 960 cubic centimetres, I found the daily excretion of urea to vary between 10.7 and 15 grammes, the average being 13 grammes. This is just half the normal average for a man of that weight, which would be (according to Parkes) 24 grammes.

Complications.—Progressive muscular atrophy is occasionally accompanied by the symptoms of some other degenerative disease of the spinal cord. The paralysis with spasm, already described, can scarcely be regarded as a complication; it is rather part of the disease, and its relation to the other symptoms will be considered when we discuss the pathology of the affection. In rare cases muscular atrophy in the arms is accompanied by the symptoms of locomotor ataxy in the legs. By far the most frequent complication is bulbar paralysis, weakness of the lips, tongue, pharynx, and often of the laryngeal muscles. It is the expression of a degenerative process in the medulla, similar to that which, in the spinal cord, causes the affection of the limbs. It may come on at any stage of the disease, may precede the spinal symptoms, or only come on when these have attained a considerable degree. In characters, the bulbar palsy may resemble perfectly that which occurs in isolated form and will be described among the diseases of the brain. Frequently, however, there is but little paralysis of the tongue, even when swallowing and articulation are much impaired. There may also be slight interference with articulation when there is no marked bulbar palsy. A minor complication is the neuralgic pain, which, as already stated, is sometimes troublesome in the early stage of cases that are due to cold. In several patients I have known headache to be frequent and severe throughout the course of the disease.

Course.—The malady, in most cases, is steadily progressive, as its name implies, but in the rate of its progress it varies in different cases, and in the same case at different periods. It may, moreover, become stationary at any period in its course, and when once actually arrested, it does not usually again become active. Unfortunately, the tendency to cessation is greatest in the later stages of the disease, when there is little except life to be preserved. Sometimes progress ceases at an earlier stage, and chiefly, I think, in those cases in which the atrophy is strictly symmetrical, and develops simultaneously, or almost simultaneously, on the two sides. Thus I have met with several cases in which there was symmetrical wasting in the two hands, or in certain muscles of the two forearms or of the two upper arms, and in which the atrophy, after attaining a considerable degree in its limited seat, had become stationary and continued so. Occasionally some accessory symptom may pass away, although the atrophy progresses. Thus I have known power over the sphincters, at first lost, to be regained in spite of the steady advance of other symptoms.

When the progress at the commencement is rapid, it usually continues rapid, until the disease has attained a wide extent. When it begins slowly, it may continue to be slow throughout, or may, after a time, become accelerated.

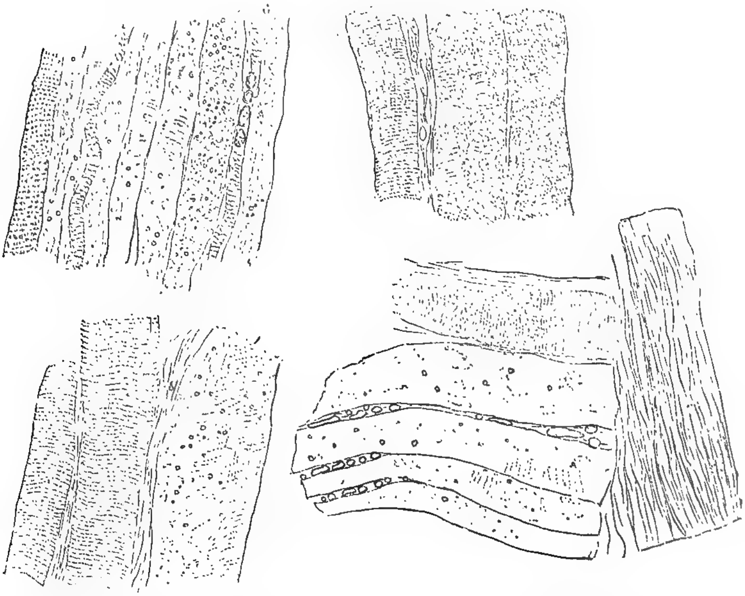
Although the disease sometimes commences in the second arm very soon after its onset, it more commonly makes some progress in its primary seat before beginning on the second side, and the interval that elapses varies with the rate of extension. It often happens that a year intervenes between the affection of the two arms, and I have known, in a very chronic case, the atrophy to slowly progress in one arm for seven years before it showed itself in the other. It is not common for the arms to be reduced to practical helplessness in less than two or three years, but the hands may become useless in as short a time as six months, while in one case the wasting, commencing at the shoulder, had invaded the whole arm in the course of a month. The shortest time in which I have known a patient to reach the last stage of the disease is nine months.

With any rate of general progress, the otherwise uniform course of the disease may be broken by the occurrence of almost sudden palsy in a certain group of muscles. Considerable loss of power, it may be absolute paralysis, comes on in a few days, or even in a few hours. It may occur at the onset of the affection. The extensors of the wrist and fingers are the muscles most commonly thus affected. The weakness is usually followed by a well-marked degenerative reaction in the muscles. In one case, which began by such subacute paralysis of the extensor muscles, first in one arm and then in the other, the initial condition closely resembled the paralysis from lead poisoning. This cause was, however, excluded, and soon the muscles of the shoulder and back presented commencing slow progressive atrophy. In another case the quick loss of power was confined to the extensors in one arm, which were already weak and slightly wasted, most of the other muscles of the upper limbs having been long atrophied. When there is weakness of the legs, without wasting, the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. In one case five years elapsed, after the arms began to waste, before the legs became weak. It is very rare for the paraplegic weakness to occur first.

The chief danger to life is from pulmonary maladies, rendered grave from the weakness of the muscles of respiration. The common complication of bulbar paralysis is another frequent cause of death, either by the interference with swallowing and nutrition or by the laryngeal paralysis. Less commonly death results from bedsores and septicæmia, or from intercurrent maladies.

Pathological Anatomy.—The wasting of the muscles is as evident after death as during life. They are reduced in size and pale in color. Sometimes there is little in the tint of what remains to suggest muscular tissue. Parts of a muscle may be hardly distinguishable from adjacent fat. On the other hand the bulk of the muscle may be dark, and pale streaks in it may mark the position of local degeneration. Under the microscope the fibres present various changes, and of these four are well defined. (1) There may be simple narrowing of the fibres, without any considerable change in their striation (Fig. 129), although the striæ often seem to be further apart than normal, and sometimes the fibrillary segmentation is unusually distinct. (2) Simple fatty degeneration, in which the transverse striation gives place to a granular appearance (Fig. 130), the granules become larger and fewer (Fig. 129), until ultimately distinct globules are scattered through the sheath.

FIGS. 129, 131. FIGS. 130, 132.



FIBRES OF WASTED MUSCLES IN PROGRESSIVE MUSCULAR ATROPHY.

FIG. 129.—Narrowed fibres with nearly normal striation: others clear, containing a few fat globules, and a few faint transverse striæ, and some longitudinal striation: large globules of fat lie in the interstitial tissue in front of one narrowed fibre.

FIG. 130.—Granular degeneration: a fibre with normal transverse striation presents also indications of longitudinal striation. Increase of nuclei of interstitial tissue.

FIG. 131.—Two normal fibres and one presenting the clear homogeneous aspect with a few fat globules and some faint striation.

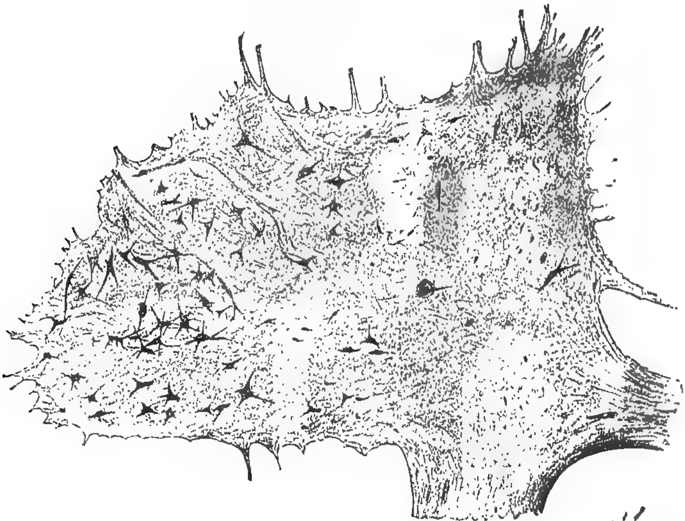
FIG. 132.—Several fibres similar to that of the last figure, with globules of fat between them. On the right is a fibre which has undergone complete longitudinal striation, the normal striæ having disappeared, so that it resembles the adjacent interstitial fibrous tissue. Other fibres in the same muscle were in intermediate stages of degeneration, some transverse striation coexisting with the longitudinal striation.

Where the muscle resembles fatty tissue to the naked eye, the microscope may show only sarcolemma sheaths containing groups of globules. (3) Muscular fibres are seen in which the sheath contains only a clear material enclosing a few fatty globules, and a few transverse striæ, faint, as if fading away. It is probable that this is not the result of fatty degeneration but of a different process, which has been termed "vitreous degeneration,"—a sort of dissolution of the striæ, indicated by the appearance of such fibres as are shown in Figs. 129, 131, 132. (4) A longitudinal striation develops in the fibre, and at first coexists with the transverse striation, but ultimately the latter becomes indistinct, and the fibre looks like a fasciculus of longitudinal connective-tissue fibres (Fig. 132). Sometimes, with the longitudinal striation, the fibre presents a transverse striation very much finer than normal, the striæ being narrower and nearer together as if from a division of the "sarcous elements." This change may sometimes be seen alone, and may be present in only one part of a fasciculus. Thus at one place the number of striæ in $\frac{1}{1000}$ inch was only seven, while in another region of the same fasciculus seventeen were to be counted in the same space. A tendency to transverse fissuring has also been described, but this is probably artificial. Fatty globules accumulate between the fibres (Fig. 132), accompanied in some cases with granules and masses of reddish-brown pigment. There is often also an increase of the nuclei (Fig. 130) and sometimes of the fibres of the interstitial tissue. Two or three rows of nuclei may lie between the fibres. The capillaries may be dilated and distended. It is very common to see muscular fibres that are much altered side by side with others that present a nearly normal appearance. Ultimately the sheaths become empty, and shrink, and may be scarcely distinguishable from the interstitial fibrous tissue.

The peripheral nerves contain many degenerated nerve fibres, and the terminal branches for the muscles a still larger number. If the nerves are traced up to the cord it is found that the degenerated fibres come only from the anterior roots. These are conspicuously changed to the naked eye, small and gray. They may resemble fine threads of connective tissue, and under the microscope no nerve fibres may be found in them, or only a few may remain of normal aspect, the rest being in various stages of degeneration or represented only by their empty sheaths. The degree of affection of the anterior roots corresponds to the wasting in the parts supplied by them. The posterior roots are normal.

The spinal cord is often softer than natural at the affected part, and the white substance of the lateral columns may be conspicuously translucent in aspect, especially in the cervical enlargement. Under the microscope morbid changes are seen in the anterior cornua, and also, in most cases, perhaps in all, in the antero-lateral white columns. The change in the anterior cornua corresponds in its intensity to the origin of the nerves to the most affected parts, and since the latter are usually the arms, the disease is generally most intense in the cervical enlargement. In stained sections the gray matter of the horn is less deeply tinted than normal, but in some parts it may stain more deeply, especially in the circumference of the cornu and the processes of gray matter which project into the white column. Occasionally larger areas have a dense aspect and stain deeply, but the central part of the cornu is generally pale and more translucent than normal. Most of the large nerve cells have disappeared; many entirely, while others may be represented only by small angular bodies. In many instances not a single large cell can be seen in a section which, in a normal cord, would contain a large number. Frequently, however, a few large cells can still be seen, but most of these have lost their processes and are more globular than normal. The interstitial tissue is also changed. The nerve fibrillæ waste with the cells, and there is an increase of the small, angular, and stellate cells and other connective-tissue elements. The larger vessels are dilated and surrounded by wider spaces than normal, but there is no considerable distention of the capillaries. The cornu as a whole is not usually changed either in size or shape. Similar alterations may be traced through the dorsal region. In the lumbar enlargement, the gray matter may be normal, even when it is much altered in the cervical region, but if the legs are wasted there are changes similar to those already described. When the atrophy begins in the legs, the disease may be more intense in the lumbar

FIGS. 133, 134.



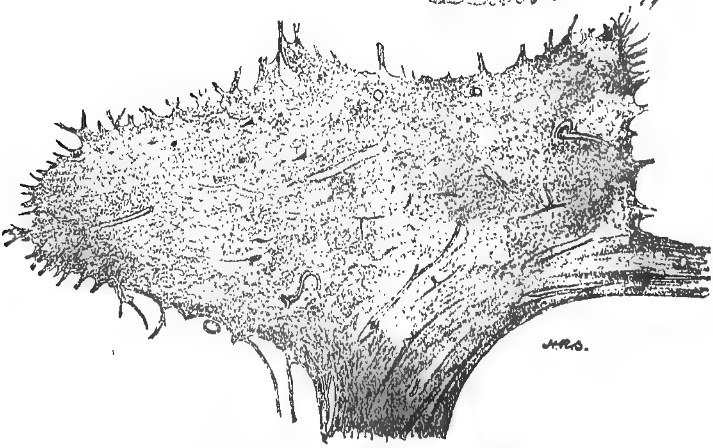


FIG. 133.—A normal anterior cornu for comparison with the next figure.
FIG. 134.—Progressive muscular atrophy; anterior cornu, cervical region.
(*From drawings by Dr. H. R. Spencer.*)

than in the cervical enlargement; but more often the degeneration is slighter, and a larger number of normal or slightly changed cells can be seen. Sometimes certain groups of cells are little affected, while others are much atrophied. The degeneration may involve to some extent the intermediate gray matter between the cornua, but here it ceases; the posterior horn is always normal.

There is distinct degeneration of the anterior root fibres passing from the cornu through the anterior column. A few fibres may remain, but whole fasciculi appear to be replaced by fibrous tissue. There is also degeneration of the fibres of the anterior commissure, in consequence of which it stains much more deeply than normal, in the regions in which the gray matter is considerably diseased.

In the white columns there is usually considerable and often almost complete degeneration of the pyramidal tracts, anterior and lateral.* The area of sclerosis, resulting from the degeneration of the two tracts, varies in extent according to the size of the anterior tract and to the distance which it extends down the cord. In Fig. 136 this is illustrated in an unusual manner in consequence of an inequality in the decussation of the pyramids at the medulla, fewer fibres of the left pyramid having crossed than of the right, so that the left half of the cord contains more than its proper share of the fibres, and the anterior tract extends into the lumbar region even further than does the lateral tract. Where the direct cerebellar tract exists, this, always unaffected, limits the sclerosis of the lateral tract on the outer side. On the inner side, the "lateral limiting layer" (see p. 134) is usually much less degenerated than the pyramidal tract, and intervenes between the latter and the gray matter, but this layer suffers in some degree. The sclerosis does not cease at the anterior extremity of the pyramidal tract, but extends forward in the "mixed zone" of the lateral column, lessening in intensity, and ceasing usually opposite the outer part of the anterior cornu. It is here most intense close to the gray substance, and ceases before it reaches the surface of the cord, the antero-lateral ascending tract being unaffected. It is probable that this sclerosis is due to the degeneration of the short vertical fibres that pass between adjacent regions of the anterior cornu, and which share the degeneration of the nerve cells. Occasionally this sclerosis extends, in slighter degree, into the anterior columns. The posterior columns are always free from definite degeneration, but occasionally there is some general increase of connective tissue throughout the whole cord. The degeneration of the pyramidal tracts is usually considerable in degree, and often most of their fibres seem to have perished. If it is incomplete in the cervical region it may be slight in the lower part of the cord, the degenerated fibres being chiefly those that end in the cervical enlargement and dorsal region. When the sclerosis is intense it is not confined to the cord; it can be traced up through the decussation (Fig. 135, B) and through the medulla oblongata, in which, as Figs. 135 and 136 show, the whole of both pyramids may be degenerated, so that they stain deeply, and no nerve fibres can be distinguished in them. The degeneration of the pyramids has been found in several cases to extend through the pons and crus to the internal capsule, and even through the white substance to the cortex. Above the capsule, however, the pyramidal fibres are so mingled with others that their degeneration is no longer recognizable by the deeper staining of a definite tract; but it can be detected by the presence of abundant products of degeneration in the path of the fibres. In the motor cortex, the large ganglion cells have been found fewer than normal, and distinctly degenerated, many having lost their processes, while in the interstitial tissue, spider cells and other connective-tissue elements are increased in number.*

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*I have not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were unaffected, and I am not aware that any case of the kind has been published since attention was directed to the affection of these tracts by the researches of Charcot.

In cases in which the symptoms of bulbar paralysis were present during life, the motor nuclei of the medulla have presented changes corresponding to those of the gray matter of the spinal cord, with a similar degeneration of the nerve cells. In other instances the degeneration of these nuclei has been slight, but in such cases the degeneration of the pyramids has been intense, and doubtless involved the fibres connecting these nuclei with the cortex of the brain.

The sympathetic nerves and ganglia, when examined, have been found normal, or have only presented such changes as are common apart from symptoms of disease of the nervous system.

FIG. 135.—Progressive muscular atrophy; degeneration of the anterior cornua and pyramidal tracts. A, medulla oblongata, complete degeneration of the anterior pyramids A P; B, at the upper part of the decussation of the pyramids; D, the decussation of the degenerated fibres; A P, the pyramids, still incomplete. C, cervical, D, dorsal, E, lumbar sections. The degeneration of the anterior cornua is complete in C, but in E a few cells remain, for the most part without processes. In C and D the degeneration of both anterior and lateral pyramidal tracts is conspicuous; in E the anterior tract has ceased, and the lateral tract extends up to the surface of the cord, from which it is separated in C and D by the undegenerated direct cerebellar tract. In C and D the degeneration extends forward in front of the lateral pyramidal tract, but gradually ceases. The fibres of the anterior commissure are also degenerated.†

FIG. 136.—Progressive muscular atrophy. A, medulla oblongata; degeneration of the anterior pyramids A P. B, cervical, C, first dorsal, D, first lumbar, E, mid-lumbar sections. The nerve cells have disappeared from the anterior cornua, except in E, in which a few remain. There is complete degeneration of the pyramidal tracts, anterior and lateral. The decussation at the medulla was unequal in this case; of the left pyramid fewer fibres crossed than of the right, and hence the left anterior pyramidal tract is larger, while the right lateral tract is smaller than normal, and, moreover, has ceased at the middle of the lumbar enlargement, to which the left anterior tract extends.‡

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* Koschewnikoff, *Archives de Neurologie, 1883*, No. 18; and *Centralblatt f. Nervenkrankheiten, 1885*, p. 409; Marie, *Neurologisches Centralbl., 1884*, p. 61.

† In this case the arms were greatly wasted and flaccid; the legs were paralyzed, rigid, with moderate wasting. The muscles of the back were also atrophied. In the arms the weakness and wasting came on at the same time, in the right arm some time before the left. The shoulder muscles suffered first, but the atrophy quickly spread, and was ultimately extreme in the muscles of the shoulders, forearms and hands. The disease ran a rapid course; death occurred two years after the gradual onset.

‡ The patient was a man forty-eight years of age. The disease followed a severe exposure to cold. The wasting of the arms was extreme, and they were absolutely powerless. The legs were much less wasted than the arms, but very weak, the loss of power being out of proportion to the wasting.

Fig. 135.

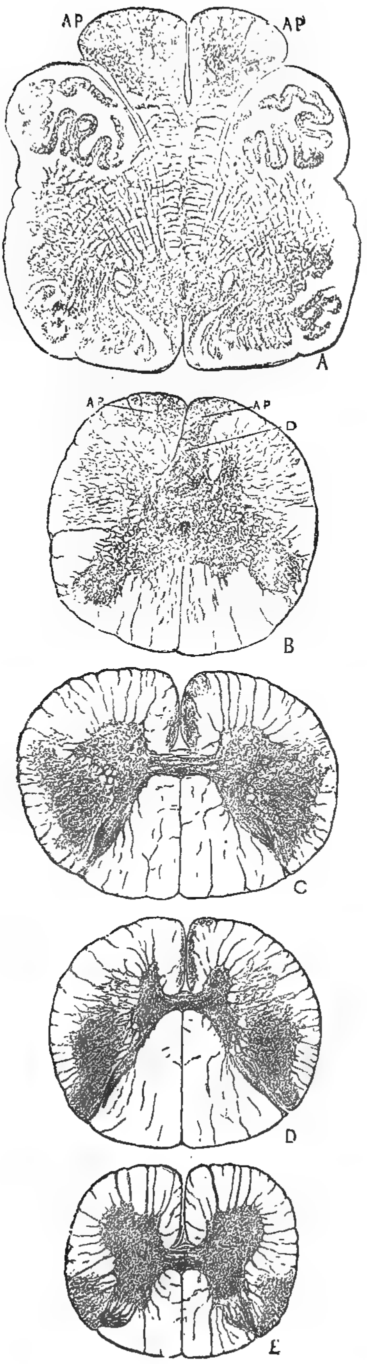
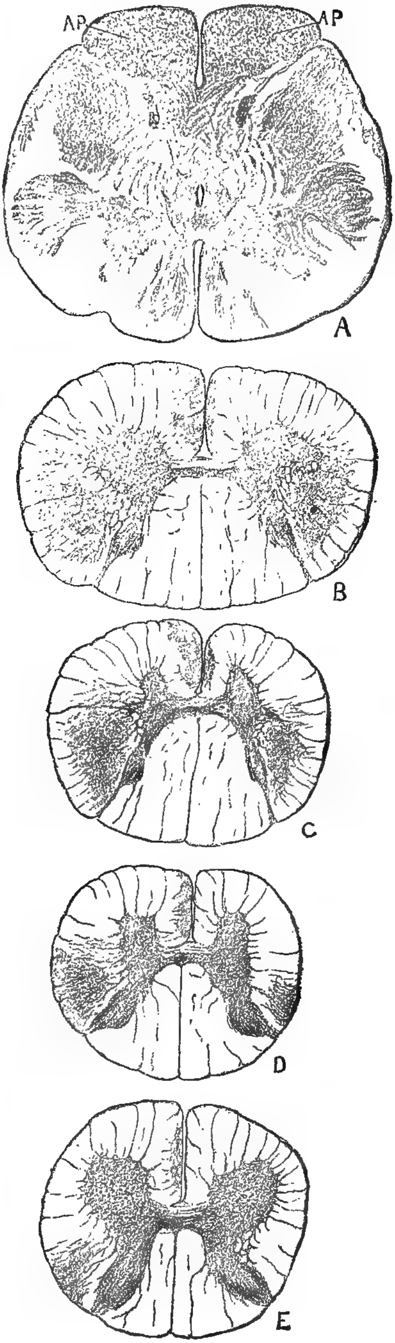


Fig. 136.



PATHOLOGY.—The theories according to which the disease was regarded as primarily one of the muscles or of the sympathetic nerves have now only an historical interest. The constancy of the changes in the ganglion cells of the spinal cord, the degeneration of the motor root fibres, and the analogous effects of acute lesions of the anterior cornua, leave no doubt of the relation of the muscular wasting to the disease of the gray matter, and essentially to that of the ganglion cells. The slowness of the change causes the impairment of the nutrition of the muscular fibres to proceed *pari passu* with that of the nerve elements, and the interference with motor conduction to be proportioned to both. We are thus able to understand the condition of electrical excitability, and its slow failure as nerve and muscle degenerate together. It is only when the slow degeneration is varied by a more acute process of destruction of cells and degeneration of fibres that the muscular tissue is for a time less damaged than the nerve fibres, and presents paralysis in excess of the wasting, and a voltaic irritability in excess of the faradaic irritability of the nerve endings (p. 43). Thus the essential lesion of the disease is a slow decay of the lower segment of the motor path, the segment which consists of the ganglion cells and their prolongations in the axis cylinders of the nerve fibres (see p. 130). To this the conspicuous lesion, the muscular wasting, is secondary. It is perhaps better thought of as a degeneration of the whole segment than as simply a lesion of the ganglion cells, although the latter being the element on which the nutrition of the segment depends, the two views are merely different modes of stating the same fact.

But the disease is rarely limited to the lower segment of the motor path. We have seen that the pyramidal tracts are commonly degenerated, and it is probable that the degeneration often extends through their entire extent, and involves the motor cells of the cortex, as it has been proved to do in several cases.* Hence the upper segment is often degenerated as well as the lower segment. In the presence of that complete degeneration of the lower segment which causes the atonic atrophy of the muscles, the degeneration of the upper segment seems to cause no symptoms. The loss of power that it would produce is also caused by the degeneration of the lower segment, and the latter abolishes the myotatic irritability, excess of which is the characteristic indication of disease of the upper segment. Hence the degeneration of the upper segment of the motor path to the muscles that present atonic atrophy cannot be recognized during life.

What is the relation between the degeneration of the two segments, between the lateral sclerosis and the affection of the ganglion cells? We cannot assume (as some have been inclined to do) that the affection of the upper segment is secondary to that of the lower, because a primary lesion of the gray matter, such as occurs in cornual myelitis, does not cause

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* At the same time we have seen that the nutritional stability of the fibres is less in the lower than in the upper parts, and that their isolated degeneration may be greatest in the lower parts. This may sometimes also be the case in progressive muscular atrophy.

any pronounced ascending degeneration of the related pyramidal fibres, although they may undergo slight degeneration in the course of many years, apparently from disuse. Even a complete interruption of the pyramidal tracts is followed by no degeneration of their upper parts. Hence the intense degeneration met with in progressive muscular atrophy cannot be regarded as secondary and ascending. Neither, when there is atonic atrophy, can we consider the affection of the upper segment to be the primary lesion, and to be the cause of that in the lower. In the early stage of such atrophy there are no indications of the degeneration of the upper segment, which would then certainly exist if such degeneration preceded that of the lower segment. Moreover, degeneration of the upper segment does not necessarily cause any degeneration of the lower. It is a matter of every-day observation that intense degeneration of the termination of the upper segment may occur, from dorsal myelitis, for instance, without any considerable wasting of the legs, and such degeneration has never been known to excite the complete degeneration of the lower segment which causes atonic atrophy. Hence the only adequate explanation of the facts is that the degeneration of the upper and lower segments is simultaneous, or if not simultaneous, at least so far independent that neither is the cause or consequence of the other; both are the result of the same tendency to degeneration of the motor path. Atonic muscular atrophy is thus, at least in many cases, the visible expression of a tendency to decay of the whole motor path from the cortex of the brain to the muscles.

The simple weakness of the legs, with excessive myotatic irritability and spasm, is explained by the degeneration of the pyramidal fibres for the legs, the lower segment being unaffected. In such a case the nerve cells of the lumbar enlargement are normal. When this condition is associated with slight wasting of the legs, without considerable change in electrical irritability, the condition exists that we have more than once considered, in which we must assume that the motor nerve cells of the cord, while structurally intact, undergo slight changes in nutrition. In this condition there are many nerve cells of normal appearance in the gray matter. The changes in nutrition are too slight to cause changes in aspect, or if they cause such changes we have not yet learned to identify them. They are perhaps results of the degenerative changes in the termination of the upper segment, but when such degeneration is secondary to a focal lesion of the cord or brain, the changes in nutrition of the cells never attain such a degree as to arrest the myotatic irritability, or to cause wasting such as attends the destruction of these cells.

In some cases, however, the muscular wasting may be great, although the increase of myotatic irritability persists. In these cases, as we have seen, there is considerable rigidity of the muscles throughout the whole course of their wasting, the condition that we have termed "tonic atrophy." In such a condition it is common to find that many nerve cells have disappeared or are very small, but others remain normal or slightly changed in aspect. Apparently, in addition to the degeneration of the upper segment and to the nutritional changes just mentioned, we have then a considerable degeneration of many, but not destruction of all, the elements of the lower segment. We cannot regard this as simply secondary to the degeneration of the upper segment, for the reasons already given. It must be the expression of a distinct pathological tendency similar to that which elsewhere causes the atonic atrophy and total wasting but slighter in extent and later in time; insufficient to prevent the less affected cells from causing rigidity under the influence of the degeneration of the upper segment. It is doubtful whether the tonic atrophy ever goes on to atonic atrophy. Theoretically conceivable, it is certain that if it ever occurs it is extremely rare. Nor does it seem that atonic atrophy ever gives place to tonic atrophy, with excessive myotatic irritability. The rigidity of tonic atrophy is due to the degeneration of the upper segment, but the effect is not produced if the lower segment is already the seat of such extensive degenerative changes as abolish myotatic irritability. The pyramidal fibres for the parts that are the seat of atonic atrophy are constantly found degenerated, although the muscles have been flaccid to the last. In the very rare cases in which the muscles, relaxed during the process of atrophy, become rigid toward the end of the process, it is probable that the rigidity is idiopathic, due to the changes in the muscles, and is not dependent on the central nervous system. It may be the result of the increase in the interstitial connective tissue, and the longitudinal division and fibrillation by which the muscular fasciculi come to resemble bundles of connective-tissue fibres. The rigidity is always attended by great sensitiveness of the muscles. It is also possible that similar idiopathic muscular changes may ultimately, in tonic atrophy, maintain and increase the rigidity that is primarily dependent upon the spinal cord.

It has been mentioned that the cases in which the legs present the simple palsy and spasm, or the tonic atrophy, which indicate degeneration of the pyramidal tracts, have been separated by Charcot, and termed "amyotrophic lateral sclerosis," the separation being based on the assumption that in such cases the primary lesion is the degeneration of the pyramidal tracts, and that the affection of the gray matter is secondary or "deuteropathic," even where the atrophy is atonic. We have seen also that this assumption is unwarranted so far as the atonic atrophy is concerned. It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy that Charcot's distinction is in effect giving a new name to an old disease. Whether there are indications of lateral sclerosis or not, depends on the circumstance whether the degeneration of the pyramidal fibres is or is not more extensive than the complete degeneration of the nerve cells that causes atonic atrophy. If the latter is universal, the pyramidal tracts may be totally degenerated, and yet there may be none of the characteristic indications of such degeneration. On the other hand, both arms and legs may be the seat of the spastic paralysis that indicates pyramidal degeneration, and atonic atrophy may be limited to a few muscles of the hands. Between these we have every gradation of degree and distribution of atonic atrophy, spastic paralysis, and tonic wasting. Of these only the last named indicates an affection of the gray matter secondary, in point of time, to the pyramidal degeneration, and even then only in part secondary in point of causation. Hence a division into two classes (into which the same case may fall at different periods) is less in harmony with the facts of disease, than is a recognition of the varying extent of the lesion and the corresponding variation of clinical character and course.

The process in the gray matter has been regarded by Charcot and others as a chronic inflammation. The occasional rapid increase in the symptoms may be thought to be justification for this view; but the process in general is at the degenerative extremity of the series of nerve lesions. The principle involved in this question has been already discussed, and we have seen that, whatever be the nature of the primary process, we must recognize secondary tissue changes of independent energy and that a distinct process of inflammation may occasionally form part of these (see p. 285). The significance of the occurrence of inflammation may therefore easily be overrated, so far as concerns the question of the tissue in which the disease begins.

Diagnosis.—The simultaneous and gradual onset of weakness and wasting, the slow but progressive increase and extension of the symptoms, render the diagnosis of the developed malady simple and easy. At the onset, when only a single muscle or group of muscles is affected, the question arises whether the atrophy is local or is the commencement of a wider affection. If there is no indication of a peripheral nerve lesion, local atrophy only occurs from great overuse of a muscle, commonly one of the small muscles of the hand, from some manual occupation, and in a person of feeble general strength. If such cause cannot be traced, the wasting must be looked on with grave suspicion, and a careful and repeated examination will, in most cases, soon show some extension of the atrophy.

In multiple neuritis, the onset of the symptoms is subacute, wasting follows weakness, and the danger of confusion is with subacute myelitis of the cornua, not with their slow degeneration. In pachymeningitis of the cervical region, with considerable damage to the nerve roots, the wasting in the arms may resemble that of progressive muscular atrophy, and there are often weakness and rigidity of the legs, but the wasting is less chronic in onset, and is always accompanied by distinctive sensory symptoms,—by more acute pains, and by anæsthesia, irregular in distribution. The same distinctions suffice for the diagnosis in diseases of the nerve roots of the cauda equina, as by a tumor. This may cause slow wasting in the legs, but there is always severe pain and loss of sensibility.

From primary muscular atrophy, "idiopathic atrophy," we may term it, the diagnosis is sometimes easy, sometimes very difficult. It is easy in pseudo-hypertrophic paralysis, even in the cases of this disease in which no muscles are large, on account of its characteristic distribution, its course, the age at which it begins, and the tendency to affect many members of a family, and males more than females. Whenever several cases of muscular atrophy occur in a family, during childhood or youth, the probability is great that they are idiopathic, and not spinal. But cases are sometimes met with in which idiopathic atrophy occurs in adult life and has not the course or aspect of the pseudo-hypertrophic form. The distinction of such cases may be very difficult. The only general rule that can be given is that they run a more chronic course than the spinal form, often slowly progressing during fifteen or twenty years; that their distribution is unlike that of the spinal form; that the affection often involves the face; that the hand muscles escape in most cases; and that such disease often occurs in many members of the same family. The distinctions will be better understood after a perusal of the account of these idiopathic atrophies.

Prognosis.—The progressive character of the disease renders the prognosis, in every case, unfavorable. The only guide is the observed tendency of the disease. As long as it is distinctly increasing, and in proportion to the rate of increase, the prognosis is grave. Although a hope of arrest is justified, such arrest cannot, in any case, be actually anticipated until it occurs, if the expression may be allowed. Some increase of atrophy in the parts already affected is compatible with an arrest of extension, the muscular changes increasing up to the extent of the disease in the nerve structures. The prospect of early arrest is greatest in the cases in which the wasting is strictly symmetrical and nearly simultaneous on the two sides. The danger to life is chiefly proportioned to the interference with the muscles of respiration, and to the indications of implication of the medulla. If the malady ceases to advance, the prospect of any recovery depends on the rate at which the disease has progressed. Recent rapid loss of power may be to some extent recovered from, especially when the muscles present the degenerative reaction. Wasting that has existed for six months will probably persist unchanged. In a typical chronic case there is little hope of any actual recovery of tissue or power. The effects depend on a slow destruction of nerve elements which are not to be regenerated.

Treatment.—The first important element is to secure favorable conditions of life, and to maintain the general health as perfect as possible. Fresh air and gentle exercise are important, but all fatiguing exertion should be avoided, and likewise all mental strain. When the patient becomes helpless, great care is necessary. Bedsores in this disease mean inattention and may always be avoided. Drugs often fail to influence the malady; those that occasionally seem to do some good are the agents that are most frequently useful in other degenerative diseases of the spinal cord. They have been described in the account of the treatment of locomotor ataxy. Among them arsenic and strychnine are certainly most useful. Strychnia injected beneath the skin has certainly more influence over the disease than when given by the mouth. I have known, more than once, the progress of the disease to cease under the influence of injections, although strychnia had been given by the mouth without effect. The nitrate of strychnia is the best salt for hypodermic use, and the injection may be made once a day, beginning with $\frac{1}{80}$ and gradually increasing to $\frac{1}{40}$. The dose should not be increased beyond this. The locality is unimportant. It is possible that, since the agent enters the blood far more rapidly when injected beneath the skin than when absorbed from the stomach, the momentum of its effect on the nutrition of the nerve elements may be greater.

Local treatment of the muscles has very little influence on the malady, as may, indeed, be expected from its nature. The most sedulous and skillful use of electricity, voltaic or faradaic, fails, as a rule, to produce any effect on the course of the disease. If the malady is progressing at the same rate in each arm, and the muscles of one arm are regularly treated with electricity, and those in the other arm are left alone, no difference can be detected in the rate of wasting on the two sides. It is possible, nevertheless, that electricity sometimes does a little good. In cases in which a rapid loss of power has occurred, and weakness is out of proportion to the wasting, some recovery is possible, and there is no doubt that the excitability of the muscular tissue is maintained for a longer time by galvanism, although the bulk of the muscle may not be influenced. In other cases all that can be said is that the influence of electricity, properly applied, is in the right direction. Moreover, the disease is one of those in which patients find it hard to believe that electricity cannot help them, and the probability is that their conviction will be fostered by some medical adviser. It is one of the diseases in which unjustifiable assertions are too often made that early electrical treatment would have been successful. It is often well, therefore, if only to satisfy the patient that nothing has been left untried, that a careful course of electrical treatment should be adopted. Faradaism may be used, if the muscles are sensitive to it, but if they present any greater irritability to voltaism, it is better to use this. It is immaterial whether the application is confined to the muscles or whether one electrode is placed over the affected part of the spinal cord. The latter method has no disadvantages, but my own observations have failed to confirm the confident statements sometimes made regarding its superiority. It is very important that the current strength employed should be moderate. Strong applications often cause much subsequent pain, and even increased disability, and should be carefully avoided. I have known a rapid increase of weakness follow a strong application, in a way that convinced the patient at least that the two were connected.

Rubbing and massage of the muscles have been frequently employed, and of this treatment also it may be said that its influence is in the right direction, although usually inappreciable so far as the muscular atrophy is concerned. Combined with passive movement, the influence of rubbing in preventing and diminishing deformities is more distinct. No special bath treatment is of service. When the disease occurs in the subjects of syphilis, specific treatment invariably fails, and I have even known the progress of the disease to be distinctly accelerated by an energetic course of such treatment by iodide and by mercury. It is important that all treatment should be pursued in moderation and that its effects should be carefully watched. The disease is one in which it is not easy to do good and not difficult to do harm.

ARTHRITIC MUSCULAR ATROPHY.

Inflammation of a joint is almost invariably attended with rapid wasting of the muscles that move the joint. This occurs equally whatever be the cause of the inflammation, whether this is spontaneous or traumatic. It attends chronic as well as acute inflammations, and occurs in animals if joint inflammation is produced in them.*

The muscles that waste are chiefly those which extend the affected joint. The atrophy is well seen in the muscles in front of the thigh, when the knee is inflamed. If the ankle is affected, the calf muscles chiefly waste; if the hip, the glutei; if the wrist, the extensor muscles of the forearm; the triceps when the elbow is affected; the deltoid when the shoulder joint is inflamed. In rheumatoid arthritis of the finger joints, such wasting is usually very conspicuous in the interossei, and especially in the abductor indicis. The wasting, however, sometimes involves the flexors as well as the extensors, and rarely muscles of the limb that are near but do not move the affected joint. In very rare cases all the muscles of a limb have presented some wasting. Cases in which the atrophy is unusual in distribution, and especially when it is on the distal side of the affected joint, must be regarded with some suspicion, because inflammation sometimes spreads from a joint to a nerve, and distant wasting may be thus produced. It is said that there is sometimes, at the onset, a considerable weakening of the muscle, interfering with movement more than can be accounted for by the pain in the joint which movement causes, and that such initial palsy is transient (Valtat, Duchenne). But the pain, by its inhibitory influence, usually obscures such palsy, which is indeed confessedly rare.

The atrophy, on the other hand, may be regarded as almost constant. If the onset of the joint affection is acute the wasting occurs with rapidity. In a day or two the muscles may be flabby, and in a week or ten days a difference in the circumference of the limb may be detected by measurement. However long the affected muscle may be, the wasting involves the whole length of it. It is not limited to the part in the neighborhood of the diseased joint, but involves the upper part of the muscle in the same degree as the lower part. The degree attained varies much in different cases. Usually moderate, and sometimes slight, it is occasionally considerable, so that the femur, for instance, may be readily felt when the quadriceps femoris is the seat of the wasting, or the head of the humerus may be distinct, and acromion prominent, if the deltoid is affected. Although the whole length of the affected muscle is always involved, if the muscle is a wide and compound one, some parts may suffer chiefly. Thus in the thigh all parts of the extensor may waste equally, or the rectus or vastus internus may suffer more than the other parts. The wasting increases during two or three weeks, then becomes stationary, and continues as long as the joint disease lasts. When the joint has recovered, the muscles in most cases slowly regain their normal size. Occasionally, especially when the arthritis has lasted a long time, the wasting may continue for months or years after the joint disease is at an end.

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* Valtat, *Archives Générales, 1877*, tome 30, pp. 159 and 321. The subject has been discussed by Vulpian ("Leçons sur l'App. Vaso-moteur," 1075, t. ii), and by Paget (*Lancet, 1873*, vol. ii, p. 727, in a lecture republished in "Clinical Lectures and Essays," 1875, p 208).

The electrical irritability of the atrophied muscles may be normal, but is often slightly lessened, equally to faradaism and voltaism. The change is trifling, to be recognized only by comparison with the other side. I have found that this arthritic atrophy is generally accompanied by a distinct, and sometimes a considerable, increase in myotatic irritability. The knee jerk is excessive, if the thigh muscles are affected, and a rectus clonus can sometimes be obtained. A foot clonus may be elicited when the ankle joint is affected. Occasionally the increased irritability extends beyond the region of atrophy, so that, for instance, a foot clonus may be obtained when the knee joint and thigh muscles are affected, although there is no wasting below the knee. As an example of this, which illustrates also the occasional persistence of the atrophy, may be mentioned the case of a young man who jumped over a hoarding and twisted his left leg in doing so. He felt immediately severe pain in the knee, the joint quickly swelled, and became the seat of an acute inflammation which lasted for several weeks, and then slowly subsided. During the inflammation, the thigh wasted. I saw him two years after the onset, and there was still considerable wasting, involving the whole of the extensor of the knee. The minimum circumference of the left thigh above the knee was three-quarters of an inch less than that of the right, although there was no difference between the two legs below the knee. There was a slight diminution in faradaic and voltaic irritability in the affected muscles. The knee jerk was much more considerable on the left than on the right side, and was obtained from above, and there was a well-marked foot clonus in the left leg, but none in the right. He complained that the leg became tired sooner than the other. A year and a half later the difference between the two thighs was still the same, and the other symptoms were unchanged. In some cases there is slight rigidity of the affected muscle; we have seen that rigidity is often associated with an increase of myotatic irritability. Very rarely some contracture occurs in the opponents of the atrophied muscles, but it is uncertain how far the atrophy contributes to this. Thus a child, aged eight, had an attack of rheumatic fever succeeding chorea; the inflammation in the knee joints was prolonged, and was accompanied by marked arthritic wasting in the thighs. During the confinement to bed the flexors of the knees became contracted. There was only a slight diminution in the electrical irritability of the muscles, and no distinct paralysis. Sensory symptoms are, as a rule, absent. I have occasionally known slight tingling in the skin to be complained of, and in one case this symptom was persistent. It is said that areas of anæsthesia sometimes develop, but it is probable that, in such cases, inflammation has extended to a nerve in the vicinity of the joint.

Pathology.—We have no facts regarding the morbid changes that occur in man. In animals Valtat found the muscles paler than normal, with simple narrowing of the fibres, but without interstitial changes, and no abnormal appearance could be detected in the nerves or the spinal cord. Thus pathological anatomy throws no light on the mechanism by which the wasting is produced. The only constant condition, in these cases, that can be regarded as the cause of the wasting, is the simple fact of the joint disease. The atrophy seems to be especially rapid when there is much pain in the joint, and Paget has observed similar wasting when there has been neuralgic joint pain without inflammation. But such pain is not always attended by wasting, and the latter may attend arthritis that is not painful. Disuse will not explain the atrophy; first, because its influence in causing wasting is trifling and tardy; and, secondly, because other muscles of a limb, equally disused, present no wasting. Nor can the effect be due to any local inflammation of the muscles, passing to them from the inflamed joint. Such a cause is excluded by the fact that the whole length of a muscle suffers equally; the part at a distance from the joint wastes as much as the part near the joint, and moreover, when such wasting occurs in animals, from traumatic arthritis, no signs of inflammation can be found either by simple or microscopical examination. We are, therefore, reduced to the assumption that the wasting is produced, in some way, through the nervous system. "A reflex atrophy, due to the disturbance of some nutritive nervous centre, irritated by the painful state of the sensitive nerve fibres," is the pathology suggested by Paget. A similar explanation has been given by Vulpian, who surmised that the wasting may be due to alterations in the nutrition of the ganglion cells of the cord. A theory of this character seems the only tenable one. It is, however, an interesting fact that the wasting is accompanied by an increase in the myotatic irritability. We have, in fact, the condition that often results from slight degeneration of the pyramidal tracts, and must be immediately due to the changes in the termination of the pyramidal fibres in the gray matter—increased myotatic irritability, with slight wasting, and merely a slight diminution in the electrical irritability, similar to each current. It is possible, therefore, that this terminal structure participates in the nutritional changes. This view is favored by two other facts, first, that the increased myotatic irritability may continue for years, and secondly, that a primary joint inflammation has been known, in rare cases, to cause persistent symptoms of impaired function of the pyramidal fibres—presumably the result of changes in the nutrition of their terminations—without muscular wasting (see p. 326). Since the sensory influence must be exerted through the sensory cells of the cord, we can understand the occasional occurrence of such subjective sensations as the tingling above mentioned.

Diagnosis.—The chief difficulty in the diagnosis of the condition arises from ignorance of the occurrence of this form of wasting. Its nature is usually recognized at once, if its existence is known, because the primary character of the arthritis which produces it is sufficiently obvious. In cases in which a joint inflammation results from a primary disease of the nervous system, which also causes wasting of the muscles, a mistake is conceivable, but it is unlikely, because such arthritis results only from lesions of the nervous system that are considerable in degree, and their existence is then sufficiently obtrusive. In some cases of neuritis in the arm, secondary arthritis may occur in the small joints of the hand, with wasting of their muscles, and the ultimate condition may very closely resemble, in aspect, that which we are now considering, but other indications of a serious lesion of the nerves, such as anæsthesia and paralysis, either are or have been present, and indicate the nature of the case.

PROGNOSIS.—When the inflammation of the joint is brief in duration, recovery of the muscles may be anticipated with confidence. In children, even after prolonged joint disease, the muscles usually regain their normal bulk. In adults the wasting often lasts for a long time after the joint is well, and a cautious prognosis should be given, if the arthritis has lasted long. Even if slight wasting is persistent, normal power is usually recovered, but, as the case mentioned above shows, the symptoms sometimes continue for years.

TREATMENT.—The chief treatment is local; electrical stimulation of the muscles, and gentle rubbing. It is doubtful whether drugs have any influence on the condition. As long as the joint is inflamed, treatment rarely causes any increase in the bulk of the muscle. When the arthritis has ended, the muscles usually recover slowly without assistance, but it is probable that the local treatment accelerates the process. The form of electricity is of little consequence; either faradaism or voltaism may be employed, but should only be used in sufficient strength to cause gentle contraction of the muscles. Painful stimulation of the sensory nerves should be avoided.

MUSCULAR ATROPHY FROM OVER-USE.

Muscles that are much used sometimes waste. The effect is seen most frequently in the small muscles of the hand, in persons of weakly constitution, who use these muscles unduly. Thus a young lady devoted many hours a day to "illuminating," during several years, and then some muscles of the thenar eminence began to waste, and became considerably atrophied. Such wasting is scarcely ever met with in the larger muscles, but has been observed in the biceps, consecutive to hypertrophy, in Sheffield smiths (Frank-Smith). The electric irritability is gradually lowered in this local wasting, equally to both currents, as it is in progressive muscular atrophy. We do not know whether this wasting from over-use is purely local or whether the related ganglion cells of the spinal cord fail first, and the muscular wasting is secondary to their atrophy. As Gull said many years ago, "It is as reasonable to infer a lesion of the gray matter from over-work as of the muscles."* The wasting often persists when the over-use of the muscles is discontinued. Hypertrophy from over-use does not usually give place to atrophy. These two facts are somewhat in favor of the view that the lesion is primarily of the nerve cells. The wasting in these cases shows no tendency to extension beyond its original limits. The treatment is, first, the cessation from the excessive exertion of the muscles; secondly, the improvement of the general health, and the administration of nerve tonics, especially of strychnine; and thirdly, the gentle electrical stimulation of the muscles by either faradaism or voltaism.

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* *Guy's Hosp. Reports, 1862,* p. 246.

IDIOPATHIC MUSCULAR ATROPHY.

MYOPATHIC ATROPHY; MUSCULAR DYSTROPHY.

Idiopathic atrophy, which is primarily muscular and does not depend on disease of the central nervous system, may be conveniently considered in this place. It does not indeed come within the class of diseases that are the subject of this book, but it is one of those maladies in which clinical resemblance may conveniently be allowed to supersede the rules of pathological classification.

The chief form of idiopathic muscular atrophy is that known as "pseudo-hypertrophic paralysis." Other cases occur which resemble more closely in their aspect the spinal form, and differ in some particulars from the pseudo-hypertrophic disease, with which, however, they have some characters in common.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS (MUSCULAR PSEUDO-HYPERTROPHY; LIPOMATOUS MUSCULAR ATROPHY, ETC.).

The affection thus designated is a disease which chiefly manifests itself during the later developmental period of childhood, and clearly depends on a morbid developmental tendency which is often present in several members of the same family. It is thus a congenital disease in its origin and nature, although not congenital in its manifestation. It is characterized by a progressive change in the size, and diminution in the power, of many muscles. They may be larger than normal, or smaller, or may be at first large and then small, and they may be harder than normal. The alterations in size and consistence depend on an abnormal growth of interstitial connective tissue, in which fat is often deposited, and before which the muscular fibres waste. The progressive weakness cripples the patient, and interferes with breathing power, until chronic lung disease or some intercurrent malady ends life.

Isolated cases, which can now be recognized as examples of this disease, were recorded in England in 1830 (Sir Charles Bell) and in 1847 (Partridge) and in Italy in 1838, but the first series of cases were described by Meryon in 1852, and Oppenheim in 1855. These observers failed, however, to distinguish the disease from the common form of progressive muscular atrophy, partly because enlargement of muscles was not so obtrusive a feature of these cases as it was in some others which soon afterward came under the keen eye of Duchenne. This acute observer was then exploring the field of muscular paralysis with the aid of "localized electrization." He recognized at once the novel features of the disease, and published, in 1861, an admirable account of it, giving to it the name of "pseudo-hypertrophic muscular paralysis," which, in spite of its clumsiness, expressed the salient features of the malady sufficiently well to have since held its own against many other designations that have been suggested by more exact knowledge of the pathological changes.

Etiology.—Our knowledge of the causes of the disease is limited to a few general facts. Males furnish the majority of the cases, but the actual degree of preponderance is not yet certain. Published cases show a proportion of seven to one, but of forty-three cases that have come, directly or indirectly, under my own notice, thirty-three were males and ten females. This discrepancy is perhaps due to the fact that the slighter liability of females influences the character of the disease as well as its occurrence. In them it is slighter in degree, later in development, and less frequently causes death. Hence the disease is more likely to escape notice in females, and cases in which it is recognized are less likely to be published, because, when a disease has become familiar, the cases recorded are chiefly those, as we say, "completed by an autopsy." Hence it is probable that a proportion of four males to one female is near the truth.

The disease occurs rather less commonly in isolated cases than in family groups. The number in a family has varied from two to eight. As many as eight brothers suffered and died in the family described by Meryon, while all the daughters escaped. In a family known to me, four sons have suffered and none of the daughters; in another instance two daughters are affected and no sons. Thus there may be a tendency in a family to the affection of one sex, and not the other; but, on the other hand, it is also common for both sexes to suffer in the same family. In many instances in which many members of one generation are affected, no antecedent cases can be traced in the family; the malady, while congenital, is not hereditary. In other families antecedent cases can be traced, and these are invariably on the mother's side. The disease is thus transmitted by women who are not themselves its subjects. In a case in which four brothers suffered, the mother's brother and sister were likewise affected. Again, a brother and sister were diseased, one daughter of a second sister, and three daughters of a third sister. In another instance a boy suffered, and his sister, unaffected, had two sons diseased and a daughter free, of whose children two sons were the subjects of the malady. Thus the congenital tendency is exclusively due to the maternal element in the embryo. This is also shown by another fact, that the children of the same woman, by different husbands, have been affected. Indirect hereditary tendencies, such as are indicated by the occurrence of diseases of the nervous system, can be traced so rarely that it is doubtful if they have any influence, and it does not appear that the occurrence of the malady is due, in any degree, to mere consanguinity of parents, to their age, or their intemperance.

The disease always manifests itself during the period of development, sometimes in the early stage of growth, at the close of infancy, often only during mid-childhood, rarely not until growth is nearly ended. In a third of the cases, the first symptoms are noted when the child first attempts to walk, which is usually a little later than in healthy children. In about another third the child seems well until it is four, five, or six years old, and then impairment of power attracts attention. In three-quarters of the cases, the disease manifests itself before the tenth year. Very rarely the patient is conscious of no symptoms until after puberty, and he has reached the age of eighteen or twenty. I have seen four instances of this late apparent onset, but in each inquiry has elicited a history of enlargement of muscles long before their power became impaired. One patient, for instance, in whom weakness was only noticed when she was twenty, had been often "chaffed," when a young girl at school, on account of her "tea-kettle calves." It is therefore probable that, in such late cases, it is rather the influence of the disease on the muscular fibres, than its development, which is delayed. Neither social state nor general constitutional condition seems to influence the occurrence of the disease, but its manifestation has sometimes been apparently accelerated by influences that disturb the general health; it has been first noticed, for instance, during convalescence from some general illness.

FIG. 137.

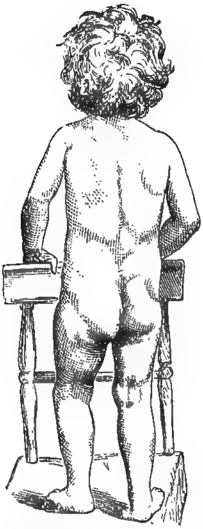


FIG. 138.

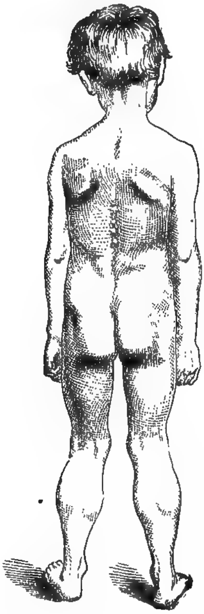
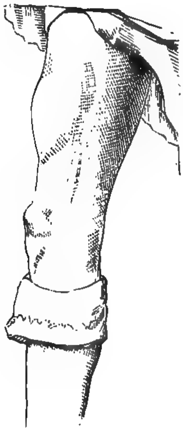


FIG. 139.



FIGS. 137 and 138.—Two brothers, aged four and seven, suffering from pseudo-hypertrophic paralysis.
FIG. 139.—Partial enlargement of rectus, the vasti being small.

SYMPTOMS.—Impairment of power usually attracts attention before any change is observed in the size of the muscles, or if these are noticed to be large, it is with feelings of parental pride rather than with suspicion. The children, who usually walk late, often also walk clumsily, fall with ease and rise with difficulty. The act of going upstairs is especially difficult to them; the child has to take hold of the banisters and pull himself up.

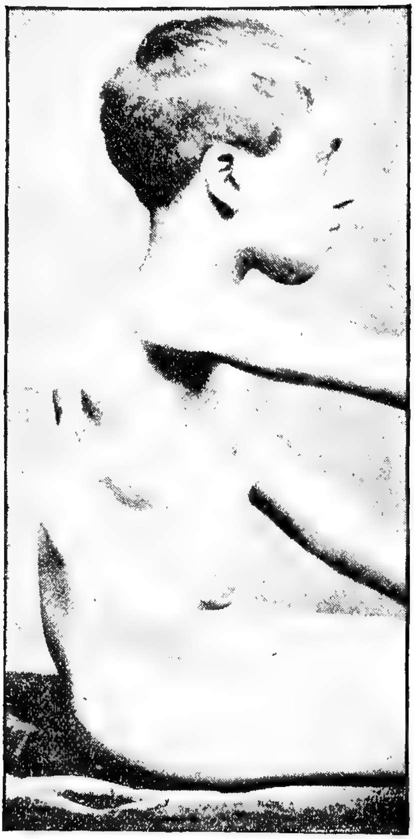
The muscles may at first present nothing unusual; if the child is fat, as is frequently the case, neither an increase nor a diminution in size can be readily observed. At the age of five or six years an unnatural enlargement of certain muscles is usually conspicuous, especially when there is a contrast between these and other muscles which are small. If enlargement is almost universal, it is usually great and conspicuous. The enlargement of muscles, although for a time it may increase, ultimately lessens, earlier in some muscles than in others, and it may give place to an actual wasting.

Among muscles that are most frequently large those of the calf take the first place. They sometimes attain a remarkable size. I have measured a calf 14½ inches in circumference in a boy of twelve. The muscles in front of the lower leg are less frequently enlarged, but sometimes project beyond the edge of the tibia. The extensors of the knee are often big; occasionally the rectus or vastus internus is alone increased in size (the rectus, in Fig. 139), and the other parts may be normal or small; less frequently all parts are small. The flexors of the knee commonly escape, the glutei are frequently conspicuously large, and so also are the lumbar muscles. The condition of those of the shoulder is peculiar and important. Of all the muscles of the body, next to those of the calf, no one is enlarged more frequently or in greater relative degree than the infra-spinatus. It often stands out so conspicuously that its limit is apt to be mistaken for the edge of the scapula (Figs. 140 and 141). The supra-spinatus is sometimes also prominent, but its condition is usually concealed by the trapezius, which is little involved. The deltoid is also frequently large; the serratus rarely. The pectoralis is never enlarged, but on the other hand, its lower half is wasted in a large proportion of the cases, and with this the latissimus dorsi, which has the same action in depressing the raised arm (see p. 49). The teres major may share the wasting of the latissimus. In many cases no trace can be found of the latissimus and lower part of the pectoralis, and it even seems as if, in some cases, these muscles may be congenitally absent. In consequence of the loss of the latissimus, the posterior axillary fold is almost absent, and the aspect of the axilla is strikingly abnormal, especially when, as is often the case, the defect contrasts with the conspicuous prominence of the infra-spinatus (Figs. 140 and 141).

The other muscles of the arm suffer in diminishing degree and frequency from above downward. The triceps and biceps are sometimes enlarged, the former more frequently than the latter, but occasionally only in one part. Both these muscles are sometimes wasted. The forearm muscles suffer in only a small minority of the cases, and the intrinsic muscles of the hand usually escape altogether.

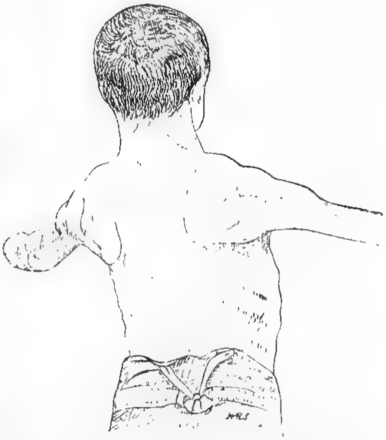
The muscles of the neck are rarely affected, but I have noted, in a few cases, wasting of the clavicular part of the sterno-mastoid. Those of the face do not suffer, but in the patient shown in Fig. 144 there was considerable enlargement of the masseters. The tongue has been increased in size in a few instances. The other muscles supplied by the cranial nerves always escape.

FIG. 140.



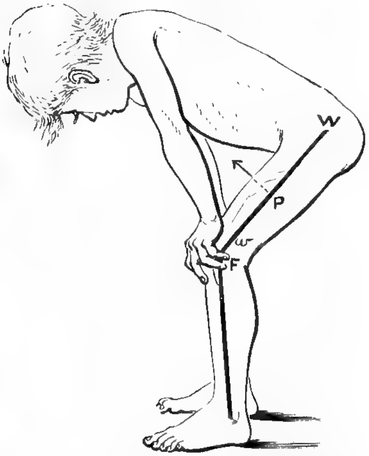
Pseudo-hypertrophic paralysis. Absence of latissimus dorsi, enlargement of infra-spinatus. (*From a photograph.*)

FIG. 141.



Wasting of latissimus dorsi and serratus; enlargement of infra-spinatus, supra spinatus, and deltoid; atrophy of biceps and triceps. (*By Dr. H. R. Spencer, from a photograph.*)

FIG. 142.

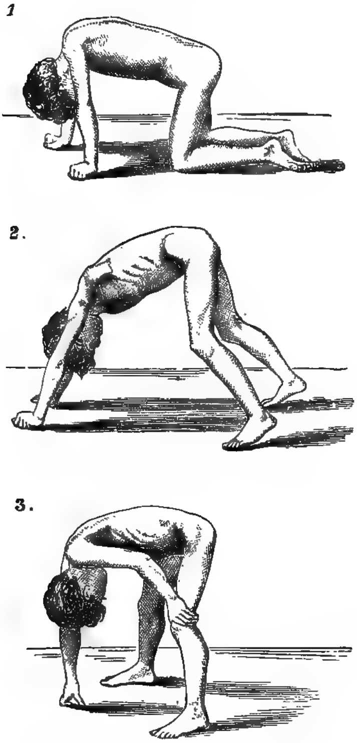


Mode of obtaining extension of hips in pseudo-hypertrophic paralysis. F, fulcrum of the lever formed by the femur. P, mean position at which the power is applied by contraction of the quadriceps femoris. W position of weight in the ordinary mode of rising. w, the place to which part is transferred by putting hands on knees.

The diseased muscles are weak, but the impairment of power is to some extent irrespective of the change in size. The muscles that are abnormally small are generally weaker than those that are abnormally large; and in the latter the weakness increases with the wasting. In the legs the greatest weakness is in muscles that are inaccessible to observation,—the flexors of the hips; next in order of weakness come the extensors of the knee, and the extensors of the hip. The muscles below the knee usually retain considerable power for a long time, and the extensors of the ankle fail before the flexors. In the upper limbs the depressors of the arm are usually alone weakened during the early period of the disease, but subsequently the shoulder muscles suffer, then the triceps and the biceps, while the muscles that move the hand commonly retain good power to the last.

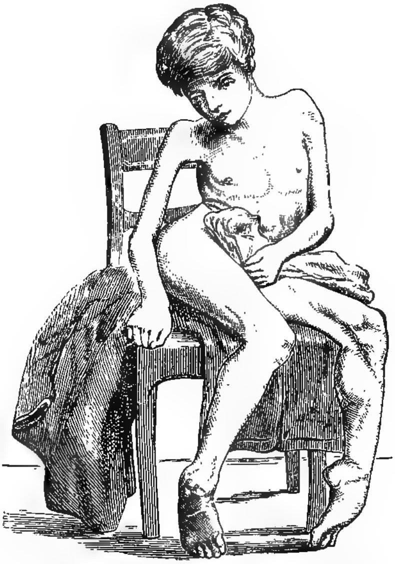
The distribution of weakness in the legs causes certain peculiar defects of movement which are very characteristic, and some are even all but pathognomonic of the disease. The difficulty in going upstairs is especially due to the weakness of the extensors of the knee and hip. The defect of the extensors of the hip causes the gait to have a peculiar oscillating character, in which the body is so inclined as to bring the centre of gravity over each foot, on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination toward the leg that is off the ground, unless the balance is exact. The greatest defect, however, is in the power of rising from the floor, and the most characteristic peculiarity

Fig. 143.



Mode of rising from the ground in pseudo-hypertrophic paralysis.

Fig. 144.



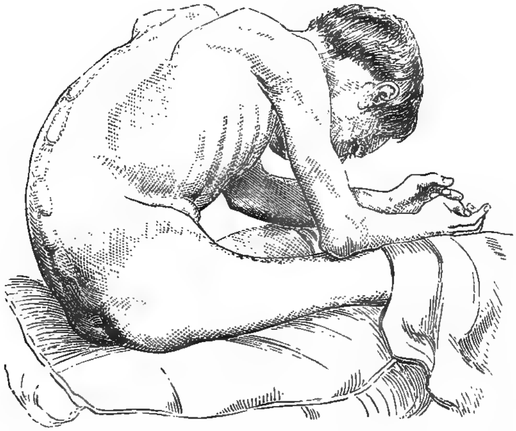
Late stage of pseudo-hypertrophic paralysis: a boy fourteen years old, with muscular contraction and wasting and lateral curvature of the spine.

is the mode in which this is achieved, if still possible, and no objects are near by which the patient can aid himself. He commonly has not sufficient power to extend the knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power, applied between the fulcrum and the weight, acts at least advantage. He therefore places his hands on his knees, as in Fig. 142; and his arms thus bring much of the weight of the upper part of the trunk on the femur close to the fulcrum, between this and the power, which can then act at greater advantage. Moreover, the mere weight of the head, which is in front of the arms, tends to aid the extension, and indeed may effect the extension of the knee without the aid of the extensor muscles, as any one may ascertain by observing the mobility of the patella in this attitude. When the knees are extended, the power of the extensors of the hip may be sufficient to raise the body into the upright position, or the patient may aid them by an upward push with the hand as he takes it off. If, however, these extensors are weak, the hands are often moved higher and higher up the thighs, grasping alternately, and thus pushing up the trunk. To get thus the requisite support, the knees must not be quite extended, and if their extensors have no power, the device cannot be employed, and the patient is altogether unable to rise. In many cases, especially when extension of the hip is easy, the patient achieves the extension of the knees in another way; he puts the hands on the ground, stretches out the legs behind him far apart, and then, the chief weight of the trunk resting on the hands, by keeping the toes on the ground and pushing the body backward, he manages to get the knees extended, until the trunk is supported by the hands and feet, all placed as widely apart as possible (Fig. 143, 2). Next the hands are moved alternately along the ground backward, so as to bring a larger portion of the weight of the trunk over the legs. Then one hand is placed upon the knee (Fig. 143, 3) and a push with this, and with the other hand on the ground, is sufficient to enable the extensors of the hip to bring the trunk into the upright position.

The shortening and contraction of certain muscles lead to another group of symptoms,—distortions due to permanent alteration in the position of joints. Some of these are produced, as are distortions in other forms of muscular weakness, by shortening of the less affected opponents of the weaker muscles. Thus the knee joints become fixed by the contraction of the flexors, and the elbow by the contraction of the biceps when the triceps has lost all power. These contractions only occur late, and are usually facilitated by the habitual flexion of the knee and elbow joints. But the deformity at the ankle joint, which results from contraction of the calf muscles, commences at a much earlier period, before there is any disproportionate weakness in the opponents of these muscles, and with little assistance from posture. It coincides commonly with the diminution in bulk of the muscle, and is the result of the shrinking that occurs in the length as well as in the breadth of the muscle,—a primary contraction. As a consequence of it the patient cannot get the heels well upon the ground, and the foot cannot be flexed passively beyond a right angle. The gradual increase of the contraction results in considerable "talipes equinus." In consequence of the muscular weakness which coincides, the patient is able to walk but little, and the loss of the extension involved in the act of walking permits a rapid increase in the unopposed contraction. The feet, as Fig. 144 shows, soon assume a posture of extreme extension, the dorsum being in a line with the front of the leg, or forming with it a convex curve. A subluxation of the ankle joint takes place, and the articular surface of the astragalus, its anterior extremity, and that of the os calcis, form three prominences under the skin. When this reversal of the ankle occurs, the tibialis anticus can no longer act as a flexor.

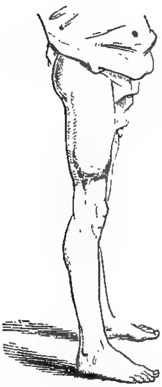
Another deformity, which is due chiefly to muscular weakness, is curvature of the spine. An antero-posterior curve, with the concavity backward, is an early symptom of the disease, and it may become extreme, the upper part of the trunk being carried so far back that a vertical line from the scapula falls an inch or more behind the sacrum. It is due, not to the weakness of the trunk muscles, but to that of the extensors of the hip, in consequence of which the pelvis is inclined forward, carrying with it the lower lumbar vertebræ and the abdomen; hence the upper part of the trunk has to be held far back to keep the centre of gravity of the body over the feet. The proof of this mechanism is that when the patient sits, and the pelvis is supported on the ischial tuberosities, the lordosis disappears. It is, indeed, replaced by an opposite curve, in which the back becomes convex, clearly due to the weakness of its extensor muscles. This curve may become very great, as in the case shown in Fig. 145. This weakness of the spinal muscles also permits the occurrence of lateral curvature (Fig. 144), influenced, in its direction, by the habitual posture and the preponderance of weakness on one side or the other.

FIG. 145.



Lad, aged fifteen; late stage; wasting of thighs; inability to sit upright in consequence of the weakness of the spinal muscles.

FIG. 146.



Enlargement of the vasti and not of the rectus. In this case all other muscles were below normal size.

The electric irritability of the muscles is not usually altered until distinct weakness is developed. Then, especially after wasting sets in, the irritability is lowered, alike to faradaism and voltaism. There is never any trace of degenerative reaction.

The knee jerk may be at first normal, but as the extensors of the knee become feeble it is always lessened and gradually disappears. It is never excessive, and in all advanced cases it is lost. Sensation is unaffected, and so also are the sphincters in the vast majority of cases. Very rarely there has been, toward the end, a slight difficulty in the retention or expulsion of urine, to be regarded, perhaps, rather as a complication than as an effect of the disease. All other functions of the nervous system are commonly normal. The sympathetic functions are undisturbed, and in most cases the mental development of the subjects of this disease is rather beyond that of other children of the same age, doubtless on account of the indirect influence of a malady which withdraws them from active amusements.

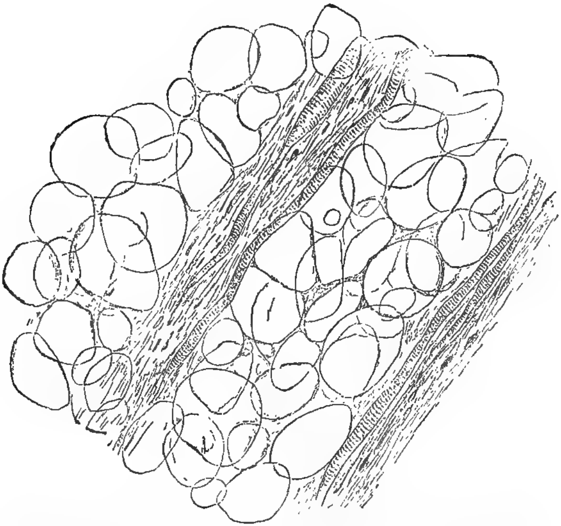
The rate of progress of the disease and its duration vary much. After some years, often between ten and fourteen, the power of standing becomes lost in consequence of the increasing weakness and the contraction of the calf muscles. The latter is chiefly influential, and if the tendons are cut, the ability to stand may be restored and may remain for some years more. When the patient ceases to walk, the muscular disease makes more rapid progress, deformities become greater, and lateral curvature of the spine intensifies the effect of the muscular weakness in limiting the respiratory movements. The muscular wasting may then become great, especially about the shoulders and thorax, but the patient may live on, helpless, except in the hands, for several years. Death is often due to some intercurrent malady, an acute specific disease, acute pneumonia or bronchitis. In other cases death is the result of chronic lung disease, a form of pneumonic phthisis or broncho-pneumonia, which develops gradually, with little febrile disturbance, apparently in consequence of the influence on the lungs of the lessened respiration. There is never sufficient impairment of the respiratory muscles to cause death directly. Such pulmonary disease generally ends life some time between twelve and twenty. The duration of the stage of helplessness depends very much on the care which the patient can obtain.

In the cases in which muscular power remains good until after puberty, the progress of the disease is generally slow. The patient may reach the age of thirty before power is much impaired. It is possible that, in some cases, the disease never attains a considerable degree. More frequently, however, after slight symptoms have lasted for some years, a rapid increase occurs, and very few patients reach the age of forty. The course of the disease is slower in girls than in boys, and females furnish a relatively large proportion, perhaps one-half, of the late cases.

Varieties.—The chief varieties of the disease depend on the age at which it commences, and on the condition of the muscles, whether they are large or small. In rare cases a single muscle may be large, and the rest small, as in Fig. 146, in which only the vasti were increased in size. Or everywhere and from the first the muscles are smaller than normal, and they progressively waste. Such cases are not uncommon; in the first group of cases described (by Meryon), enlargement of muscles was inconspicuous. Many cases in which all muscles are small belong properly to the form considered in the next section. The cases described by Meryon are generally regarded as examples of pseudo-hypertrophic paralysis, but they present many points of resemblance to the "simple atrophy" described further on.

Complications.—Congenital mental weakness, due apparently to defective development of the brain, sometimes complicates pseudo-hypertrophic paralysis. In rare cases there have been indications of some other morbid condition of the central nervous system, such as epilepsy. It is uncertain in what light the slight occasional affection of the bladder is to be regarded, whether as an invasion of the vesical muscles or as a central complication. Vigoroux has recorded a case in which the symptoms of pseudo-hypertrophic paralysis were combined with the peculiar rigidity of Thomsen's disease. Of course the subjects of the disease are liable, like other children, to various diseases of the nervous system, such, for instance, as chorea, which brought one well-marked case under my notice, but these are merely accidental complications.

FIG. 147.

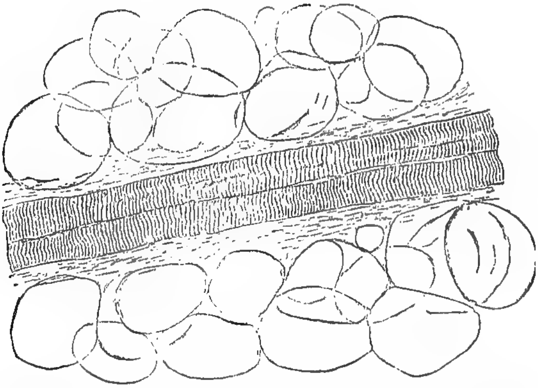


Gastrocnemius muscle; muscular fibres, irregularly narrowed and in part degenerated, lie among tracts of nucleated fibrous tissue, separated by adipose tissue.

PATHOLOGICAL ANATOMY.—It is rare, at the time of death, for any muscles to be actually larger than natural, and most of those that are affected are usually below the normal size. They are pale and yellowish in color, and often, to the naked eye, resemble perfectly masses of adipose tissue. The resemblance is not merely one of aspect. As seen under the microscope, it may be difficult for the observer to realize that he is not looking at a fatty tumor. Nothing may be at first visible but fat cells, precisely like those of adipose tissue. Among the cells, however, are tracts of nucleated fibrous tissue, and a closer examination of these shows that the tracts contain also muscular fibres (Fig. 147), most of them much narrower than normal. They are also irregular in width; a broad fibre, for instance (as in the figure), suddenly becoming narrow.

The fibres for the most part preserve their transverse striation, but where they are narrowest this may have in part disappeared, either by granular degeneration, or, more commonly, by a simple fading of the striæ. In the narrowed fibres the striæ are sometimes further apart than normal. In other parts broad fibres may be seen, normal or nearly normal in aspect (Fig. 148), coursing among the fat cells, and accompanied by a smaller amount of fibrous tissue. Large fibres occasionally present fatty degeneration, or a longitudinal striation, but these are rare. Some empty sarcolemma sheaths may be seen where the narrowing of the fibres is greatest. In muscles that still preserve some red tint, the amount of fat is less, and there is often a relatively larger amount of interstitial fibrous tissue.

FIG. 148.



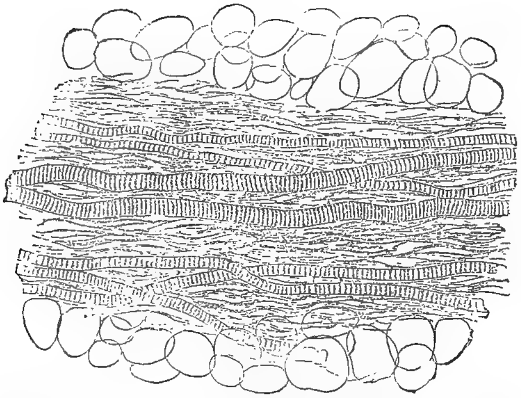
Gastrocnemius muscle; two nearly normal muscular fibres, accompanied by fibrous tissue, surrounded by fat cells.

In some of these the interstitial tissue may be almost entirely fibrous, a few fat cells only being visible here and there. In such muscles it is common to find the fibres more damaged than in those in which the growth is partly fatty. It is chiefly in the latter that many normal fibres are seen. No muscular fibres larger than normal have been found after death, but such fibres have been seen in fragments removed during life by excision or by a "harpoon trocar;" it is probable that the increased size was due to a vital contraction under the mechanical stimulus involved in the extraction. (See note, p. 402.)

The motor nerves to the muscles have not in any case been found diseased. The condition of the sensory muscle nerves (which terminate in the interstitial tissue in which the primary morbid process occurs) has not been ascertained in any instance. In several cases in which the spinal cord has been examined it has been perfectly normal. In a few there have been slight and irregular degenerative changes. They have not been so marked in any other case as in one examined by Lockhart Clarke and myself.* In this case the cervical and dorsal regions were normal, with the exception of here and there slight accumulations, at the bottom of the fissures, of products of degeneration, probably derived from the perivascular erosion common at all ages. At the last dorsal segment, however, there was an area of granular disintegration in the intermediate gray substance on each side, in front of the posterior vesicular tract. This part was unduly translucent for half a centimetre in vertical extent, and in the middle of this area the disintegration had produced an actual cavity across which the fibres for the cerebellar tract ran unchanged.

PATHOLOGY.—The common integrity of the anterior gray matter of the cord, and especially of the motor nerve cells, seems conclusive evidence that the disease of the muscles is not due to a primary lesion of the spinal cord. Pseudo-hypertrophic paralysis is not therefore, as was at first thought, merely a form of spinal muscular atrophy with a special muscular change. The significance of the pathological anatomy is that the malady is a primary disease of the muscles, consisting in an overgrowth of the interstitial connective tissue, in which fat may or may not be deposited, and that the wasting of the muscular fibres is secondary to the interstitial growth. The significance of the conditions under which the disease occurs is that the disease is congenital, the result of a perverted tendency of growth, inherent in the embryo, and derived from the germ from which the embryo proceeds. In this connection it is instructive to note that there is one form of congenital tumor the structure of which is almost exactly the same as that of the muscles in

FIG. 149.



Section of a myo-lipoma which was attached to the spinal cord of a man suffering from locomotor ataxy, for comparison with Figs. 147 and 148.

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* *Med.-Chir. Trans.*, vol. lvii, p. 247.

pseudo-hypertrophic paralysis. Fig. 149 might be a fragment of a muscle in this disease, but it is a section of a myo-lipoma attached to the conus medullaris of the spinal cord of a patient whose muscles were healthy. (The tumor is shown also in Fig. 155.) Such a tumor, in such a situation, must have been of congenital origin, due to the misplacement of some of the embryonal elements from which muscular tissue is developed, and it shows that from such germ tissue the structural condition found in pseudo-hypertrophic paralysis may arise.

The essential lesion is the growth of connective tissue, by which the fibres are damaged, whether fatty tissue is formed or not. Indeed, the fibres seem to suffer more when there is only fibrous tissue than when there is the fatty deposit. It is chiefly the fat that causes the enlargement of the muscles. There seems to be a tendency to the formation of fat in the early stage of the disease, and to its removal in the later stage, since in the former the muscles may often be observed to increase in size, and in the latter they become smaller, partly from some removal of fat and partly from the increasing atrophy of the muscular fibres. It is then that the greatest impairment of power occurs. In the cases in which the muscles are small from the first the tendency to the deposit of fat seems slight, and the condition is an almost pure muscular sclerosis. The late shortening which occurs seems to be due to the contraction of the interstitial fibrous tissue, and it often coincides with the shrinkage from the removal of some of the fat and the atrophy of the muscular fibres.

If it be true that the latissimus dorsi and lower half of the pectoralis are sometimes congenitally absent in pseudo-hypertrophic paralysis, the fact is quite consistent with the explanation of the nature of the disease given above. These two muscles stand perhaps lowest, in functional importance, among the muscles of the body, being used chiefly for the rare depression of the arm against a resistance (see p. 49). It is readily intelligible that a defect in the embryonal tissue of the muscular system should be quantitative as well as qualitative.

The loss of the knee jerk is sufficiently explained by the muscular lesion. According to the theory that the irritability on which the jerk depends is due to a muscle-reflex action, the loss is readily intelligible, since the afferent impulse is due to the stimulation, by tension, of the sensory muscle nerves, and these end (or begin) in the interstitial tissue which is the seat of the primary morbid process in this disease. But the wasting of the muscular fibres must also be capable of abolishing the knee jerk when the atrophy reaches a considerable degree, and it is also possible that the motor nerve endings share the structural damage. On any theory of the nature of the knee jerk, this will explain its loss. It is only when the muscular changes have attained a considerable degree that the loss occurs. The fact therefore does not, in itself, suggest any lesion of the spinal cord.

Diagnosis.—The diagnosis of the disease is usually easy, if its characters are known. The peculiarity of gait and the mode of rising from the floor, the age of the patient, and the progressive character of the impairment, are in themselves sufficient to suggest the disease, and examination reveals enlargement and often contraction of the calf muscles, and a change in the size of others, which confirm the diagnosis. The mode of rising is not absolutely pathognomonic, because it is present in other diseases which cause a gradual weakening of the extensors of the knee, but such gradual paralysis is exceedingly rare in early life, from any other cause. Hence the peculiarity is of great diagnostic value, greater even than might be anticipated.

Of the condition of the muscles, that which is most characteristic is the combination of enlargement of the infra-spinatus combined with a wasting of the latissimus and lower part of the pectoralis. I pointed out some years ago* that this condition, when it exists, is of very high diagnostic importance, and subsequent observations have fully confirmed the opinion. The condition is, moreover, rarely absent. The cases in which the latissimus is not wasted are often untypical, and the diagnosis frequently cannot be made with absolute certainty. Next in importance is the enlargement of the calf muscles, especially in combination with contracture that cannot be overcome. A distinct enlargement of part only of the extensor of the knee is also of very great diagnostic significance. It is important, however, to remember that enlargement of muscles is not an invariable symptom of the disease. They may be gravely diseased, and yet of normal size, or from the first smaller than normal. In such cases they are often hard, and the distribution of the atrophy is the same as that of the double change in typical cases. But some cases of this character properly belong to the form described in the next section.

The disease which is most frequently mistaken for pseudo-hypertrophic paralysis is the so-called "congenital spastic paraplegia," described at a preceding page. Both diseases affect children; in both there are weakness of the legs and contraction of the calf muscles, and in both the muscles are frequently large. The chief points in the diagnosis have been already mentioned. The most important are the preservation and excess of the knee jerk in spastic paraplegia, the tendency to spasm of the legs, the facts that the contracture is active, and can be overcome, that the patient does not rise from the ground in the way peculiar to pseudo-hypertrophic paralysis, and the opposite tendency of the two diseases. Between spinal muscular atrophy and this disease a difficulty in diagnosis can arise only in untypical cases in which no muscles are large. The age of the subjects is different; the spinal form involves the hand muscles in most cases, and rarely affects the legs more than the arms, while in many cases the legs are the seat of spasm.

The form of idiopathic muscular atrophy described in the next section is allied to pseudo-hypertrophic paralysis, and if a diagnostic difficulty arises it will chiefly be as to the category in which a case should be placed in which there is no conspicuous muscular enlargement. The most important distinction is the freedom of the calf muscles from enlargement in the atrophic form. The face is never affected in pseudo-hypertrophy as it is sometimes in idiopathic atrophy. If more than one member of a family is affected, some of the sufferers will usually present characteristic symptoms if the malady is of the pseudo-hypertrophic type, for it is remarkable with what constancy the two forms remain distinct in the various families (see p. 394). If an excised fragment presents no increase of interstitial tissue, pseudo-hypertrophy is excluded, but the presence of some fibrous tissue between the muscular fibres does not prove that the case is of the pseudo-hypertrophic type, since some muscles may present this change in a case of the atrophic form.

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* "Pseudo-hypertrophic Muscular Paralysis," London, 1879.

Prognosis.—In any case of pseudo-hypertrophic paralysis the prognosis is most grave. It is almost certain that each year will bring increasing disability, and that the patient will not reach adult life. It is only when the disease develops late, and the symptoms do not become considerable until after twenty years of age, that there is a possibility that the disease may not attain its ultimate degree, but even in such cases this hope is seldom realized. In any case, and at any age, it is unlikely that the patient will live more than seven years after the power of standing is lost.

Treatment.—Pseudo-hypertrophic paralysis is one of those diseases against the essential elements of which the physician is powerless, and will probably remain so. So far as our present knowledge goes, a developmental tendency to tissue growth is beyond control. It is not surprising, therefore, that, of the many drugs which have been given in this disease, not one has been found to exert a distinct influence. Arsenic and phosphorus have been thought by some to retard the progress of the weakness, but in no case has permanent arrest been obtained. In a malady of which the course is so prolonged, and in which the process of failure is so gradual, it is easy to fancy that arrest has taken place when the course of the disease is not actually retarded. The stimulation of the muscles by electricity has been employed and advocated, but, however sedulously employed, I have never seen distinct effect from the use of either faradaism or voltaism. Indeed, we have no facts whatever to justify the expectation that any form of electricity that could be applied to the muscles, would influence the interstitial growth of fibrous tissue, or that any electrical stimulation of the fibres can save them from the destructive influence of the compression they endure. It must be remembered, moreover, that electricity is a very feeble agent in stimulating fibres to growth, compared with the physiological stimulus of voluntary effort.

Muscular exercise does seem to have some influence in retarding the failure of power. It may perhaps cause some growth, or increased capacity for contraction, in the muscular fibres that have not yet suffered. It is possible, also, that it may, to some extent, divert the trophic energy from the interstitial tissue, since cessation of muscular exercise is certainly followed by quicker failure of strength. Hence it is desirable that the patient should carry out carefully-planned gymnastic exercises, so arranged as to call into action the muscles that most need help. These, thoroughly persevered in, have seemed, more than any other means, to retard the disease. But they have not in any case arrested it. Rubbing and massage are useful, combined with passive movements, in lessening the tendency to muscular contraction and consequent deformities.

The influence of muscular exercise renders it very important to keep the patient on his legs as long as possible. The ability to stand and walk is generally lost, through the contraction of the calf muscles, some time before the muscular weakness would take the patient off his feet. In such cases, tenotomy may restore the power of walking for some years, and when contracture returns, its removal has, a second time, set the patient on his feet again. The operation is thus distinctly beneficial. It should be performed without delay as soon as the actual need for it arises, and division of the tendon is far better than the removal of the deformity by splints, which requires a certain time during which additional structural damage will occur.

During the later stages of the disease great care is required to preserve the patient from catarrh, which helps to excite the pulmonary mischief that so often ends life. Similar care is also needed during any intercurrent malady which the patient may contract.

SIMPLE IDIOPATHIC MUSCULAR ATROPHY.

The name "simple idiopathic muscular atrophy" is perhaps the most convenient term under which to include those forms of widespread muscular wasting which differ in their characters from the pseudo-hypertrophic disease, and which, according to present evidence, are not central in nature. The affection is a rare one, and the cases present wide differences in character and course. But they possess certain common features by which the disease can be identified with considerable confidence. Of these the most important is the remarkable tendency of the affection to occur in many members of the same family.*

The separation from the pseudo-hypertrophic form is not an absolute one. Intermediate cases are met with, usually isolated, and it may be difficult to decide in which category they should be placed. An obvious distinction is the presence or absence of enlargement of muscles, but we have seen that this is a very inconstant feature of the pseudo-hypertrophic form. Nevertheless there are other differences which will be considered in the account of the symptoms, and it is remarkable with what constancy the two forms keep separate. In those families in which simple muscular atrophy occurs, however numerous the cases, however different the distribution of the disease, and however various the ages at which it begins, no cases present the distinctive characters of pseudo-hypertrophic paralysis.* It may reasonably be anticipated that there is some absolute pathological difference between the two diseases, underlying the difference in their clinical characters and their separate incidence. The anticipation may seem to be verified by the fact that in some cases of simple atrophy there has been no increase of interstitial tissue, but merely a primary wasting of the muscular fibres. But in some other cases there has been such interstitial overgrowth in some muscles, and this when not one of many cases in the family resembled pseudo-hypertrophic paralysis. Hence it is doubtful whether the presence or absence of an increase in the interstitial tissue can be made a ground of separation, although it is certain that, in some cases of simple atrophy, there is no such increase, and on the other hand that it is invariable in pseudo-hypertrophy.

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* The most important writings on the subject are those of Duchenne in "Électrisation Localisé" (p. 60 of Poore's translation, published by the New Sydenham Society); Barsickow, "Inaug. Dissert.," Halle, 1872; Leyden, *Klin. d. Ruckenm. Krank.*, Bd. ii, p. 525; Möbius, "Hered. Nervenk.," Volkmann's *Klin. Vorträge*, No. 171; Erb, *Deut. Archiv f. klin. Med.*, Bd. 34, 1884, and *Neurol. Centralbl.*, July 1st, 1886; Landouzy and Déjérine, *Revue de Méd.*, *1885*, pp. 81 and 251.

Among the symptomatic varieties presented by idiopathic muscular atrophy it is easy to distinguish certain forms, especially in regard to the part in which the wasting begins. But if we regard these as distinct types, and attempt to test the validity of the types by ascertaining how far they are associated with differences in the conditions under which the disease occurs, we discover at once that the symptomatic differences do not furnish grounds for the distinction of definite varieties. The types between which there is the most marked difference may all occur in the same family. For instance, the atrophy sometimes commences in the face, and Duchenne described this commencement as a characteristic of one variety of muscular atrophy. But in a family in which the atrophy commenced in the face in most cases, it commenced elsewhere in one or two members, and conversely the face has suffered first in one member of a family although the disease began elsewhere in all the others, and the face escaped altogether in some. Thus the test of association invalidates our subdivision of the cases of simple atrophy, as distinctly as it serves us in the separation of simple atrophy from the pseudo-hypertrophic form.

Causes.—The ultimate cause in all cases, and in most the only cause which can be traced, is a congenital tendency, often clearly inherited. The proof of this tendency is the occurrence of many cases in the same family, and the evidence of its inheritance is the distribution of the cases through more than one generation. In one remarkable series recorded by Barsickow, twenty-four cases were distributed through five generations, and the disease was also traced through five generations in a group described by Landouzy and Déjérine. It is very rare for the disease to be confined to one generation, far more rare than for pseudo-hypertrophic paralysis to be so confined. As in the case of most congenital hereditary diseases, cases that are apparently isolated are occasionally met with. Such isolated cases are rare; how rare we cannot at present say, because it has generally been the family tendency that has suggested the idiopathic nature of the disease, and it is probable that many isolated cases have been thought to be spinal, the more readily since the disease has little tendency to cause death, and its real nature, if not suspected during life, has seldom been discovered. That isolated cases occur is undoubted. In the families of the patients shown in Figs. 150–3 and 154 no example of analogous disease could be heard of.

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* This is an important fact. We must not actually unite in our classification forms of disease which are habitually separate in occurrence.

Both sexes suffer, and it is doubtful whether either sex is the more prone to the disease. In a few families, females have suffered chiefly, in others males, but in most groups the individuals affected have been of both sexes. The age at which the disease first manifests itself is extremely variable. It may begin as early as three and as late as sixty years. But the onset is rarely during childhood; in the majority of cases the disease shows itself between fifteen and thirty-five; that is, during the later period of growth and the early period of adult life. In the same family, the variations may be extreme. In that of which the history has been given by Barsickow the date of the onset of seventeen cases was known; in one the disease began at twelve, in four between fifteen and twenty, in seven between twenty and thirty, in three between thirty and forty, and in two after forty. Sex has no influence on the date of onset, nor, as a rule, can any relation be traced between the date and the place at which the wasting begins. When the wasting begins in the face, the disease more frequently commences in childhood than when the first wasting is in the limbs; but, in some instances, the atrophy has commenced in the face late in life, and this in the same family in which other sufferers have been young.* Thus, in the sexual proclivity, and in the date of onset, there is a marked difference between this disease and pseudo-hypertrophy; the latter showing a strong tendency to affect males, and to manifest itself in childhood.

As a rule no direct exciting cause can be traced. In a few instances the onset has succeeded some other morbid process,—such as chlorosis, acute disease, rheumatic affections due to exposure to cold,—and the depression of general health resulting from these may have determined the time of onset. But in other individuals of the same families the disease has developed without the aid of any exciting influence.

Symptoms.—The onset is always gradual. Weakness and wasting come on together, and are noticed simultaneously, unless the commencing atrophy is concealed by subcutaneous fat. The atrophy generally begins in the upper arm muscles, the legs, or the face; the most frequent commencement is in the arms, but in some families there has been a tendency to commencement in the face, in others in the legs. The face was first affected in the lad figured on p. 398. In the part first affected the disease slowly increases, and thence it usually spreads. The onset may be symmetrical on the two sides, or one side may suffer some time before the other.

Of the arm muscles the weakness and wasting are noticed first in the biceps and triceps, and with these the supinator longus often suffers. But examination generally shows that the lower part of the pectoralis and latissimus dorsi are greatly wasted, a loss of which the patient may be little aware, on account of the relative unimportance of these muscles. Sometimes the upper part of the pectoralis, and even the pectoralis minor, are also affected (Fig. 151). The tendency to atrophy of the lower part of the pectoralis and latissimus is a character common to this disease and pseudo-hypertrophic paralysis, but not invariable in either. The deltoids are rarely involved; in many cases they are normal; sometimes they have been thought to be unduly large; in a few instances they have been wasted (Fig. 154). The serratus magnus is often affected (Fig. 153), but may escape, even in a severe case. The supra-spinatus, infra-spinatus, and subscapularis are rarely involved; the trapezius and rhomboids have been affected in many cases, sometimes entirely atrophied (Fig. 153).

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* It seems therefore undesirable to form a separate variety of "juvenile muscular atrophy," as Erb has proposed. Spinal atrophies may also be juvenile.

The forearm muscles generally escape, with the exception of the supinator longus, which usually wastes entirely when the other forearm muscles are intact. Occasionally there has been some weakness of the long extensors or flexors of the fingers, with or without slight visible wasting. In the case mentioned on page 398, the extensors of the phalanges of the thumb were involved on the left, and the radial extensor of the wrist on the right side. Rarely the forearm muscles have been much atrophied. In several instances there has been some atrophy of the small muscles of the hands, the thenar and interosseal muscles (as in a case presently to be described), or the interossei only, as in the case of Landouzy and Déjérine, but in the majority the integrity of these muscles was a marked feature of the disease, and a contrast to the spinal form.

The affection of the face is peculiar. There is commonly a failure of the zygomatic muscles, and in consequence a loss of the naso-labial furrow, and a curious alteration in the smile; instead of the angles of the mouth being drawn outward and upward, they are moved upward by the elevators of the upper lip and angle of the mouth. The orbicularis oris is also affected, and in consequence the lips are habitually separated, the lower lip projects, the patient cannot "pout," and the articulation of labials is imperfect. The face has a dull expression and the aspect is very peculiar; it has been termed, by Landouzy and Déjérine, the "myopathic face." Rarely (as in Fig. 151) the frontales have been involved, and the orbicularis palpebrarum has been weak. In this case the affection of the face is unusually complete. Wasting of the muscles may be indistinct, because the contour of the face is only to a slight degree influenced by the substance of the muscles. The projecting lower lip may, indeed, appear to be thicker than normal. In many cases the buccinators have been affected, in some instances they have been normal, and then have drawn out the angles of the mouth in smiling. The tongue has been always unaffected, and so also have the pharynx, larynx, muscles of mastication, and the eyeball muscles.*

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* In a singular case under my care some years ago, an affection of the facial muscles, similar to that of idiopathic muscular atrophy, was associated with paralysis of ocular muscles. The patient was a girl of twenty-seven years of age; there were no indications of syphilis,

The muscles of the spine have sometimes been normal, sometimes they have been considerably atrophied. The intercostals are often affected in the later stages, but rarely in extreme degree. The diaphragm also sometimes suffers, as in the case shown in Fig. 150. The abdominal muscles have been involved in only a few instances. In the legs the muscles most commonly affected are the flexors of the hip, the extensors of the knee, and less frequently the glutei. The muscles below the knee have escaped in many cases; when they have suffered, the atrophy has generally involved the peroneal group, and the calf muscles have been normal, a very marked contrast to pseudo-hypertrophic paralysis. The calf muscles, however, shared the general wasting in the case shown in Fig. 154.

The electric irritability of the affected muscles is usually lessened in proportion to the wasting, and equally to both currents. Indeed, the diminution is often out of proportion to the wasting, and may be great when the atrophy is slight. There is no trace of degenerative reaction, and there is not even the longer persistence of voltaic than of faradaic irritability which occurs in the more chronic cases of spinal atrophy. Fibrillation is also absent, as an almost invariable rule; it has been observed only twice. Myotatic irritability is lessened or lost, it is never increased. Some shortening of muscles has been occasionally noted, frequently in the calf muscles, rarely in the biceps.

All other functions of the nervous system are unaffected. Sensibility is normal. In a few cases transient rheumatoid pains have accompanied a rapid development of the disease in the arms, but in the vast majority of cases the disease is painless. The sphincters are unaffected, and there is no tendency to trophic or vasomotor disturbance.

As the muscular atrophy progresses, the form of the affected parts becomes changed just as in spinal atrophy. Deformities may also occur, chiefly from the shortening of unaffected muscles, but these rarely reach the considerable degree common in other forms of muscular atrophy. Lordosis occurs in the upright posture, and is probably due to the same mechanism as in pseudo-hypertrophic paralysis, ceasing, as in that disease, when the patient sits (compare Figs. 150 and 153). When the muscles of the lower leg are involved, talipes may develop.

The course and duration of the disease are exceedingly variable. The

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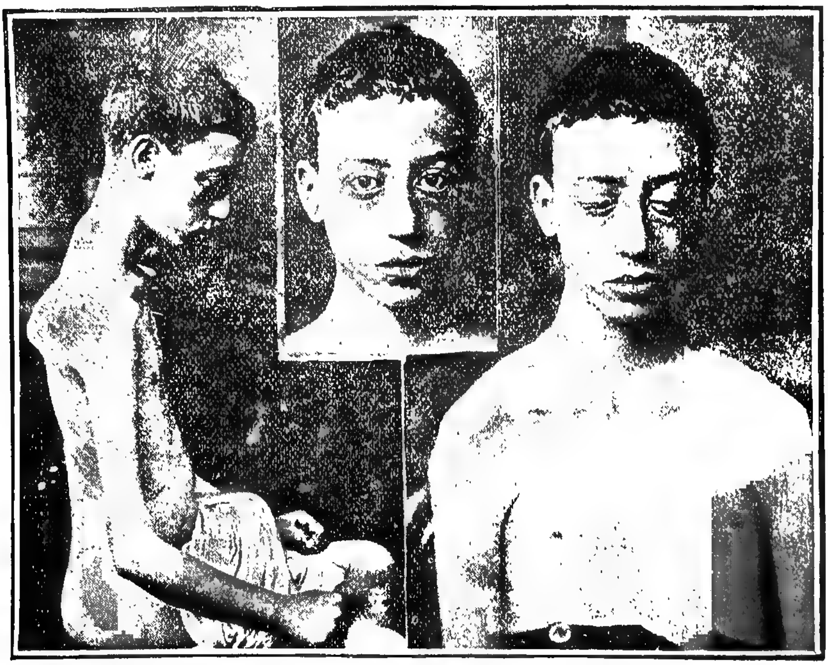
nor could any history of muscular atrophy in the family be ascertained. The ocular palsy commenced gradually at twenty-four, and increased until the movements of both eyes upward, of the left eye inward and the right outward, were lost, and all other movements were weakened. The eyelids drooped slightly; the internal ocular muscles were normal. The affection of the face followed that of the eyes; the zygomatic muscles were powerless, so that the smile consisted only in elevation of the upper lip; the orbicularis was weak. The palate, pharynx, and larynx were normal. The arms became feeble, and the flexors of the hips almost powerless. There was no visible change in the nutrition or electrical irritability of the muscles; the knee jerk was normal. If the case was central, as it appeared to be, the peculiar affection of the lips and zygomatics is not confined to idiopathic muscular atrophy. If the disease was muscular, the eyeball muscles do not invariably escape.

atrophy may remain limited to the part in which it begins. The face alone has been affected in some members of a family, although in other members the limbs subsequently suffered. In cases in which the wasting spreads, years may intervene before the extension takes place. In one case, for instance, the right arm became affected at nineteen, the left at twenty-five, the legs at thirty. In another the arms began to waste at thirty-five, power of

FIG. 150. FIG. 151. FIG. 152.



FIGS. 150 TO 153.—Idiopathic muscular atrophy affecting the face. Fig. 150 shows the wasting of the deltoid, upper arm muscles and supinator longus, and the rotation of the scapula from the loss of the trapezius and rhomboids. Fig. 151 shows the habitual appearance of the face, and Fig. 152 the greatest possible movement of the facial muscles in closing the eyes and smiling, and also the wasting of the deltoids and pectorals. The general distribution of the atrophy is seen in Fig. 153 *

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* The patient is a lad, aged sixteen. No family history of any similar affection. He had primary and secondary syphilis at three. The wasting began gradually during childhood; movement of the face almost ceased at six; walking became difficult at twelve. Now, the face is almost motionless; lips full and can be brought together, but not shortened. Eyeballs prominent, their movements normal, and also those of masseters, tongue, pharynx, and larynx. Sterno-mastoids small, amohyoids large. Quite atrophied are the diaphragm, trapezii, rhomboids, deltoids, pectorales, latissimi, serrati, biceps, brachiales, triceps, supinators, the left extensors of the phalanges of the thumb, and right ext. carpi radialis. Unaffected are the other forearm and hand muscles, the lev. ang. scap., supra- and infra-spinatus, the last being large, probably from over-use. Erectors of spine feeble, intercostals strong. Extensors of hip and flexors of knees normal; flexors of hip and extensors of knee very feeble, the latter small; muscles of lower leg small and feeble, peronei powerless; some contraction of calf muscles. Knee jerk absent. Electric irritability very low to each current in affected muscles; in most, no contraction to be obtained. Functions of nervous system normal.

standing was only lost at fifty-five, and the patient lived till seventy-five. Even when the malady begins in childhood its progress is sometimes very slow. Thus in one case the affection of the face was first noticed at five, at twelve the arms began to suffer, and a few years later the flexors of the hip became weak, but the patient was still able to walk at the age of forty. On the other hand, the progress of the disease may be more rapid and uniform, and the atrophy may reach its widest distribution in eight or ten years. In most cases, even of severe type, the wasting does not become universal, but remains limited to the muscles mentioned above. Occasionally hardly any muscles of the body may escape. An example of such rare universal atrophy is shown in Fig. 150, and the case is also noteworthy from the commencement in a somewhat rare seat, the lower legs, and in the degree of involvement of the hands. The patient was a man aged twenty-seven at the time of his death (in University College Hospital). No history of any analogous case in his family could be ascertained. The affection began at the age of fourteen, when his feet began to turn in so that he walked on the outer side of the foot, and soon he noticed gradual wasting of the legs, which slowly progressed, and about the age of twenty-four the arms also began to suffer. When first seen, a few months before his death, the muscular atrophy was universal, and the subcutaneous fat had also disappeared. Even the hands were greatly wasted; there was a hollow in the position of the thenar eminence, just as in progressive muscular atrophy, and the interosseal muscles were also greatly wasted. There was slight fibrillation. The intercostals were paralyzed. The wasting of the legs was extreme; the maximum circumference of the calf was only seven inches; the feet were inverted, the sole hollowed, and the toes flexed. The patient died from diarrhœa. The muscles were found to be small and pale. The chief microscopical change was intense granular and

Fig. 153.

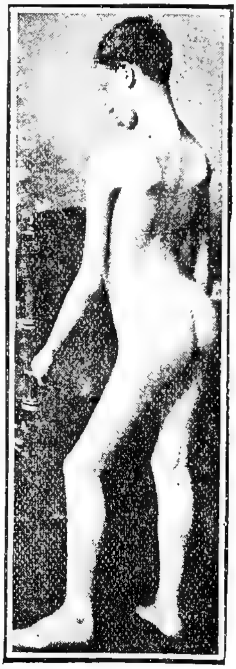
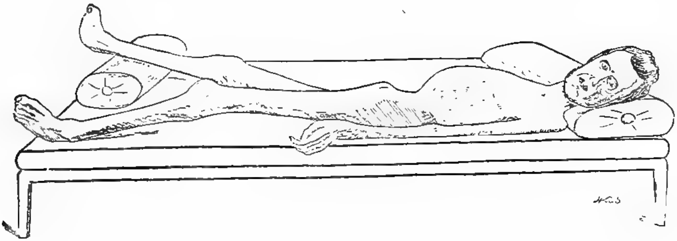


Fig. 154.



Idiopathic muscular atrophy. (*Drawn by Dr. Spencer, from a photograph.*)

fatty degeneration of the fibres. A very few normal fibres were seen in some muscles. There was no increase of interstitial tissue. Microscopical examination revealed no morbid changes in the spinal cord.

The duration of the disease varies from ten to fifty years. Death has never occurred as the direct result of the malady. In the cases of most severe degree and rapid course, the patient has usually died from phthisis, probably related to the deficient breathing power, just as in the sufferers from pseudo-hypertrophic paralysis. In most cases, however, death has been due to other maladies, and has not been in any degree the consequence of the muscular disease.

Pathological Anatomy.—The pathological facts are limited to two or three post-mortem examinations, and a few observations on excised fragments of muscles. The conditions found have varied much. In the case recorded by Landouzy and Déjérine, simple narrowing of the muscular fibres was the chief morbid appearance. In some cases excised fragments have shown considerable increase in the interstitial tissue; in the case shown in Fig. 154, a granular and fatty degeneration of the muscular fibres was the only change. In some excised fragments, fibres have been seen larger than normal, but whether these were really hypertrophied, or merely thickened by contraction, is uncertain (see note on p. 402). Longitudinal striation and fissuring of the fibres has been described by Erb. The spinal cord and nerves, when examined, have been found normal. The condition is thus diverse, and it is difficult to conceive that they can all be results of the same cause, and manifestations of the same disease. But it is to be noted that in cases of the pseudo-hypertrophic type considerable diversity has been met with in the muscular changes. So far as our present knowledge goes, the muscular changes must be ascribed to a congenital tendency by which they perish prematurely. The tendency may lead to overgrowth of the interstitial tissue, or to simple wasting of the fibres, or to early decay, either in consequence of a simple difference in the characters of the tendency, or more probably by the coöperation with it of other constitutional influences, congenital or acquired.

Diagnosis.—The two most important diagnostic indications are the affection of more than one member of the same family, and the onset of the disease before adult life is reached. The former is alone conclusive; but the latter should always suggest the probable idiopathic nature of the case. In isolated cases the diagnostic difficulty is much greater, and no indication is actually conclusive, except, perhaps, the affection of the face. Commencement during childhood is also of great weight. It is most unlikely that progressive muscular atrophy, beginning under ten, is of spinal origin, but I have known atrophy, almost certainly spinal, to commence at the age of fourteen. The peculiar affection of the zygomatic muscles is very characteristic, but we cannot yet say that it is pathognomonic (see note on p. 396), and the affection of the lips must be carefully distinguished from that due to the bulbar palsy so commonly associated with spinal atrophy. Of the distribution of the wasting, that of the latissimus and lower half of the pectoralis, and the escape of the hand muscles, are the most important characteristics, but they are not, by themselves, conclusive, since they are sometimes met with in spinal atrophy.

Other most important distinctions between spinal and idiopathic atrophy depend upon the symptoms of the former, which are absent in the latter. Among these are the affection of the tongue, palate, and larynx, the commencement of atrophy in the hands, and the excess of the knee jerk which results from lateral sclerosis. The latter is a conclusive indication of spinal disease, and therefore renders the spinal origin of the atrophy all but certain. The distinction from the pseudo-hypertrophic form has been already considered in the account of this disease.

PROGNOSIS.—The extreme variations in the course of the disease render the prognosis in any individual very uncertain. Unfortunately, recovery from atrophy that has actually occurred cannot be anticipated, but on the other hand, the disease may remain limited to its first seat, or may not involve other parts until after many years. In a considerable number of cases, perhaps one-half, the malady has not appeared to shorten life. Even in cases that begin during youth it is therefore possible that the patient may reach old age.

TREATMENT.—It is not yet clear that idiopathic muscular atrophy can be influenced by treatment in any considerable degree. Most published records of cases are almost silent on the subject, and the disease is so rare that few individuals have an opportunity of forming an opinion. It might be assumed that the essential cause of the disease, a congenital tendency, withdraws it from the range of therapeutics. But the course of the malady is very different from that of some other diseases which own a similar cause. The extreme variations in the date of onset, the fact that the disease may not be manifested until late in life, and the long period that may intervene between its onset and extension, all suggest that other influences coöperate with the congenital tendency in determining the development of the malady. It is quite possible, therefore, that the first inference from the history of the disease may not be altogether correct, although it must be admitted that we have not as yet any evidence that the disease is susceptible of influence from drugs. Electrical treatment and massage have been thought to do good and even to arrest the disease (Erb), but the variable tendency of the malady renders the evidence of arrest insufficient. If voluntary exercise is practicable, this is a far more efficient stimulus to muscular growth than any electrical application, and it is probable that such exercise, carefully persevered in, may do something to prevent the occurrence of the malady in those predisposed to it, and even to retard its progress in those who are already attacked. In this connection it is noteworthy that very few of the sufferers have been engaged in occupations that involve active muscular exertion. Over-exertion should, however, be carefully avoided. The general health should be attended to, and any defect removed as speedily as possible, both in those who suffer and in those who are related to sufferers, since, as we have seen, depressing influences may apparently excite the development of the disease, and it is therefore reasonable to suppose that they may accelerate its progress, and lessen any tendency there may be for the morbid process to become stationary.

MUSCULAR HYPERTROPHY.

The occurrence of a true hypertrophy of muscles, as a condition of disease, is exceedingly rare. In most cases in which the muscles are enlarged, the increase in size is due to a growth of interstitial tissue, fatty or fibrous, such as has been described in the chapter on pseudo-hypertrophic paralysis. We have seen that muscular fibres larger than normal have been met with in that disease, and also in some muscles of normal or increased size in cases in which other muscles were simply atrophied. But this increase in size has been met with only in excised fragments, and it is possible that the condition may have been due to a vital contraction excited by the process of excision. The same ground of doubt exists in all cases in which a true hypertrophy has been described; the fact rests upon observations made on excised fragments, and has not yet been observed after death.*

The condition has been found in the singular malady described in the next section (Thomsen's disease). It has also been met with, in very rare instances, as a widespread or partial condition, usually associated, strange to say, with either diminished power or with a morbid readiness of fatigue.† The muscles most frequently affected have been those of the shoulder and upper arm, or of the thigh and calf, on one side or on both. It has also been observed in the glutei, spinal muscles, and trapezii. The causes are obscure, but it has been ascribed to over-exertion. The diameter of the fibres has been increased to double the normal size, a maximum of $\frac{1}{200}$ inch having been met with (Eulenberg), whereas the normal maximum may be taken as $\frac{1}{400}$ inch. An increase of the nuclei has been observed, without any overgrowth of the interstitial tissue.

The condition is manifested by an increase in the size of the muscles, which are also firm. The circumference of the limb is greater than normal, and when the change is unilateral, the difference between the limbs of the two sides may be very striking. The maximum circumference of the calf has been as much as seventeen inches. The muscles are soon exhausted, and have been weak in some cases, while in others there has been abnormal strength for brief exertion. The electrical, mechanical, and myotatic irritability of the muscles has usually been found normal.

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* Since the above was written, the doubt there expressed has been confirmed by an observation made for me by Dr. H. R. Spencer. A fragment of the gastrocnemius of an amputated leg was excised immediately after the amputation, and the fibres were compared with those of the muscle twenty-one hours later. In order to ascertain more definitely whether a vital contraction persisted as an apparent increase in size, a part of the excised fragment was separated and faradized. The average size of the fibres in the muscle was $\frac{1}{740}$ inch, in the fragment excised $\frac{1}{825}$ inch, in the fragment faradized $\frac{1}{417}$ inch. Since the division of the nerve during the amputation may have caused some contraction, it is possible that the difference in size produced by excision may be even greater than these figures represent

† Auerbach, *Virchow's Archiv*, Bd. 53, pp. 234 and 397; Berger, *Deut. Archiv f. klin. Med.*, Bd. 9, 1872, p. 363; Friedreich, "Ueber Prog. Muskelatrophie, etc.," 1873; Eulenberg, "Real-Encyclopädie," Bd. ix, p. 354.

The disease may be suspected if a marked increase in size in an adult is accompanied by impaired power of sustained exertion; if the muscles are firm, and the patient does not manifest other indications of pseudo-hypertrophic paralysis. The diagnosis can, however, only be made with certainty by the microscopical examination of an excised fragment. The meagre facts regarding the course of the disease suggest that it usually persists without getting either better or worse. Treatment appears to have but little influence upon it.

A singular case has been reported by Eulenberg,* which differs in many particulars from the form of disease above described and illustrates the complex relations of enlargement of the muscular fibres and its connection with degenerative processes. A man aged thirty-six presented an enormous enlargement of the muscles of the left leg, which were soft and flabby and weak, with lowered irritability. The condition had slowly developed after a fall on the back at ten, which caused imperfect paraplegia, motor and sensory. A year later, during pleurisy, he had thrombosis of the left femoral vein. In an excised fragment of the gastrocnemius the muscular fibres were large, the maximum being $\frac{1}{276}$ of an inch, and presented fatty and vitreous degeneration, and fat was seen between the fasciculi. The muscles of the other leg were somewhat wasted, but the fibres were also enlarged and degenerated. It would appear as though the condition had resulted from a traumatic lesion of the spinal cord, and had been intensified in the left leg by the influence of the thrombosis and resulting vascular disturbance.

THOMSEN'S DISEASE.

The malady thus designated may be considered here, notwithstanding the obscurity of its nature, because it has at least these points in common with the disease last described, that the symptoms are muscular and that the disease is commonly congenital and occurs in families. It has been named after the physician whose description gained for it general notice, and who is himself its subject, but it had been previously described by Leyden, and hinted at, long ago, by Sir Charles Bell.†

The disease is characterized by a peculiar rigidity of the muscles, which comes on when they are first put in action after a period of rest. The rigidity is transient, and when it has passed off it does not return as long as exertion is continued. The malady is usually congenital and often hereditary, affecting many members of the same family. In that of Thomsen, cases can be traced through five generations. It appears to be more common in Scandinavia and Germany than in France and England, but it is a rare disease; only about thirty instances have been recorded, and it is probable that these are all the cases in which the affection has been recognized. At the same time there is little to attract attention in the slighter forms of the disease, and it may ultimately be found to be less rare than at present it appears to be.

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* *Deutsch. Med. Wochenschrift, 1885,* No. 12.

† The most important papers on the disease are those of Leyden, *Klinik der Rucken-markskr., 1874,* Bd. i, p. 128; Thomsen, *Arch. f. Psychiatrie,* Bd. 6, 1876, p. 702, and *Centralbl. f. Nervenkr., 1885,* p. 193; Bernhardt, *Virchow's Archiv,* Bd. 75, 1879, p. 516, and *Centralblatt f. Nervenkr., 1885,* p. 122; Ballet and Marie, *Archives de Neurologie, 1883,* No. 13; Möbius, *Schmidt's Jahrb.,* Bd. 198, 1883; Ringer and Sainsbury, *Lancet, 1884,* pp. 767, 816, and 860. The mention of the symptoms by Sir Charles Bell is at p. 436 of his "Nervous System," Case 184, "Affection of the Voluntary Nerves."

Both sexes suffer. In most cases the symptoms have been first noted in early childhood, at four or five years of age, sometimes even in the cradle. They increase during the period of muscular development, and then remain stationary. In a few instances the patient has seemed free until about the period of puberty, but even in such instances it is probable that the disease is congenital in origin, although its manifestation is deferred, since the cases have occurred in families, other members of which have suffered earlier. But in a few cases symptoms apparently identical with those of the congenital disease have come on in early manhood after some exciting cause, in one case a lightning stroke, in another a sudden alarm.* In such cases the disease has been apparently acquired.

The characteristic symptom is tonic spasm of the muscles when they are put in action after a period of rest. As soon as the patient attempts to move, the muscles become rigid. The rigidity may make movement impossible, while it lasts, or may merely lessen the possible range of movement. After a few minutes or less the spasm passes away, to be renewed, but in slighter degree, by a fresh attempt, and if the movements are persevered in, the spasm, in a few minutes, becomes trifling, and does not return until after rest; the individual can walk for hours without fatigue when the spasm has passed off. The rigidity is always increased by attention and by nervous apprehension of it, and the more the sufferer tries to overcome the stiffness, the less he is able to do so. In a severe case, a slight impulse will make the person fall, and it may then be impossible for him, for some minutes, to rise from the ground. The arms are usually less affected than the legs, but in some cases the rigidity fixes the fingers for a short time on an attempt to use the hand. The muscles of the face are usually free; mastication may, however, be interfered with by the spasm in the muscles of the jaw. Rarely the tongue has been affected; still more rarely the muscles of the eyeball. Sometimes the spasm is greater on one side of the body than on the other.

The muscles are always well nourished; they are often, indeed, above the normal size, and possess more than normal strength. Thomsen believes that the more the muscles are habitually used, the less severe is the spasm, and that a life of active exertion produces some permanent amelioration in the disease. It is, however, a source of some disability and of considerable annoyance; in the words of Thomsen, "it casts a shadow over the lives of the sufferers."

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* Engel, *Phil. Med. Times*, *1883*, p. 412; Schönfeld, *Berlin med. Wochenschr.*, *1883*, No. 27.

Careful investigations of the muscular phenomena have been made by several observers, and that by Erb is especially instructive. In his case a single brief effort caused tonic contraction which lasted for twenty-five seconds. Momentary electrical stimulation of the nerves caused only a momentary contraction of the muscles, but continued stimulation caused a prolonged contraction similar to that excited by the will. In certain muscles, moreover, an unbroken current caused peculiar wave-like contractions, about one per second, passing from the negative to the positive pole. Any strong stimulation of the muscle itself caused a similar after-contraction. The irritability of the nerves was normal, but the voltaic excitability of the muscles was unduly great, and they were remarkably sensitive to mechanical stimulation. Firm pressure caused a tonic contraction lasting from twelve to twenty seconds.

PATHOLOGY.—Of the morbid anatomy of the disease we know only that the muscular fibres, in excised fragments, have been normal in aspect in some cases, but in others, especially that observed by Erb, many fibres have been seen exceeding the normal maximum in size. The minimum fibres were the same size as in health, but the largest were twice as large as any met with in normal muscle.

The manifestation of the disease is in a disorder of the functions of the muscles, and most writers on the subject have followed Leyden in regarding the malady as essentially muscular in nature, as consisting in an altered functional condition of the muscular tissue. This opinion is strongly supported by the frequent alteration in electrical excitability, which can hardly be otherwise interpreted than as an indication of a change in the mode of action of the contractile muscular protoplasm. This opinion is supported, also, by the interesting experiments of Ringer and Sainsbury, who found that certain salts, such as sodium phosphate, are capable of causing in the frog tonic spasm bearing considerable resemblance to that of Thomsen's disease, and that such spasm persists, not only after the nerve has been divided, but after the intra-muscular nerve endings have been polarized by curara. Although this fact does not, of course, prove that Thomsen's disease is muscular in nature, it shows that a similar condition may have its origin solely in the muscles, and it harmonizes with the indications afforded by the muscular irritability in this disease. But a proof that there is a derangement of the function of the muscles is no proof that this is the sole element in the disease, and that it is not is at least suggested by two other facts. First, the disease has apparently resulted, in rare cases, from morbid influences acting on the nervous system in adult life, and, secondly, in the congenital cases, the spasm is increased by emotion. Wide as is the difference between the muscular and nervous tissues, we must remember that they have some conditions in common. The dependence of the nutrition of the muscles on that of the motor nerve fibres and cells is a very remarkable fact, and so also is the functional activity of the cells and fibres in causing a similar condition in the muscular tissue. To say that the two structures are connected is hardly an explanation of the fact. Whatever is the nature of the relation between them it is at least conceivable that an abnormal functional state, congenital in nature, may be common to the two, and that the peculiar over-action in the muscles may be accompanied by a similar over-action in the ganglion cells of the cord, and even in the pyramidal cells of the cortex. It is even conceivable that the condition of the nerve cells may be the primary change, and that of the muscles may be secondary, although, when produced, it is in some degree independent, and may be excited independently by local stimulation. Such a theory enables us to understand the two facts above mentioned, the influence of emotion and the acquisition of the disease, each of which seems to be inconsistent with a purely muscular pathology.

TREATMENT.—No treatment, properly so called, appears to exert any influence on the disease. The congenital malady persists through life; in the cases in which a similar condition has apparently been acquired, it has also been persistent. The only influence that has appeared to Thomsen to ameliorate the condition is a life of active muscular exertion.

Under the name of *Congenital Paramyotone* Eulenberg has described * a strange family affection allied to Thomsen's disease in its general character, although differing very much in its special features, and equally obscure in nature. The malady was widely spread in the affected family, and could be traced through six generations, but appears now to be dying out. Its congenital character was shown not only by its multiplicity, but also by the fact that in some individuals it was manifested immediately after birth. The symptom was tonic spasm, lasting from a quarter of an hour to several hours, excited chiefly by cold, although often by merely slight cold. The rigidity was followed for a time by weakness. The facial muscles were very prone to become thus rigid, especially the orbiculares palpebrarum and oris, and while the contraction lasted the patient was often unable to speak or to open the eyes. The rigidity was slighter in the legs than in the arms, but the subsequent weakness was equally marked. Warmth removed the spasm. There was no persistent loss of power, and no increase of mechanical irritability. The electrical excitability of the nerves was normal; that of the muscles was lowered to each current, and there was an abnormal tendency to tetanic contraction during the passage of the current. Eulenberg speculates that the symptoms may be due to reflex vaso-motor spasm in the muscles, because the diminution of the blood supply to muscles renders them weak; but it is clearly equally possible that the two certain phenomena, the sensory impression and the muscular contraction, may be directly connected.

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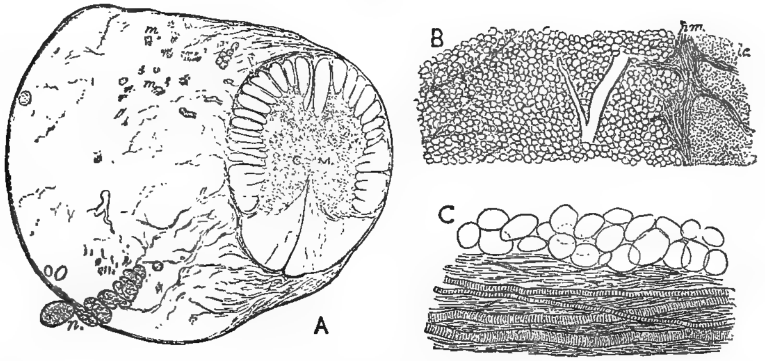
* *Neurologisches Centralblatt, 1886,* p. 265.

TUMORS OF THE SPINAL CORD.

Morbid growths within the spinal canal may spring from the membranes or may grow in the substance of the cord itself. The difference in seat entails some difference in symptoms, but it is nevertheless convenient to consider the two classes together, because they have many symptoms in common, and it is often impossible to carry the diagnosis farther than the existence of an intra-spinal tumor.

Etiology.—Of the diathetic conditions which give rise to tumors elsewhere, only two—syphilis and tubercle—are effective in causing growths which commence with the spinal canal. Parasitic tumors occur, due to the same influences which produce them in other situations. A few rare growths appear to be congenital in origin, due to the development of germinal tissue in an abnormal position. Of the causes of other forms of tumor we know practically nothing. Injuries, such as a blow on the spine, have been supposed to be occasional causes, and their influence in rare instances has seemed possible, but the evidence is not so strong as it is in the case of tumors in some other situations. In many cases the first symptoms have immediately followed some exposure to cold and wet, and it seems probable that this influence may have excited secondary processes in the nerve elements which were already deranged by the growth, but it can have had no share in the production of the growth itself.

Fig. 155.



Myo-lipoma of the spinal cord. A, transverse section of the tumor and of the conus medullaris of the spinal cord, to which the growth was attached, and of which the gray matter is represented by the dotted shading, C M. *n*, sections of nerve roots partly enclosed in the tumor; *m*, bundles of muscular fibres. B, part of the tumor more highly magnified, showing the fat cells of which it was chiefly composed; *p. m.* pia mater of the cord, of which *l. c.* is part of the lateral column C, part of the tumor, still more magnified, showing striated muscular fibres, fibrous tissue, and fat cells (see also Fig. 149, p. 389). The tumor had caused no symptoms.

Pathological Anatomy.—The growths within the spinal canal may develop outside the dura mater, inside the dura mater, or within the substance of the cord. The extra-dural tumors may spring from the membrane, or from the tissue between the membrane and the bone, or may grow into the canal from the outside, through the intra-vertebral foramina. Subdural tumors may proceed from the inner surface of this membrane, from the arachnoid, or from the pia mater (Fig. 157). The growths in the cord may

Fig. 156.

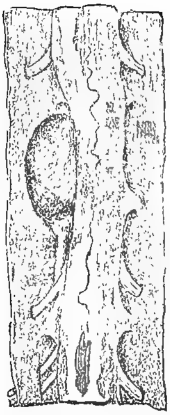


Fig. 157.

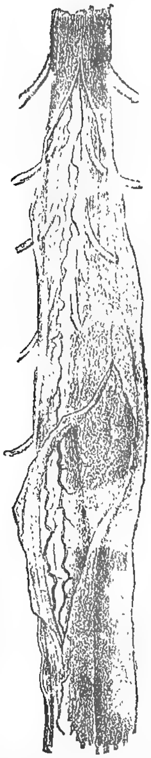


Fig. 158.

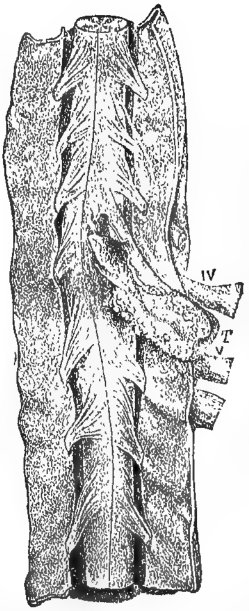


Fig. 159.

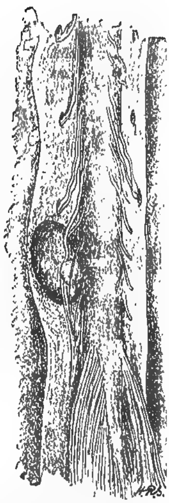


Fig. 156.—Tumor growing from the inner surface of the dura mater, and compressing the spinal cord in mid-dorsal region. The tumor, 3 cm. long, was a sarcoma in which the cells were arranged in concent groups, the centres of which had undergone calcification. The spina cord was softened and dark in tint the compressed part.* (*After Lancereaux.*)

Fig. 157.—Sarcoma growing between the arachnoid and pia mater in the mid-dorsal region of the cord. T tumor had sprung from the meshes of the arachnoid, and was only slightly adherent to the pia ma The spinal cord was compressed and softened. In the figure the dura-matral sheath has been laid op except at the top, and the arachnoid has been opened over the lower half of the tumor which, 5 cm. lo lies on the posterior and (right) lateral aspect of the cord.† (*After Lancereaux.*)

Fig. 158.—A tumor (T) growing from the dura mater, and compressing the right side of the spinal cord at origin of the 4th, 5th and 6th cervical nerves. The tumor was a spindle-celled sarcoma springing from arachnoid.‡ (*After Leyden.*)

Fig. 159.—Tumor of dura mater opposite the upper part of the lumbar enlargement, composing nerve r and spinal cord. (*From a preparation in University College Museum. Drawn by Dr. H. R. Spenc*

spring from the pia mater, or may develop in the substance of the cord. They sometimes proceed from the peculiar tissue which surrounds the central canal.

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* The patient was a woman seventy-one years of age, paraplegic, with strong flexor c tracture of the legs, loss of power over the sphincters, and considerable impairment, but absolute loss, of sensibility. The symptoms came on gradually six years before dea (Lancereaux, "Atlas d'Anat. Path." p. 444.)

† The patient was a woman twenty-eight years of age. The first symptom was pain ra ating over the upper part of the abdomen on the right side, and afterward extending to leg and to the left side. After a few months, weakness of the legs gradually came on, became absolute, with loss of sensation and of power over the sphincters. Extensive b sores were the immediate cause of death, which occurred eighteen months after the on (Lancereaux, "Atlas," p. 447.)

‡ The patient was a man aged thirty-five. At twenty-eight, pain commenced in the ri forearm, and continued, intermitting for some years, until, at thirty-four, it extended thro the whole arm to the heck beside the spine, where it was increased by movement. Sim

The forms of extra-dural tumors are lipoma, from an overgrowth of the fat which normally exists between the membranes and the bone, and parasitic tumors, chiefly echinococci. Both are rare. Growths may also spring from the bones or intervertebral tissue, enchondroma, sarcoma, and cancerous tumors, but these have been already considered. Far more frequent are collections of inflammatory products from bone disease, but these do not come into the category of morbid growths.

The tumors which begin with the dura-matral sheath are chiefly syphilomata, sarcomata, and myxomata, sometimes containing cysts or "brain sand." Tubercular and parasitic tumors are rare, but both echinococci and cysticerci have been met with, developing in the meshes of the arachnoid. Fatty tumors have been found in a few instances. Fig. 155 represents a probably unique tumor, a myo-lipoma, consisting of fatty tissue and striated muscular fibres, which had grown from the pia mater or meshes of the arachnoid, and was no doubt congenital. Neuromata may occur on the nerve root, and may compress the cord.

The growths within the substance of the cord itself are more diverse in character. Syphiloma and glioma are the most common; sarcomata, myxomata, and tubercular tumors also occur. A cysticercus has once been met with. Some growths have a compound character, and have been termed myxo-sarcoma, glio-sarcoma, and fibro-sarcoma. Sarcomata and gliomata are sometimes very vascular, and such growths have been termed "angio-sarcoma" and "angio-glioma." It is far more common for the tumor to spring from the pia mater or from the peri-ependymal tissue around the canal, than for it to begin among the nerve structures.

Extra-dural growths are always single; those within the dura mater are often single, but sometimes two or three coexist, and neuromata or sarcomata on the nerve roots are often multiple. Tumors within the spinal cord are usually single, but occasionally more than one growth is found, especially when the tumors are tubercular.

The size attained by growths outside the cord is necessarily moderate in consequence of the limitation of the space in which they grow. They vary from the size of a pea to a width of one and a length of two inches, which is rarely exceeded, and chiefly by extra-dural tumors. Those that develop from

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pain then came on in the left arm. Then followed weakness in the right arm, slight spasm in the right leg, and tingling in the left. The arm became almost powerless and wasted, the right leg weak, and flexion of the neck caused severe local pain. These symptoms continued and increased, and pain in the left leg came on. The cervical spine became tender, and movement of the head to the right was limited. Then both legs became weak, and sensibility was lost as high as the nipples. The paraplegia became absolute, bedsores formed, and the patient died seven years after the onset of the symptoms. Softening of the spinal cord extended down into the dorsal region. (Leyden, *Klin. der Rückenmarkskr.*, Bd. i, p. 450.)

the pia mater are usually smaller than those that spring from the dura mater Multiple tumors on the nerve roots are generally small. Adhesions ofte form between the growth and the membrane from which it does not arise The tumor compresses the nerve roots and the spinal cord (Figs. 156, 159) The compression produced by tumors within the dura mater is alway greater than that by tumors outside this membrane. The amount of pressur is proportioned to the size of the tumor and its consistence. A soft growt outside the sheath may attain a considerable size, and even extend throug the intervertebral foramina, without exerting much pressure on the cord itsel At the compressed part the cord is narrowed and softened, usually indente or flattened, because the pressure is either from one side, from the front, from the back. Rarely the degree of pressure is so great that the cord narrowed to the size of a crow-quill, and it has even been apparently inte rupted at the spot, the upper and lower portions being conical and the pointed extremities connected only by membranous tissue. The softenir of the cord depends on inflammation, the "pressure myelitis" described a preceding chapter (p. 248), and it presents the tissue changes that ha been there enumerated. As a rule, the spinal cord is not invaded by growth that develops outside it, even if this begins in the pia mater.

FIG. 160.

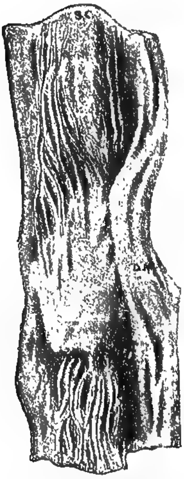

FIG. 161.

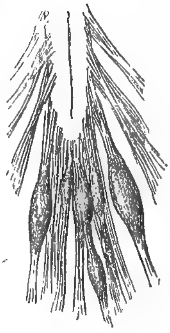

FIG. 160.—Tumor of the cauda equina surrounding and enclosing many of the nerve roots. The cord itself was unaffected. The structure of the tumor was that of a fibro-sarcoma.*

FIG. 161.—Neuromata of the cauda equina. (*After Lancereaux.*) The growths had apparently given rise to no symptoms.

The growths that involve the cau equina often attain a larger size th those that occur higher up, because t part of the vertebral canal is large, a the nerve roots occupy but a small p of it. Most tumors in this situation a sarcomata or fibro-sarcomata. Th usually spring from the tissue of t arachnoid and often surround a include the nerves (Fig. 160), t amount of damage to which is ve variable.

Multiple tumors outside the cord sometimes very numerous. They usually sarcomata and spring from membranes and sheaths of the nerv They are various in size, ranging fr that of a hazel nut to that of a pi head, and many very small growths often scattered among the nerves of cauda equina. In some cases of character similar growths have exis in the cerebral membranes.

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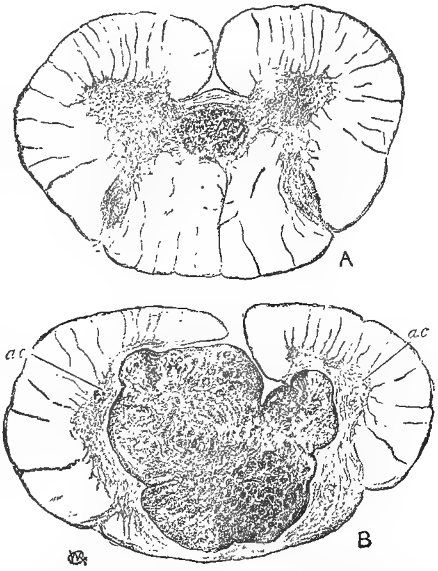
* The patient was a man aged twenty-eight, under the care of my colleague, Dr. Radcl in the Queen Square Hospital. The symptoms commenced only nine months before de and consisted in severe pains in the legs and progressive weakness, the power of stan

The tumors within the spinal cord are usually small, seldom exceeding half an inch in diameter, and usually less, even when they give rise to considerable impairment of function. But their vertical extent often exceeds their transverse diameter, and sometimes they grow through a considerable extent of the spinal cord. Gliomata and growths that spring from the central tissue are those which have most frequently a considerable vertical extent. A glioma has been known to reach from the medulla oblongata to the lumbar region. The cord is enlarged at the seat of the tumor, and the precise character of the enlargement depends on the position of the growth. In an infiltrating glioma the increase in size has been so great that the wall of the foramen magnum caused a constriction around the swollen cord. Often the abnormal color of the tumor is perceptible on the surface. The consistence of the cord may be lessened in soft growths, but the tumor is commonly firmer than the normal cord, and the difference is frequently increased by softening in the vicinity of the growth. If the tumor reaches the surface, the pia mater and arachnoid may be thickened at the spot, and they may even be adherent to the dura mater, but there is rarely any extension of the meningitis to other parts. On section, the growth is usually very distinct, since its aspect contrasts with that of the nerve substance. Only

FIG. 162.



Tumor of spinal cord, springing from the tissue around the central canal. A, upper part of the tumor in the middle of the cervical region; the growth is here within the posterior commissure. B, section through the largest part of the tumor, which occupies the whole central region of the cord. The vertical extent was 2 cm. It was a sarcoma composed chiefly of spindle cells arranged in fasciculi and in concentric layers. The patient was suffering also from a cerebral tumor causing hemiplegia, and the spinal growth, which apparently developed rapidly during the final period of palsy and prostration, was not suspected during life.

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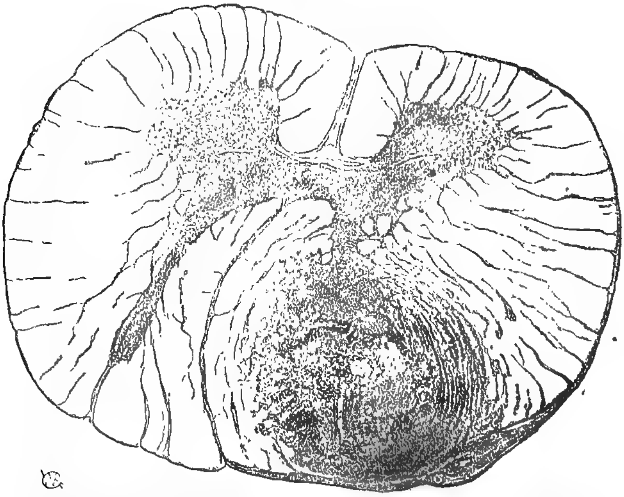
being lost about four months after the onset. A little power in the flexors of the hips and the extensors of the knees persisted almost to the last, but the knee jerk was lost. All the muscles of the legs wasted, those below the knee extremely, and even when the patient was first seen electrical irritability was greatly lowered to both currents, without any reaction of degeneration. Tactile sensibility was impaired in each foot and lower leg, more in the right than in the left, and chiefly in the region supplied from the sacral plexus. Sensibility to pain was not affected. The bladder was affected early, and, from imperfect evacuation, cystitis and symptoms of pyelo-nephritis already existed, a catheter having never been passed. The kidney disease continued, in spite of local treatment, and was the immediate cause of death, which was preceded by several convulsions, apparently uræmic. A microscopical examination showed considerable damage to many nerve roots involved in the tumor, while others had escaped. The muscles presented intense granular degeneration with some longitudinal striation, and increase of the interstitial nuclei.

glioma and myxoma may resemble the normal gray substance in appearanc but the new tissue is distinct in position. These tumors in some cases blei with the substance of the cord, but in other cases they are bounded by area of softening which often isolates even invading growths. Other tumc may be sharply limited. It may be difficult to say in what structure t tumor began, but this may sometimes be ascertained by an examination the upper and lower parts, when the growth is commonly limited to th part of the cord in which it first originated. Thus in Fig. 162, B, t tumor occupies a very large area, although the nearly equal extent on ea side suggests its central origin, and this is clearly seen in a section throu the upper part of the growth, in which the posterior commissure can

FIG. 163.



Syphiloma in the spinal cord, occupying the position of the posterior cornu and adjacent parts of the posterior and lateral columns, in the lower cervical region. The tumor presented the usual combination of gray translucent and yellow cheesy areas. A second similar, but much smaller, tumor existed higher up. The symptoms were complicated by hemiplegia, etc., of cerebral origin, due to a gumma in the brain. Paralysis with rigid flexor contracture of the left arm and leg were apparently due to the growth. The disease was certainly syphilitic, but it is noteworthy that it had developed during continuous treatment by full doses of iodide, which had entirely removed earlier symptoms, but to which the system had apparently become accustomed.

traced in front and behind the small area of growth, pointing to the pe ependymal tissue as its source. Syphilitic tumors commonly originate fro the pia mater, as they do also in the brain, and the membrane in the vicinity is often thickened. They both invade and compress the ner tissue. An instance of such a gumma of the cord is shown in Fig. 16 It probably commenced at the furrow opposite the head of the posteri cornu, which it has destroyed, and the pressure which it exerted is evide from the displacement of the posterior median septum. In all forms tumor the central canal is often obliterated by the pressure and may slightly dilated higher up, and this dilatation is especially common fro growths that begin in the peri-ependymal tissue. The considerable dilat tion of the canal which is termed "syringo-myelia" is associated with a peculiar growth in this position, probably congenital in origin; it is described in another chapter. In other forms of tumor cysts occasionally develop, and hemorrhage may occur into such cysts, or into the softened tissue in the vicinity of the tumor, or even in the substance of a soft growth itself, especially when this is a glioma. From the region of the growth, secondary degenerations can frequently be traced upward and downward, but these are often slighter than the apparent damage would suggest, probably by reason of the remarkable tolerance of the nerve fibres to pressure if this is slowly developed.

SYMPTOMS.—The symptoms produced by tumors within the spinal canal, whether these are within the cord or outside it, resemble in their general characters those which we have already considered as resulting from growth and caries in the bones of the vertebral column. The resemblance is most close in the case of extra-dural growths, and least so in that of tumors within the substance of the cord. The chief differences are the relative preponderance of symptoms of irritation of the cord itself, and the indications of a transverse extension of the damage from one side of the cord to the other.

In the majority of cases pain is a prominent symptom through the whole course of the disease. It is usually the earliest symptom, and is severe both before and after the development of other symptoms. The pain may be felt along the course of the nerves which arise at the level of the tumor and also in the parts supplied by nerves below that level, *e. g.*, in the legs. Both kinds of pain are frequently very severe, darting, stabbing, rending in character. Dull aching pain may be felt between the attacks of severe pain. The intensity of the suffering is usually so great that it has more than once led the unhappy sufferer to attempt suicide. The pains are commonly felt on one side first, in one arm, in one side of the trunk at a given level, or in one leg, and a considerable time may elapse before they extend to the other side. Occasionally the pain is bilateral from the first. The pains are sometimes increased by movement, but rarely in the intense degree common in tumors of the bone. Pain is sometimes felt in the spine; frequently when the growth begins in the dura mater, rarely when it is within the substance of the cord. Tenderness of the spine often exists in the former cases, but is on the whole a rare symptom. Other subjective sensations may occur with the pain or in the intervals; "numbness," tingling, formication, etc., and they give significance to the pains as an indication of organic damage to the nerve structures. The root pains in the trunk are often accompanied by a sense of constriction, which may be very distressing. Hyperæsthesia of the skin often accompanies the pains felt at the level of the tumor, less commonly those in the parts below.

Muscular spasm is another common symptom, most pronounced when the tumor springs from the membranes. There may be some rigidity of the back opposite the seat of the growth, usually associated with local pain. It is most marked when the disease is at the more mobile parts of the spine, especially when it is in the cervical region. In such cases the rigidity may be painful, and the pain may be increased by movement. Severe spasm in the abdominal muscles is often associated with severe girdle pain. Contractures often develop in the limbs, both in those supplied by the nerves which arise at the level of the tumor, and are directly irritated by it, and also, although less commonly, in the parts supplied from the spinal cord below the level of the growth. Thus the tumor in one-half of the cord in the cervical region (shown in Fig. 163) caused persistent flexor contracture in the arm and leg on the side of the growth.

Paralysis is almost invariably one of the symptoms produced by an intraspinal tumor. Its onset is usually gradual. Paraplegia is the most common form, but all four limbs may be paralyzed by a growth in the cervical region. Very commonly one leg becomes weakened before the other, and occasionally arm and leg suffer before the other side. Tumors which are situated in the middle line or in the centre of the cord, however, usually affect both sides at the same time. The palsy, gradual in onset, is usually also slow in its extension, the rate of its increase depending on the rate at which the tumor grows and compresses the spinal cord. This is the characteristic course of the paralysis produced by a tumor. But in a large number of cases the loss of power comes on in a subacute, or even an acute manner, in consequence of the myelitis excited by the compression, which, in such cases, may run a course of independent acuteness. When the inflammation is thus in excess of its cause, the palsy may lessen as damage from the inflammation subsides to the degree of the damage from pressure. The symptoms that depend on compression slowly but surely increase as the tumor grows, and usually attain a high degree of intensity, the loss of power in the legs, for instance, becoming absolute and remaining so.

Loss of sensation comes on sometimes with the loss of motor power, more often after this has become considerable. It may be partial at first, but tends to become complete as the damage to the cord increases. The loss of sensation corresponds in distribution to the motor palsy when the disease is below the middle of the dorsal region, but if it is situated above this, and on one side, the sensory loss is often on the side opposite to the motor paralysis. Tumors of the spinal cord furnish a considerable proportion of the cases in which the crossed symptoms of a unilateral lesion are well marked (see p. 166). In addition to the loss of sensation which results from the damage to the cord itself, areas of anæsthesia may exist in the region supplied by the nerves which are damaged by the growth, and in which the severe radiating pains are felt. If there is crossed sensory and motor palsy, these root symptoms are intense chiefly on the side opposite to the anæsthesia in the limbs. Incoördination of movement is not a common symptom of spinal tumors, but has been occasionally met with in cases of growth in the posterior column, of central tumor, and also as a result of multiple tumors of the nerve roots (see p. 418). Loss of power usually coexists and quickly becomes predominant.

The condition of reflex action depends on the position of the tumor. In the lumbar enlargement or the cauda equina, a growth abolishes reflex action in the legs, and in any position the trunk reflexes are lost at the level of the lesion. But when the disease is situated higher up, reflex action is increased and the excess of cutaneous reflex action is a very marked and early symptom of the disease. The myotatic irritability presents also the increase that invariably results from damage to the pyramidal tracts, and the usual tonic spasm also gradually develops and often reaches a high degree. Muscular contracture is added to it more frequently than in many other diseases, and rigid spasm in the flexors of the knee and hip may keep the heels in contact with the nates.

Wasting of the muscles is for the most part confined to those parts to which the damaged nerve roots go. In tumors of the lumbar enlargement and cauda equina, the atrophy of the muscles of the legs is a very conspicuous symptom. Vaso-motor disturbance sometimes occurs in the early stage of the disease, chiefly in the distribution of irritated roots, where flushing of the skin has been noted, and the so-called "meningitic streak (" *tache cérébrale* ") may often be produced. Occasionally vaso-motor œdema is a conspicuous early symptom. In the later stages, bedsores are very common and often severe. Dilatation or contraction of the pupil is common when a tumor is situated in the lower cervical region.

The course of the symptoms is variable, and depends on the rapidity of growth of the tumor, and on the share which secondary myelitis takes in the production of the symptoms. As a rule, the root symptoms occur first, and in growths outside the cord they may exist alone for months, and even, in the case of a slowly-growing tumor, for years before the symptoms of damage to the cord itself are added. On the other hand, they may be entirely absent in growths which commence within the cord itself. In some cases the symptoms are steadily progressive from first to last. In others the progress is intermittent; periods of increase in the symptoms alternate with stationary periods. Occasionally a rapid increase of the cord symptoms may be followed by positive improvement for a time, but scarcely ever by an actual disappearance of the manifestations of the disease. The most important element in the course of the disease is the tendency to a lateral extension of the symptoms, the expression of the transverse extension of the damage, as the several structures of the cord, at the same level, successively suffer. There is sometimes an extension downward of the interference with the central functions of the cord, when secondary myelitis descends to the lumbar enlargement, but there is scarcely ever an extension upward above the level of the initial interference with function. A small, slowly-growing tumor, even within the cord, sometimes causes no symptoms.

The chief differences between the symptoms of tumors of the cord and of the membranes are that, in the latter, the root symptoms are more severe in a larger proportion of the cases, and that the cord symptoms occur later, and are at first more limited in extent. Spinal pain, tenderness, and rigidity are more common. In cord tumors the radiating pains, due to irritation of the nerve roots, are more often trifling or absent, but if the growth begins in the neighborhood of the posterior horn they may be very pronounced.

The cord symptoms are often bilateral from the first, and in central tumors they may develop equally on the two sides. If they are unilateral at the onset they extend to the other side sooner than in the case of meningeal growths. Extensive muscular atrophy is more common in tumors of the cord than in those of the membranes (except in the case of tumors of the cauda equina), because central tumors may damage the gray matter through a considerable vertical extent, and are most common in the enlargements, and also because myelitis spreads in the gray matter far more frequently when the exciting growth is in the cord itself than when it is in the membranes.

In tumors of the cervical region the interference with movement of the neck, from the rigidity of the muscles, is often well marked, especially when the tumor is in the membranes. The pains due to irritation of the nerves are felt in the arms, and are often associated with muscular atrophy. The excess of reflex action in the legs is very great, and if the influence of the tumor is chiefly on one side, hemiplegic weakness, with crossed anæsthesia, is present.

Growths in the dorsal region of the cord usually cause severe radiating pains in the trunk, often accompanied by an intensely painful sense of constriction, hyperæsthesia, and sometimes by anæsthesia. The simple, nearly level course of the dorsal nerves reveals the precise position of the disease more clearly than do the symptoms in the arms when the tumor is in the cervical region. The interference with the trunk reflexes often corroborates the indications afforded by the subjective symptoms. Spinal tenderness may be also present, but rigidity of the spinal muscles is not common. Reflex action in the legs presents the same excess that results from tumors in the cervical region, but occasionally, when the growth is on the confines of the lumbar enlargement, a descending myelitis may abolish the reflex action, and remove spasm, if this previously existed, rendering the muscles flaccid.

The symptoms produced by tumors which involve the lumbar enlargement vary considerably according to the precise position of the growth. The characteristic pains are felt in the legs, and the muscles supplied from the part invaded or compressed undergo atrophy and present loss of reflex action and of myotatic irritability, but the anæsthesia is often limited in area. A growth in the upper part of the enlargement may cause wasting only in the flexors of the hip and extensors of the knee, with loss of the knee jerk and anæsthesia in the front of the thigh, while the muscles below the knee present simple palsy with myotatic excess. Thus a man, who had had syphilis, complained of pain in the left groin and thigh, and presented wasting of the extensors of the knee, with anæsthesia in the front of the thigh; there was loss of the left knee jerk, and a foot clonus on each side. He recovered, but there was doubtless a small syphiloma at the upper part of the lumbar enlargement on the left side, interrupting the reflex arc, damaging the motor and sensory nerves, and compressing both pyramidal tracts. A growth in the membranes adjacent to the middle or lower part of the lumbar enlargement usually causes symptoms of wide distribution, affecting the whole of both legs, and the early symptoms of irritation often involve the distribution of the upper lumbar nerves. But a growth at the same level within the cord may affect chiefly the muscles of the lower leg, and cause anæsthesia in the region supplied from the sacral plexus. There may also be palsy and wasting of all the muscles of both legs from an extensive secondary myelitis, but even then the sensory loss is usually limited. In lumbar tumors there is early palsy of the bladder and sphincters. Examination of the sphincter ani shows absolute paralysis, and not the pure reflex action present when the disease, arresting voluntary influences, is higher up the cord. The tendency to bedsores is commonly strong in tumors of this part.

Growths situated in the cauda equina, which are below and do not affect the cord itself, produce symptoms very similar to those due to tumors of the lumbar enlargement, but the symptoms are bilateral from the first in the majority of cases. Rarely one leg is affected alone at the outset. The symptoms usually begin in the lowest parts of the legs, which are throughout affected in greater degree than the upper parts, and the flexors of the hip may escape altogether. Muscular contraction is much less common than in tumors situated higher up. The anæsthesia is chiefly in the region supplied by the sacral plexus, and the pains, usually very severe, are first felt in the same region, and there may be severe pain on each side of the sacrum radiating to the region of the sciatic nerves. The muscles below the knee rapidly waste, and often (but not always) present the degenerative reaction. These symptoms were well marked and characteristic in the case figured and described at p. 410 (Fig. 160), and the condition of the muscles suggests that the absence of any voltaic irritability in the muscular fibres was due to their early and intense fatty degeneration. In many cases the symptoms are irregular in distribution; the nerves are included in the growth, and are often damaged unequally, and less than might be expected from the size of the tumor, which may be considerable before its expansion is arrested by contact with the bony walls of the canal. Hence the loss of sensation may be partial, and reflex action may not be completely abolished. In the case above mentioned, for instance, a prick on the sole caused a reflex contraction of the muscles in the upper parts of the legs, but of these alone. In some cases, however, the loss of sensation has been absolute and reflex action also has been entirely lost. The tendency to trophic changes is great, and an affection of the sphincters is almost invariable. Cystitis and its consequences readily occur and may reach a high degree, in consequence of residual accumulation, when the patient is conscious only of slight difficulty of micturition.

Multiple tumors may give rise to very complex symptoms. If there are only two tumors they may be manifested by the successive development of focal symptoms, similar in character but different in seat. In many cases in which there are many growths only one or two of the largest have caused symptoms, and the smaller tumors have run a latent course. In some other cases in which many tumors have caused slight symptoms, these have simulated closely those of a system disease of the cord. Thus, in the remarkable case recorded by Hughes Bennett, multiple growths on the posterior nerve roots produced the characteristic manifestations of locomotor ataxy.* When, as has often been the case, cerebral tumors coexist with multiple spinal growths, the symptoms of the latter have usually been lost in those of the intra-cranial disease which has preceded the spinal lesion.

The duration of the symptoms of tumors of the cord and membranes, those of syphilitic nature being excluded, varies from three months to five, or even ten, years. In the majority of cases death ensues at the end of from one to three years from the commencement of the symptoms.

Diagnosis.—Although no symptom produced by tumors within the vertebral canal is actually pathognomonic, and the disease is, of course, not to be directly recognized, yet the combination of symptoms, and the course which they present, are in most cases sufficiently characteristic to render the diagnosis a matter of no great difficulty. The symptoms of greatest diagnostic importance are, first, the pain, severe in degree and constant in seat, and, secondly, the progressive paralysis. Of less but still considerable importance are the rigidity of the muscles of the spine, the muscular contractions in the limbs, and the early and marked excess of reflex action when the cord itself is involved. In the course of the disease, the most important features are the steady progress of the symptoms, and especially their commencement on one side and extension to the other. Causal indications occasionally corroborate the diagnosis, and sometimes give additional weight to suggestive symptoms. Of such indications the most important are a history of constitutional syphilis, the presence of tubercular disease elsewhere, and, in rare instances, the existence, in other parts, of such multiple growths as are known to occur also within the spinal canal, especially multiple sarcomata and neuromata.

The diagnosis of multiple growths depends on the recognition of the successive development of the characteristic symptoms in more than one focus. It is rare for more than two of many tumors to cause definite symptoms. The greatest absolute difficulty in the diagnosis of a spinal tumor is presented by the cases, by no means rare, in which an intra-cranial tumor precedes the growth within the vertebral column, and the symptoms of the latter are overlooked in the presence of the severe manifestations of the former. In most cases, however, the disease can be recognized if the additional symptoms, which usually exist, are detected by careful observation, and their significance is recognized.

The differential diagnosis involves the distinction from diseases of the vertebral bones, of the membranes, and of the cord itself. Caries of the spine has many symptoms in common with tumor, but the root pains are rarely very severe, and the effects of compression of the cord are usually bilateral, either from the first or very soon after their commencement. The paralysis seldom reaches a considerable degree in one leg before the other suffers. The signs of bone disease are rarely long absent, and repeated examination usually reveals at least irregularity of the vertebral spines. From growths in the bones of the spinal column the diagnosis may at first be impossible. But the pain of tumor is rarely increased by movement to the same intense degree as when the growth commences in the bone, unless the tumor is in the cervical region of the cord, and then the greater accessibility of the vertebral column renders the exclusion of bone growths comparatively easy by local examination. In the dorsal region a growth in the bone may long escape detection, but even here the influence of movement on the pain is often extreme, far greater than in the case of tumors within the canal. The effect of movement of the vertebræ on the nerve roots, when compressed as they pass through the foramina, is far greater than when they are compressed within the canal. Sooner or later the local enlargement manifests itself externally and shows the nature of the case.

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* *Clinical Transactions, 1885.*

"Hypertrophic pachymeningitis," affecting the cervical region, is the only meningeal disease which closely simulates the symptoms of tumor, but its effects are usually from the first bilateral, and they have a considerable vertical extent. Hence it is only with central tumors, which grow through several inches or more of the cord, that the disease is likely to be confounded. In each malady there may be muscular atrophy in the arms, and paralysis, without wasting, in the legs. But in central tumors the sensory symptoms in the arms are usually much slighter than in the thickening of the membrane; there is rarely the early and limited anæsthesia which results from the damage to the nerve roots in the latter disease.

The affection of the cord itself which most closely simulates the symptoms of tumor is chronic transverse myelitis. In such a case as that shown in Fig. 96, p. 242, the diagnosis may be a matter of extreme difficulty. The radiating pains in this case were severe and local, and the limitation of the symptoms, for a long time, to one side, with crossed sensory and motor palsy, presented an almost perfect analogy to the effects of a growth. But it is very rare for severe and acute radiating pains to result from focal myelitis, although a painful sense of constriction is common; moreover, in tumor, one-half of the cord seldom suffers severely without the functions of the other half also soon being impaired in some degree. The limitation of chronic myelitis to a single focus is also very unusual; a considerable vertical extent of the cord is usually involved in varying degree. Hence the practical difficulty in diagnosis is not so great as at first sight might appear. Acute or subacute myelitis can only be confounded with a growth in cases in which inflammation results from pressure, and develops with independent energy and rapidity. In such cases the occurrence of myelitis must be recognized, and the diagnostic problem is the detection of the preceding symptoms of the growth that excites the inflammation. This is usually easy; the severe pains, and commonly, also, the slighter symptoms of paralysis, which existed before the rapid increase in the loss of power, are sufficient proof of a chronic morbid process, to which the inflammation is secondary.

The early pains are often thought to be neuralgic, but their constancy and continuance should suggest organic irritation; their seat, moreover, is rarely that in which neuralgia is common. The pain produced by tumors of the cauda equina is often felt first in the sciatic area, and thought to be sciatica, but it is commonly from the first bilateral, while sciatica is almost invariably one-sided, and bilateral sciatic pain should always suggest irritation where the sensory fibres from both sides are near together, and can be irritated by a single cause, *i. e.*, it should suggest disease within the vertebral canal. In all cases, however dubious the early pains may be, other symptoms soon become added, and prove that there is more than a simple neuralgia.

If the existence of a tumor is clear, the question remains, What is its nature? If the patient has had constitutional syphilis, the syphilitic nature of the growth is highly probable, and the probability is further raised in degree if the growth developed rapidly, so that the symptoms attained a considerable intensity in less than three months from their onset. The coexistence of a cerebral lesion also increases the likelihood of the syphilitic nature of the growth. Either of the last two considerations suggests the same conclusion in any case in which syphilis, although not proved, is possible, but it must be remembered that cerebral and spinal tumors, of other than syphilitic nature, may coexist. In the same way, tubercular and scrofulous processes suggest the tubercular nature of a tumor, and this is further supported by indications of the existence of more than one tumor in the spinal cord, and by long periods of arrest of the progress of symptoms that were slow in their development. Such arrest indicates an arrest of the growth of the tumor, which we cannot infer from the subsidence of symptoms that developed rapidly, and may have been due to secondary inflammation. Tumors elsewhere always constitute strong evidence of the nature of one in the cord, but such an indication is rarely available, since the spinal canal is rarely the seat of secondary growths. Multiple tumors are probably either sarcoma or neuroma. In the absence of these indications a meningeal tumor is probably a sarcoma, and a tumor within the cord is probably a glioma, because these are the most frequent kinds of growths which occur in those situations. A diagnosis founded on mere frequency of occurrence has necessarily only a low degree of probability, and must be sometimes wrong.

PROGNOSIS.—Unless the tumor is syphilitic, the prognosis is necessarily most grave. Of all syphilitic lesions, growths are those that are most amenable to treatment, and the effects of which most certainly pass away, if they have not reached an extreme degree or lasted too long a time. If, however, the damage has been allowed to remain unchecked for several months, recovery may be imperfect. Tumors of other kinds steadily increase, and the resulting damage is on the whole steadily progressive, although its course may be varied by stages of rapid (myelitic) increase in the symptoms, followed by stationary periods and even by transient improvement.

TREATMENT.—If the growth is syphilitic, iodide of potassium should be given in full doses of 30–90 grains a day, and unless the symptoms rapidly lessen, mercury may also be administered. It is important that the treatment should be energetic and prompt, so as to effect some diminution of the pressure as quickly as possible, since the longer this continues, the more considerable are the degenerative changes. Even a difference of a few days in the influence of treatment, and consequent duration of pressure, may make a difference of weeks in the duration of symptoms and have a distinct influence on the degree of ultimate recovery. If the symptoms suggest a tumor, and syphilis is possible, appropriate treatment should be adopted for a time, since it can do no harm if the tumor is not syphilitic, and may save the patient's life and strength if it is. It should be remembered, moreover, that syphilis can only be excluded when there has been no *possibility* of infection. In a certain proportion of the cases of late syphilitic lesions, in the nervous system and elsewhere, there is no history of secondary syphilis, and in another proportion of cases of secondary syphilis there is no history of a primary sore. Hence it is *à priori* certain that in some cases of late lesions a history of both primary sore and secondary symptoms will be absent, and, as a matter of fact, such cases are met with not unfrequently. Of course, these cases cannot be counted as syphilitic in any scientific investigation into the influence of this disorder, but we have often to allow weight, in treatment, to considerations which are insufficient to influence scientific investigations, and treatment determined by the mere possibility of syphilis is often justified by its results.

In other than syphilitic cases we can do little more than treat symptoms, relieving pain by sedatives, watching the state of the bladder, preventing the occurrence of cystitis and guarding the patient from bedsores. We must remember that sedatives will probably be needed for a long time, and they must therefore be used as sparingly as possible, lest custom and tolerance deprive them of their power. Cocaine may be used in many cases as an aid in economizing the influence of morphia. It is highly probable that surgery may ultimately be able to cope, in some degree, with meningeal tumors. Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumor from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumor from the brain.* On the other hand, growths within the spinal cord are altogether outside the possible range of surgical procedure, since the removal of such a tumor would entail an injury equal to the greatest damage the growth could ultimately inflict, and equally disastrous in its consequences.

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* Since the above was written, an opportunity has presented itself to me of acting on the opinion here expressed. I was consulted in the case of a middle-aged man suffering from characteristic symptoms of compression of the spinal cord by a morbid growth, pains around the thorax and in the legs, with complete motor and sensory paraplegia, the anæsthesia reaching to the level of the radiating pains. I urged that an attempt should be made to remove the tumor, believing that, if this should prove impracticable, the removal of the arches would lessen the pressure on the cord. The operation was performed by Mr. Victor Horsley. A small tumor, the size of an almond, was found within the dura mater; the cord was greatly compressed. The whole tumor was easily removed. It was higher than the symptoms suggested, four inches above the level of the pains and anæsthesia. In spite of the size

CAVITIES AND FISSURES IN THE SPINAL CORD.

SYRINGOMYELIA, HYDROMYELIA, HYDRORRHACHIS INTERNA.

Cavities in the spinal cord, distended with liquid, are met with at all ages. They are found sometimes in young children, a condition termed "hydromyelia." In such cases they are clearly due to an arrest of development, which often affects the brain also. The cavities met with in adults have been distinguished from those found in young children, and have been termed "syringomyelia." This condition has been supposed to be acquired, the result of morbid processes commencing in adult life, but the two conditions present so many points of resemblance that they are probably of the same nature, and the adult cases, as well as the infantile forms, are congenital in origin, although they are no doubt modified by secondary changes occurring during their longer course.* The following figures illustrate all the most important facts regarding this morbid state, and the somewhat complex conditions will be best understood by considering the appearances presented in the several figures, and their significance.

The great fact of all cavities within the cord is their association with the presence of abnormal tissue, resembling closely that which commonly surrounds the central canal of the cord, and that which exists as a thin layer on the surface of the cord, between the pia mater and the nerve tissue. The two are probably of the same nature, but that around the canal is modified by the growth in it of small cells or nuclei. It is probable that this abnormal tissue, accompanying the cavities, is due to a persistence of the embryonal tissue from which the cord is developed, and that the presence of the cavities is due partly to defective closure of the tube which is first formed (residual portions of which become distended by accumulation of liquid), and partly to the breaking down of this persistent embryonal tissue or to tissue formed from it by a process of growth. The cord sometimes suffers serious damage from pressure, owing to the distention of the cavity by the liquid which accumulates within it.

To understand the origin of these conditions it is necessary to remember the mode of development of the spinal cord. The sides of the primitive furrow of the embryo coalesce so as to form a canal of relatively large size, the walls of which are thinner in front and behind than at the sides, and

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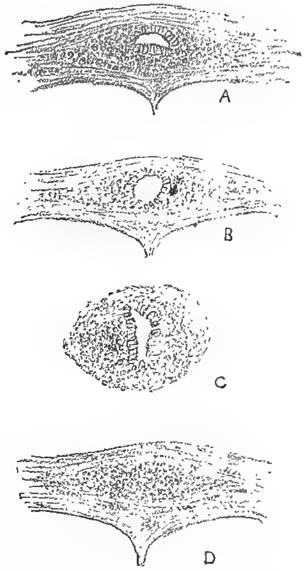
of the wound and the length of the opening in the dura mater, the patient had no bad symptom, and now, six weeks after the operation, the wound is healed, sensation is normal, pain is trifling, and motor power has already returned on the side opposite to the tumor.—*Author's note to American Edition, July, 1887.*

* See Leyden, *Virchow's Archiv*, Bd. 68, p. 1.

consist of elongated cells, some of which are arranged regularly at the inner surface of the cavity, like an epithelium. The anterior wall first becomes thickened, to form the white commissure and the front part of the gray, while an abundant growth of cells occurs at the sides of the tube, and extends forward (*i. e.*, downward in the position of the embryo), thus forming the lateral and anterior columns; between the two projections thus formed is the depression of the anterior fissure. Next occurs a growth backward of the now thick side, which forms the posterior parts of the lateral columns and the adjacent parts of the posterior columns, and with this the posterior roots are connected; this forms the "root zone" of the posterior columns. The posterior median columns are formed later. The central canal extends backward between these two rudimentary postero-external columns, being closed behind only by a thin layer of cells. As the posterior columns increase in thickness the posterior part of the canal becomes narrowed, and its walls unite near its anterior extremity so as to form the posterior gray commissure, and divide the canal into two parts. Of these the anterior becomes the permanent canal, while the posterior is reduced to a narrow fissure between the posterior columns. Ultimately this posterior narrow part becomes closed by a growth of cells which occurs from behind forward, as the median parts of the posterior columns develop. All these parts consist first of embryonal cells, which afterward undergo a transformation into the nerve elements proper, and the order of transformation is the same as that in which the parts were formed, the posterior median columns latest. The cells of the gray matter are formed before the fibres of the white columns. Some of the embryonal elements undergo a different and slighter change. They may also be said to persist, constituting the neuroglia and the gelatinous gray substance continuous with it around the cord, at the posterior root fissure, and around the central canal.

FIG. 164.



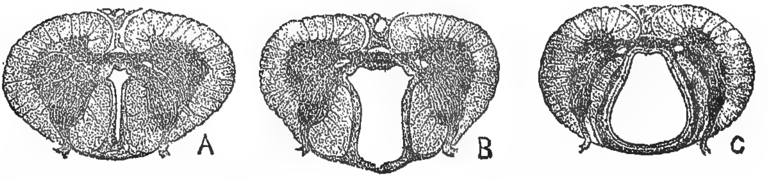
Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

The central canal ultimately lies in the anterior part or middle of the gray commissure. It may persist through life as a cavity, circular in section, or as a slit, antero-posterior or transverse, and is lined with epithelium (Fig. 164). Around the canal there are usually many nuclear elements, especially on each side of it, and these frequently fill up the lumen of the canal, so that its position is indicated only by an oval mass of small cellular elements (Fig.

164). The obliteration of the canal often occurs quite early in life, and is evidence of any morbid process. The canal may be found obliterated in o part of the cord, and patent in another (C, D, Fig. 164). The simp closure of the canal by nuclei does not cause any distention of the pa which is above the closed portion, or, at any rate, not more than a ve slight increase in size, so trifling that its significance is open to question.

In the congenital condition found in young children the morbid chan presents two distinct features. There is usually a quantity of tissue translucent aspect, which, from its structure, is clearly persistent embryon tissue in which nerve elements have not developed, and there is a cavi which is usully clearly due to imperfect closure of the canal. The chan is always confined to the posterior half of the cord, and usually to t posterior columns, which, as we have seen, are formed last. The persiste embryonal tissue is sometimes increased by a process of active growth, a the persistent canal or cavity is often enlarged by a process of distentio Further, when the persistent tissue is considerable in quantity it may bre down, and thus give rise to a new cavity, or enlarge that which has dev oped from the primitive canal.

FIG. 165.

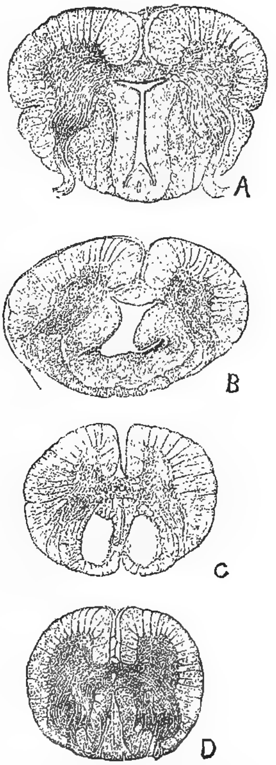


Hydromyelia, from a child two years old, with encephalocele and absence of cerebellum. (*After Leyde* In A, cervical region, the central canal is large, lined with epithelium, and from it a medial fissure exte backward nearly to the periphery of the cord, limited by a layer of homogeneous tissue, more abundan the posterior limit. In B, lower down, the fissure is enlarged to a cavity of considerable size, while in C i still larger, and the posterior columns are reduced to a narrow zone between the layers of tissue wh bounds the cavity and the gray substance.

These conditions are shown very well in the adjoining figures, after Le den (Figs. 165, 166). In the former, at A, the cavity presents nearly t shape which it has at one period of development, before the formation the posterior columns is completed, and without the separation into t parts produced by the formation of the posterior commissure. It is bound by a narrow layer of embryonal tissue, which widens out behind, havi nearly the shape of the posterior median columns, which are, as we ha seen, the last to be formed. In B and C the cavity is much larger, and t posterior columns are smaller, even by an earlier arrest of development by greater distention, or, most probably, by the influence of both the agencies. The cavity extends up to the posterior surface of the cord, a is closed in there only by a narrow layer of cellular structure.

In Fig. 166 we have somewhat more complex conditions. The cavity the first section closely resembles in appearance that in A of the last figure, b differs in the important fact that the posterior commissure has apparently be formed and the central canal developed in the normal manner, although it has become obliterated by nuclei. This is clear if the commissure is compared in the several figures. The cavity therefore does not, as in Fig. 165, represent the whole of the primitive canal, but only the posterior portion, after its division. The adjacent tissue is much more abundant than in the other case. The projection into the posterior extremity of the cavity indicates a progress of active growth, which is also shown by the irregularity of the cavity in B, the large amount of tissue, and the wide separation of the posterior horns. In C, from the lower part of the cord, the embryonal tissue occupies two oval areas in the posterior column (clear in the figure), while the cavity is closed or nearly so. (A little lower down a cavity existed in the centre of these areas, apparently due to breaking down of the tissue.) In D, again, the embryonal tissue occupies only the middle line, as a wedge-shaped area, due to the defective formation of the medial part of the posterior columns. It has been mentioned that the neuroglia may be regarded as a persistent, slightly modified, embryonal tissue, and these exuberant masses of tissue bear considerable resemblance in structure to glioma, so that the condition has been called "gliomatosis." In each of these cases the brain participated in the defect of development; there was internal hydrocephalus, and the cerebellum was absent.

FIG. 166.



Hydromyelia, from a child aged two and a half years, with internal hydrocephalus and absence of cerebellum. (*After Leyden, Virchow's Arch.*, Bd. 68.) A, B, cervical region; gelatinous (embryonal) tissue in the posterior columns encloses a medial cavity, lined in places with cylindrical epithelium, and believed therefore to be the dilated central canal. C, lower dorsal; in each posterior column is a mass of similar gelatinous substance (which, by a mistake, is shown in C as an open cavity). In D, lumbar region, this tissue occupies only the position of the posterior median columns.

The cavities in the adult cord present many varieties. The most common form resembles in its essential features the congenital disease which we have just considered. Between the two there are also differences, but the resemblance is sufficiently close, and the origin of the differences is sufficiently clear, to make it practically certain, as already mentioned, that the two forms are really the same, and the condition met with in adults is the congenital condition persisting until later life. Of the characters which this form has in common with the congenital disease, the most important are the position of the cavities, and the presence of tissue in their vicinity of embryonal or gliomatous nature. The differences depend on an apparent increase of this tissue by a process of growth, on the greater damage to the other parts of the cord by the distention of the cavity, and on the extent to which the formation of new cavities or enlargement of old ones takes place by a process of disintegration of the newly-formed or persistent tissue.

In the first place, as the simplest condition, we may have a dilatation of the central canal surrounded by gliomatous tissue disposed as in congenital cases. This tissue is often more abundant, and forms a more distinct mass, lower down the cord, below the cavity. A good example of this condition in slight degree is presented by the cord, the seat of chronic myelitis, represented at Fig. 97, p. 243. In the cervical region (A) there is a large central canal, bounded by a thick layer of gliomatous tissue. In the inner layer of this tissue, and bounding the cavity, is a sinuous membrane, fibrous in structure. The origin of this membrane is not easy to explain, but it is often found in cavities of this character, and is evidence of the common nature of those that differ much in other respects. In the lumbar region (B), the tissue, which bounds the cavity in the other section, forms a large round mass in the position of the canal, obliterating it.

In other cases a central cavity appears not to be the canal itself, but to be situated behind it, as we have seen may be the case in infants. It is then apparently due to a persistence of the fore part of the posterior portion of the canal, after the posterior commissure has been formed. In Fig. 167, for instance, we have a central cavity, but in the commissure in front of this is a mass of nuclei having the usual aspect of an obliterated canal.* The cavity is surrounded by a zone of tissue, which in A and B sends off a fringe of processes, especially backward. This tissue increases in the lower part of the cervical region (C). In B a small cavity is formed between the tissue and the gray matter, by breaking down, and in C a small cavity exists in the middle line behind the growth, probably due to deficient closure of the original fissure, and increased by a destructive process. Still lower the commissure resumes its normal appearance, but the collection of nuclei in the position of the canal is usually large (D). In the lumbar region the canal is patent (E). The degenerations of the posterior median column and of the lateral columns were secondary.

We have seen that even in the congenital cases we must recognize a process of growth of the persistent embryonal tissue to account for some of the conditions met with, and a like process is suggested by the central mass in Fig. 97, B. Hence it is not surprising that in many cases the growth should attain the dimensions, and assume the characters, of a positive tumor. In most cases the tumor has been central in position, and has

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* This is the probable interpretation. At the same time it is possible that the nuclei have not this significance, and that it is really the central canal. It is often more difficult than might be imagined to say whether a cavity does or does not represent the central canal. The presence or absence of an epithelial lining has generally been taken as a criterion, but it is doubtful whether this has any significance. Around the whole of the original cavity the inner layer of the cells is arranged as an epithelium, and if any part of it persists it is probable that epithelium will persist also. Moreover, the epithelium often disappears from the dilated central canal itself; always where it is enlarged by breaking down of tissue.

FIG. 167.

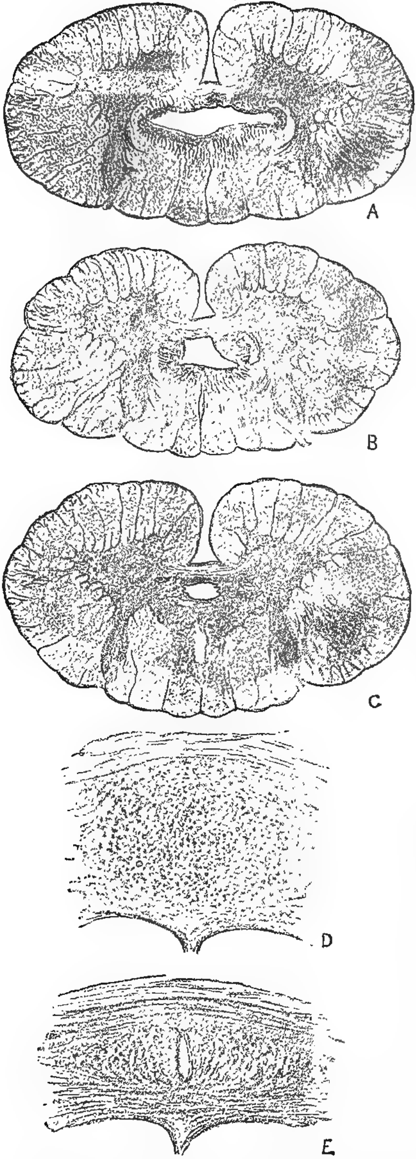

FIG. 168.

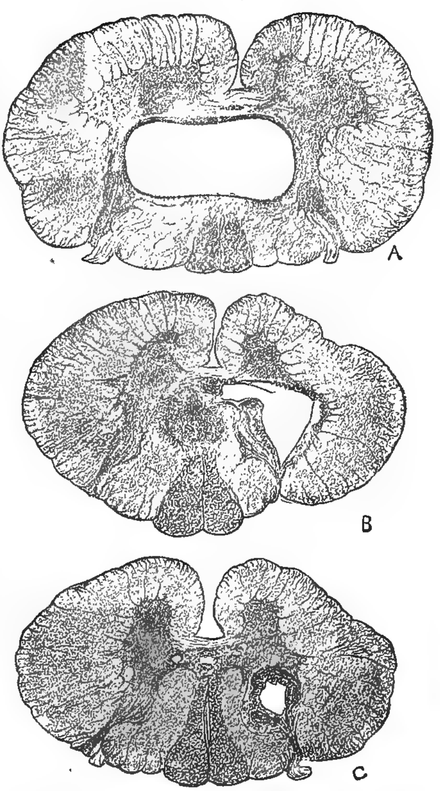

FIG. 167.—Syringomyelia, from a case of tumors of the pons and cauda equina, with some diffuse sclerosis in the dorsal region of the cord. A, B and C are sections of the cervical enlargement. The large cavity in A is perhaps the dilated canal, since it is lined in front with epithelium, but in front of it, in the posterior commissure, is a group of nuclei like an obliterated canal. The zone of tissue around the cyst is composed of interlacing fibres and nuclei, and processes extend from it into the posterior columns, both in front and behind. In B the cavity is smaller and the wall thicker, while in C the tissue about it forms a considerable mass, and a second small cavity has formed in the middle line, apparently by the breaking down of the tissue. In the dorsal region there was merely an unusually large accumulation of nuclei in the position of the canal, which, in the lumbar region, had the normal aspect (E). The cord presents degeneration of the right pyramidal tract (descending from the tumor in the pons), and of the post.-med. cols. and ant. lat. ascending tract, probably ascending from the sclerosis.

FIG. 168.—Syringomyelia, from a case in which there were tumors in the dorsal region of the cord, the cauda equina, and the pons Varolii A, mid-cervical region. A large oval cavity lies behind the gray commissure, surrounded by a narrow wall, chiefly composed of fine fibres, but with a wavy membrane on the inner surface. In the front of the posterior commissure an oval group of nuclei has the aspect of an obliterated central canal. A few short processes extend from the wall of the cavity into the posterior columns. There is secondary ascending degeneration of the post.-med. cols., right direct pyramidal tract, and ascending antero-lateral tract in all the sections. B, first dorsal. Behind the posterior commissure is a growth consisting of small cells, round and fusiform, the latter with round nuclei. It contains many vessels with thickened walls. A fringe of fine processes extends from it into the posterior columns. An irregular cavity occupies, on the right side, the posterior half of the gray matter, which is reduced to a narrow layer around the cyst. This is lined by a delicate nucleated membrane, outside which is a thicker membrane, lying in folds, the section of which is thus sinuous. Outside this again is a small-celled growth similar to that behind the commissure. In C, a little lower, this cavity is smaller, the sinuosities of the wall greater, and the growth outside it more abundant. (Still lower the area was entirely occupied by the growth.) In the posterior commissure there is only a quantity of loose nucleated tissue in the situation of the growth.*

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* For the opportunity of drawing these sections, I am indebted to Dr. Dreschfeld. A full account of the case has been published by Dr. Harris in *Brain*, Jan., 1886. The large growths were sarcomata.

had the structure of a glioma. It has occupied a large part of the area of the cord at a certain part.* In the chapter on the tumors of the cord it has been pointed out that sarcomata also may grow from the tissue around the central canal, as in the case shown in Fig. 162. Hence it is not surprising that the condition of syringomyelia, even if this is congenital in origin, should be frequently associated with definite tumors. The growths have often been multiple, apparently the result of a widespread tendency, and, perhaps, connected with a widespread persistence of embryonal tissue, of which we shall presently consider a remarkable instance. In the case shown in Fig. 167 there was a tumor of the pons and also one of the cauda equina. The nature of the growths is uncertain, but they were probably either sarcoma or glioma. The same tendency to morbid growths is illustrated also by Fig. 168, which is similar in many respects to that just considered. In this case, also, there was a tumor of the pons and one of the cauda equina, and there was also a central growth in the dorsal region, occupying the greater part of the area of the cord, to which the ascending degenerations are secondary. In A there is a large central cavity which is probably not the central canal, since, as in the last figure, the position of this is marked by the oval group of nuclei in the anterior part of the gray commissure. The zone of tissue which bounds it is narrow, but a few processes are given off behind. In B, first dorsal, this tissue forms a compact mass, and the processes are numerous and very similar to those in the last figure. In C, the commissure is reduced almost to normal conditions. In this cord we have also an instance of the fact that cavities may occur in other parts, very similar in their general characters to those that occur in the central region. In B, a large cavity occupies the intermediate gray matter and posterior cornu on the right side, with some morbid tissue bounding it on the medial side. In C, the cavity is smaller and the tissue around it is more abundant, but easily distinguishable from the gray substance, while a little lower down the cavity disappeared and the tissue formed a compact rounded mass. The cavity is lined by a delicate layer of cellular membrane; outside this is a fibrous membrane, lying in folds which appear as sinuosities in the section. The existence of this membrane shows that the cavity is not simply formed by the breaking down of the tissue. The tissue is quite similar in structure to that which lies behind the commissure in B.†

The posterior cornu is not an uncommon seat of cavities, which may extend through the whole length of the horn. An example of this is shown in Fig. 169. Hemorrhage had occurred into the cervical region of the cord (A), which was enlarged and distended with blood, so that the precise character of the cavity in this region was indistinguishable; the blood also filled the cavity through the entire length of the cord. In the upper dorsal region (B) there are two cavities, one in each posterior cornu, but that on the right ceased a little lower down, while the other extended to the lumbar region, as shown in the figure, reaching almost to the surface of the cord.. The upper and inner extremity of this cavity is in the position of the central canal, no other trace of which exists. There is some abnormal tissue in the neighborhood of the commissure, and also in C, irregular tracts pass transversely from the median septum in the fore part of the posterior columns. The presence of this tissue, and the relation to the central canal, suggests a congenital origin. During the reduction in size of the canal, in the development of the cord, the cavity presents at one period short lateral processes (compare Fig. 171, A), and we can therefore understand how a still earlier arrest of development may lead to the persistence of a lateral process on one side or both, surrounded by persistent embryonal tissue, and that the cavity should afterward extend in this tissue or in the gray matter by a process of disintegration compare Fig. 171, D. A comparison with this figure (E) suggests that the two cavities were united in the middle line on the upper part of the cord, in which the hemorrhage took place.

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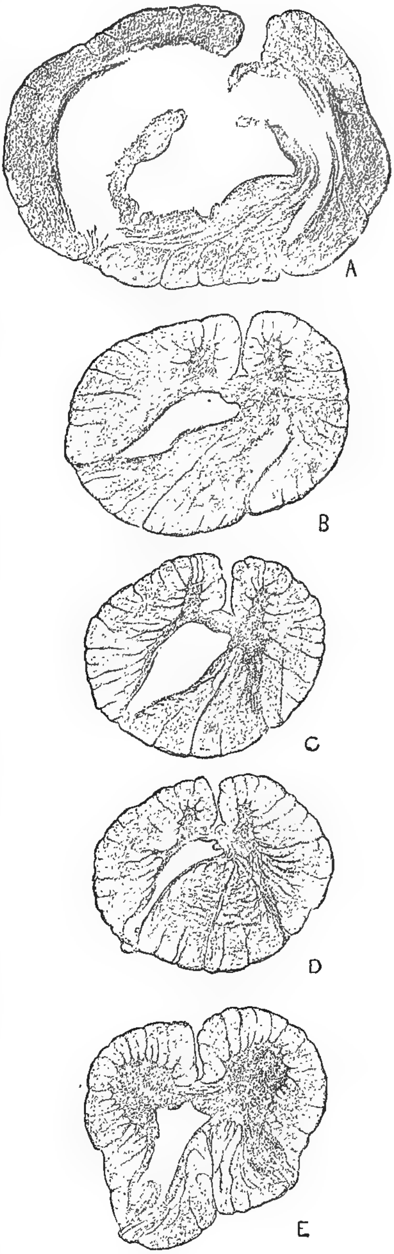
* As in an interesting case described and figured by Riesinger, *Virchow's Archiv*, Bd. 98.

† In connection with the tumor of the cauda equina in these two cases, it may be mentioned that the same coincidence has been observed in other cases. There was a tumor in that situation, for instance, in the case described by Riesinger, who, rightly regarding the condition as congenital in origin, suggests that the tumor may be connected with the fact that the embryonal cord occupied the entire length of the canal.

In the conditions we have hitherto considered, definite limited regions of the cord have been involved through a considerable vertical extent. The

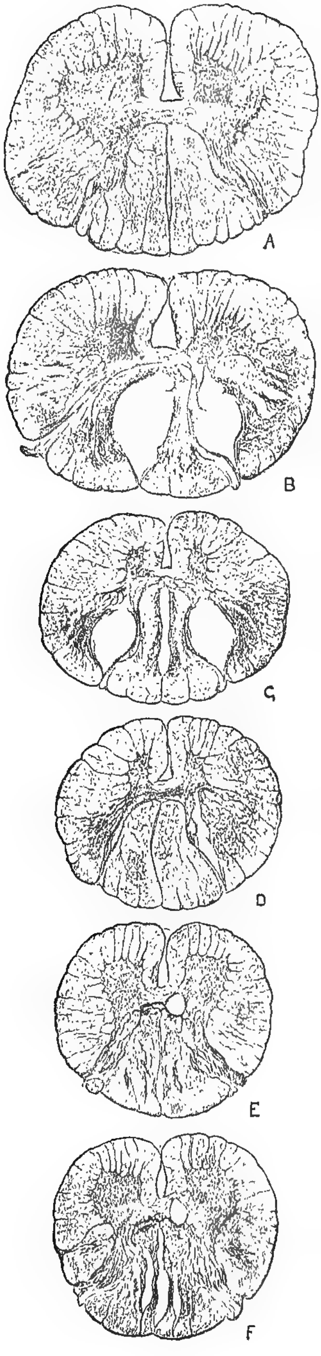
FIG. 169.



Sections of a spinal cord in which a cavity existed throughout its length. Hemorrhage in the cervical region was the immediate cause of death. The extravasation had there distended the cord (A), and had burst into the cavity, which was filled with blood. A smaller cavity existed in the right cornu, in the upper dorsal region (B). In most parts the inner surface was slightly irregular, as if produced by breaking down of the tissue, but near the position of the central canal a sinuous membrane existed in places, without, however any epithelial covering.

changes are sometimes, however, much more diffuse in their distribution, as is shown by the remarkable lesions represented in Fig. 170. In this cord, which is that of an adult, we have a combination of the same two conditions of abnormal tissue and cavities. Scattered through the nerve substance are tracts of a peculiar tissue represented by the darker shading of the figure, and in many regions thus affected there are cavities, for the most part fissure like, as if the cord had been split here and there. At first sight the appearance suggests an active and recent morbid process; nevertheless, I think that the congenital origin of the condition is beyond doubt. It will be observed that the morbid changes, like the other forms of syringomyelia, are chiefly in the posterior half of the cord; small areas of disease in the anterior columns, in the sections E and F, are the only exceptions. The minute structure of the abnormal tissue is exactly like that of the normal gelatinous layer beneath the pia mater. Besides the conspicuous areas figured, it could be traced here and there, in small tracts, through the whole of the posterior columns and the hinder parts of the lateral columns, and also in the posterior horns, in the gray matter of which it could be readily distinguished. The relation of the cavities to this substance is distinct; where they extend beyond it this is apparently the result of the distention of the cavities and an actual

FIG. 170.



Cavities in the spinal cord. A, mid-cervical; presenting merely some increase of connective tissue in the left lateral column. In B there are several cavities having the aspect of distended fissures, the largest in the middle line of the posterior columns and the right posterior cornu; smaller ones in the lateral columns. Adjacent to each is a quantity of dense tissue, which had a peculiar and uniform structure. It consisted of a very fine network of fibres with minute nuclear bodies in the interstices. No larger cells could be seen. Throughout the posterior and lateral columns this tissue could be seen, here and there, thickening the neuroglia, where no conspicuous masses were formed. It could also be traced to the posterior cornu, and was readily distinguished from the spongy substance by its slighter staining. The distribution of this tissue is shown in other sections by the darker shading. In C the fissure in the middle line is narrow. In E and F the anterior part of the posterior columns contained a coarse network of this tissue, and no nerve fibres. The central canal was obliterated, but the group of nuclei that marked its position could be traced throughout the cord.

fissuring of the cord, along the lines of connective tissue, by the pressure of the effused liquid. The medial cavity in C is doubtless due to defective closure of the original fissure between the posterior columns. In the lumbar region the nerve fibres are absent from the front of the posterior columns, where a coarse network of this residual tissue encloses empty spaces.

SYMPTOMS.—In many cases syringomyelia is discovered after death, when it has given rise to no symptoms and its existence has not been suspected during life. In young children it appears never to cause any definite symptoms. In adults, however, considerable disturbance of function is sometimes produced. In some cases the growths, which often coexist and which probably start from the residual tissue, may cause the symptoms of tumor. These are often not characteristic, on account of the central position of the growth; the irritation of the nerve roots is commonly absent. In other cases the distention of a central cavity may cause such compression of the white columns and gray matter as leads to definite disturbance of function.

Such disturbance generally involves the central functions of the cervical enlargement of the cord, because it is in this region that the cavities attain their largest size, and the nerve elements of the cord suffer in greatest degree. The gray matter is often especially damaged by pressure, and hence muscular atrophy is one of the most conspicuous of the symptoms that are produced. With it is usually associated loss of sensibility, due to the damage to the posterior gray matter and nerve roots. The wasting resembles in its characters that of progessive muscular atrophy; the muscles of the shoulders and back have suffered more frequently than those of the forearm and hand. The cutaneous anæsthesia usually involves the same region as the muscular wasting, but extends beyond the limits of the latter, and may involve, for instance, the forearms and hands, while the atrophy is limited to the shoulders and upper arms. Pain is not usually a prominent symptom. The conducting functions of the cord are also involved in some degree, but often much less than the central functions, so that there is merely slight weakness of the legs when there is considerable atrophy in the arms. In other cases, however, the paralysis of the legs has been considerable, and there have been muscular contractions in the unwasted parts. The sphincters are often involved, and trophic changes in the skin are frequent, and occasionally are severe. When disturbance of the central functions has developed, the disease usually runs a progressive course and terminates in death at the end of two or three years.

Even when well-marked symptoms have been present, they have so far resembled those of other more common maladies, that the lesion has been seldom suspected during life. The combination of atrophy with more extensive anæsthesia, in the absence of considerable pain, would justify a suspicion of the disease. Pachymeningitis, which also causes muscular wasting and sensory loss, is usually attended with a considerable amount of pain in the affected region, and the anæsthesia is not more extensive, and is usually less extensive, than the muscular wasting. It is not likely that any treatment can influence the morbid process.

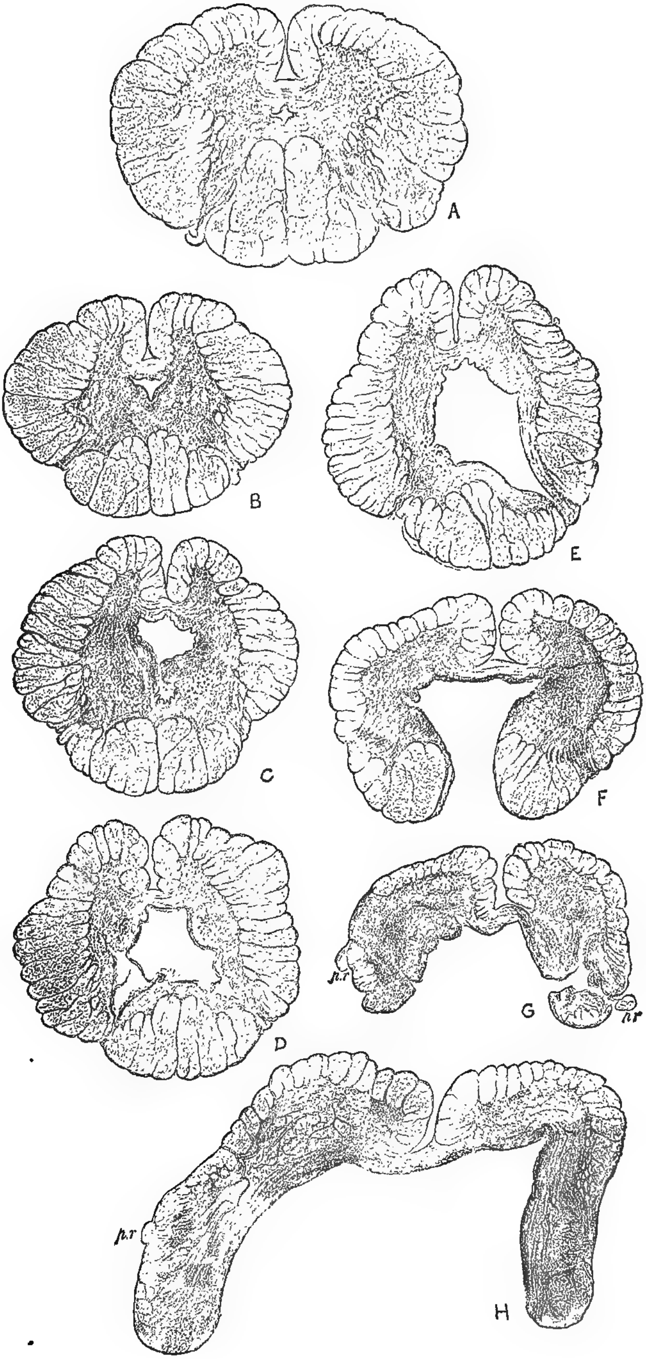
SPINA BIFIDA.

Spina Bifida depends on a defect in the closure of the vertebral arches. It is most common in the lumbar region of the spine; sometimes is present at more than one place, very rarely in the whole length of the vertebral column. It is present in about one child out of every thousand born (Chaussier). The spinal cord is sometimes normal, although the fluid outside it is greatly increased in quantity, and distends the external sac. The cord, however, reaches to the lower extremity of the vertebral canal, as it does at an early period of development, being kept adherent at its lower extremity by the morbid process. In many cases the central canal of the cord is enlarged, and the lower part of the cord may be distended in what has been termed "internal hydrorrhacis." In this condition the central cavity may be closed in behind by a very thin layer of nerve substance. More frequently, however, the cord is the seat of a developmental defect similar to that of the bony canal. In the lumbar region the primitive canal has failed to close, so that the cord is open posteriorly, and the two posterior columns may even be far apart, so that the cord has the form of a thick lamina in the anterior wall of the sac.

An example of the condition is shown in Fig. 171, which illustrates also another fact of the disease, viz., that some developmental effect is often present in other parts of the cord. In the cervical region the gray commissure is unusually large, and the canal is cruciform, a shape which it presents at one period of development, and which is here persistent. This condition obtains through the dorsal region until, in the lower part, the tissue behind the commissure is so abundant as to unite the necks of the posterior horns. Below this the canal enlarges into a condition of syringomyelia, surrounded by the abnormal tissue, which, as we have seen, is constantly associated with enlargement; the canal is limited, moreover, by the sinuous membrane that is so often met with. The cavity extends also into the posterior horns, clearly by a process of disintegration, since in D the membrane lining the enlarged canal persists and marks the limit of the canal and the extent of the simple disintegration. The progressive separation of the posterior columns is shown in F, G, and H. In the lowest part of the cord the development of the posterior columns has been apparently hindered, as will be

FIG. 171.—Spina bifida: sections of a spinal cord. In A, cervical region, the only abnormality is a large cruciform central canal and an unusually deep gray commissure. This continued through the dorsal region, at the lowest part of which a great change occurred. In B the canal is larger, and the necks of the posterior horns (and posterior vesicular columns) are united by much commissural tissue; in this many vertical fibres could be seen, and fibres ran from before backward in the middle line. In C an extension of the canal backward has taken place, and it is bounded by a sinuous membrane. The outer part of the gray substance is atrophied. In D the cavity has extended into each posterior horn almost up to the surface of the cord, in part by a breaking down of tissue, because the membrane limiting the enlarged canal remains undestroyed. In E, still at the junction of the dorsal and lumbar region, the cavity has receded from the left horn. In F the division of the cord has taken place, not in the direction of the cavity in the horn (which is filled up), but in the middle line, at or close to the median septum. In G a wider separation of the posterior columns has taken place, and a cavity has formed on the right side, which extends into the horn, and almost separates the posterior column. In H the cord is spread out and formed part of the wall of the sac. The posterior nerve roots (*p r*) mark the position at which the posterior horn comes to the surface. The gray matter is in many parts atrophied and translucent, and in H contains many large vessels. The septal lobulation on the surface of the cord is greater than normal.

Fig. 171.



observed if G be compared with F. In the former there are also some fissures in the gray substance, also formed apparently by disintegration. The gray substance of the cord is conspicuously wasted in the lumbar region, thin and translucent.

In cases of spina bifida in which the cord is normal there are commonly no symptoms. When the central cavity is large, and the lower part of the cord is greatly distended, or when, as in the case figured, the cord shares the defect of union, there is frequently paralysis of the legs and sphincters, often with muscular atrophy, which is explained by the wasting of the gray matter. Occasionally, the child has been unable to stand, although the legs have been neither wasted nor weak; perhaps the inability in this latter case has been due to the arrested development of the posterior columns.

The treatment of spina bifida is purely surgical.

TRAUMATIC LESIONS OF THE SPINAL CORD.

Causes.—In fractures and dislocations of the spine, the cord generally suffers compression or laceration, as already described in the chapter on injuries of the vertebral column. The cord may also be directly wounded by stabs and gunshot injuries, in which it may be divided, completely or partially. Such injuries furnish a considerable proportion of the cases of unilateral lesion of the cord. In gunshot wounds the cord more often suffers from displaced fragments of bone than from the ball itself.

Far more common than direct injury is damage to the cord by concussion of the spine, either local or general. Gunshot wounds occasionally furnish examples of local concussion; a bullet may strike the spinal column, and lodge in its vicinity, with the effect of causing immediate paraplegia, as complete as if the cord were divided, and yet it may be found that the spinal column has not been injured, and the cord is merely softened at the spot. Other causes of concussion are the fall of heavy bodies on the back, such as a beam of timber or a sack of corn, a blow on the back from some blunt weapon, a fall upon the back, either on a flat surface or on some projecting object. Less frequently the cord suffers from a general concussion of the body, in which the spinal column is not specially involved. The cervical region is occasionally damaged in falls on the head. Railway accidents are frequent causes of concussion of the spinal cord; the back being struck with violence when the body is thrown from one side of the carriage to the other. Another common cause is a fall from a horse upon the back, or a fall downstairs, in which the spinal column is bumped against the edges of the steps. Occasionally, effects very similar to those which are produced by a blow on the spine, are caused by a sudden contraction of the spinal muscles in some violent effort, a "rick of the back" as it is popularly termed. It is possible that, in such cases, the primary damage is sometimes to the vertebral ligaments and articulations, and that the cord suffers secondarily.

Pathology.—The anatomical lesions, in cases in which the vertebral column is not injured, vary much in different cases. Hemorrhage is often found, sometimes outside the dura mater, sometimes on the inner surface of the membrane, in the pia mater, or in the substance of the cord itself. Occasionally, the substance of the cord has been found lacerated when the vertebral column has not been injured. In many cases there is local softening, commonly yellow in tint, sometimes mingled with red, often involving the whole thickness of the cord, and occasionally extending, as central softening, through a considerable vertical extent. Under the microscope, the usual products of degeneration are seen, sometimes with hæmatoidin crystals. Such softening may occur rapidly in severe local concussion, and be found complete a few weeks, or even a few days after the injury.* On the other hand, in some cases of complete paralysis, no lesion of the cord has been found, either with the naked eye or the microscope, a few days after the injury.† In other instances, in which the cord is examined some weeks or months after the accident, the signs of chronic myelitis are found, in scattered foci or more diffuse tracts, in the white columns or gray substance. The nerve fibres are wasted, and the connective-tissue elements are increased in quantity, and, in the early stages, there may be a leucocytal infiltration about the vessels, dilatation of the capillaries, and often minute extravasations, although none were visible to the naked eye. In the anterior cornua, the motor-nerve cells may be damaged, sometimes swollen and vacuolated, or shrunken, and the anterior root fibres may be degenerated. The gray matter is especially apt to suffer when the enlargements are injured; in the dorsal region the change may be confined to the white columns. The usual ascending and descending degenerations may be found above and below the most damaged parts. Occasionally there are indications of meningitis, diffuse or disseminated, and sometimes confined to the dura mater.

Symptoms.—The effects, immediate and remote, of injuries of the spinal cord extend over almost the whole range of symptoms of cord disease, and their variations in character and course are almost infinite. It is therefore neither practicable nor necessary to do more, in this place, than to describe their general characters. According to the differences in course, we may divide them into three classes. (1) Those in which the injury causes immediate and severe paralysis. (2) Those in which there are at first either no symptoms or only trifling disturbance of function, but in which grave symptoms come on a few days or weeks after the injury. (3) Those in which there are no early symptoms, or only slight and transient disturbance, but at the end of one or several months symptoms gradually come on, often such as indicate disease of some definite system of structure of the cord, degenerative in nature.

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* Edmunds, *Brain*, vol. vii, p. 103; Obersteiner, *Wien. Med. Jahrb.*, Bd. iii, 1879; Lochner, *Bayer. Aerztlich. Int.-bl.*, *1875*, No. 42; Fromüller, *Memorabilien*, *1870*, No. 12. In the last, described further on, softening was found thirty-two hours after the injury.

† Fischer, *Deut. Zeitschr. f. Chirurg.*, *1883*, Bd. xix.

The first class, in which the injury causes instant and considerable disturbance of function, includes the cases in which the cord is directly injured, and also some in which there is no visible sign of damage if the patient dies within a few days. In the latter, the mechanical influence has apparently abolished the function of the nerve elements. It is probable that, if such patients lived longer, either quick recovery or local softening would follow. It is an interesting fact that concussion may thus derange function. The absolute integrity of structure on microscopical examination shows that the result is not due to any minute vascular lesion. The effect has been compared by Reynolds, not inaptly, to the demagnetization of iron by a blow. Doubtless the influence is exerted on the molecular nutrition of the nerve elements, and the possibility of recovery, or the subsequent structural disintegration, depends upon the degree of nutritional damage.

The symptoms in these cases of severe and immediate effect are generally those of complete impairment of function. When the injury is direct and partial, such as a hemisection by an incised wound, or a partial bruise of the cord by a spiculum of bone being driven against it (as in the case mentioned at p. 143), the effect may be a partial (*e. g.* one-sided) derangement of function, but in most other cases there is abolition of all the conducting functions at the level of the injury. There is complete paraplegia, motor and sensory, with loss of power over the sphincters. The symptoms are thus those of a total transverse lesion at the affected level (see p. 164). Loss of consciousness is occasionally produced by an injury which does not directly involve the head, and vomiting is very common at the outset. The cases of direct injury often run a severe and rapid course; the tendency to trophic changes in the skin, and to the occurrence of cysitis and pyelonephritis, is very great. Œdema of vaso-motor origin, and effusion into joints, may be present in the early stage. If the cord has been directly injured in its whole thickness, survival beyond a fortnight is rare. If the cord is damaged indirectly, with secondary softening—in what may be termed concussion-myelitis—death occurs less rapidly, but many patients die at the end of four or six weeks, while in those who survive the first two months slow recovery commonly occurs. Even in such cases death sometimes occurs very quickly, but it is possible that there is then a laceration of the cord. A man, whose case has been recorded by Fromüller, was struck on the back, at the level of the third dorsal, by a heavy beam, and had loss of motion and sensation up to the level of the nipples. The palsy of the muscles of respiration increased, and he died from asphyxia at the end of thirty-two hours. At the spot struck the cord was reduced to a pulp for 3½ cm., without any hemorrhage.

In cases of the second class, initial symptoms are absent or slight. After the accident, the sufferer may not imagine himself injured, may be able to walk some distance without inconvenience. In other instances there is tingling in the legs, or in all the limbs, immediately after the concussion, sometimes with some weakness of the limbs, more often with a feeling of stiffness. In the course of a few days graver symptoms come on, usually attended with spinal pain and tenderness, sometimes with some stiffness of the back, and often with pyrexia. Tingling in the limbs increases or develops, and is accompanied by weakness, which often goes on to complete paralysis in the course of a week or fortnight. Sometimes there is also loss of sensation, but less commonly than in the cases in which the symptoms come on immediately. There is often pain in the limbs, and sometimes hyperæsthesia. The symptoms vary in their character and distribution, according to the position of the morbid process and its extent. In some cases, in which the dorsal region is most affected, the symptoms are those of simple paraplegia, usually with spasm, and sometimes with early contraction of the muscles. In other cases, in which the gray matter of the enlargements suffers, there is scattered muscular atrophy in the limbs, often with indications of the degenerative reaction. Tremor in the limbs is a conspicuous symptom in some cases. A girdle pain, or sense of constriction in the limbs, is very common. The sphincters generally suffer, and the tendency to trophic changes is usually strong. The character and course of the symptoms is that of a subacute myelitis, and the meagre facts of morbid anatomy leave no doubt that this is the common lesion. Thus a lady was severely shaken in a railway collision. She seemed, immediately after the accident, to have suffered no injury, but in a few days paraplegia developed, and from its consequence she died six weeks after the accident. Throughout the dorsal region of the cord, I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but, in most parts, considerable in the pyramidal tracts. The symptoms in these cases are sometimes unilateral. A man, driving under a low archway, leaned back to save his head, and pressed his spine suddenly against the sharp edge of the seat back. He felt but little immediate effect, but in the course of two or three days complete motor palsy of the left leg came on, accompanied by hyperæsthesia, but without any loss of sensation either in that leg or in the other. Power began slowly to return at the end of three months. Again, a clergyman was thrown from his horse, and there was immediately sufficient weakness of the legs to prevent him from walking ; this subsequently increased, so that at the end of a fortnight the right leg was completely paralyzed, while the left retained considerable power. There was a girdle pain at the level of the umbilicus, and a bedsore formed, but he slowly improved, and regained the power of standing at the end of eight months.

When the damage involves the gray matter, the extent of the muscular wasting varies greatly. It seldom affects both arms and legs, but it is usually irregular in distribution, sometimes widespread, sometimes limited. Thus a young man fell from a horse and pitched on the head. He was stunned, and on recovering consciousness about two hours later, felt "pins and needles" in both hands, and pain in the back, followed by swelling of the neck and difficulty in moving it, The tingling ceased, but was followed by a sense of oppression about the shoulders, and persistent pain in the cervical region of the spine. When I saw him, two months later, there was some weakness of the right arm and wasting of the two outer interossei and of that part of the long extensor which acts on the two outer fingers, with loss of faradaic and preservation of voltaic irritability.

In cases which survive the acute stage, there is usually slow improvement, which is often ultimately very great. In many cases recovery is incomplete, but improvement goes on for years, and the ultimate degree attained is commonly much greater than in cases of corresponding character and severity due to other causes than injury. But some permanent symptoms are often left when the initial derangement of function has been severe, and has lasted for some time. Thus a heavy weight fell upon a man's back; he was unconscious for two days, and at the end of that time the legs were completely paralyzed, and continued so for three months, with retention of urine. Then improvement commenced in the left leg before the right, and in the latter some spasm developed. At the end of six months he could walk across the room on crutches. Improvement continued, but at the end of two years his condition became stationary, and when I saw him, six years after the accident, he could only walk half a mile; the right leg was still weak in all parts, and a foot clonus was present in each leg, greater in the right than in the left.

In the cases of the third class, chronic symptoms slowly follow an injury at an interval usually of some months, and the cases have the aspect less of a traumatic lesion of the cord than of a primary cord disease, the relation of which to the injury is rather an inference from the sequence, coupled with the absence of other causes, than an obtrusive fact, as in the cases of the first and second classes. The symptoms are usually those of a definite system disease of the cord, less frequently those of a disseminated chronic myelitis of irregular distribution. Primary spastic paraplegia, locomotor ataxy, and progressive muscular atrophy, are the diseases which most frequently thus result. The fact that an injury is occasionally the cause of these maladies has been already mentioned in the account of their etiology, and the symptoms of such cases so far resemble those that are due to other causes that it is not necessary here to add anything to their history as already given. We must assume that the shock to the nerve elements causes a slow perversion of nutrition, which is only manifested by disturbance of function when it has gradually attained a certain degree. Occasionally other causes coöperate, producing a tendency to degeneration. Thus previous syphilis, or neurotic inheritance, can sometimes be traced, and it is a reasonable assumption that the predisposition thus arising may assist in rendering the traumatic influence efficient, or in augmenting its effects.

The consequences of concussion, which we have considered as occurring in separate form, are also sometimes combined. Thus an actual and immediate lesion of the cord may be combined with an early and severe increase in the symptoms from the development of subacute myelitis in other parts. The traumatic inflammation at the spot injured often attains an intense degree, and runs a severe course, in consequence of the influence which may give rise to such inflammation apart from a positive lesion; and, in both cases, the damage to the cord may be perpetuated by a degenerative tendency, aided, in many cases, by the additional predisposition just mentioned. It is very common, for instance, for an injury to the cord, in those who have had syphilis, to cause acute symptoms which subside, but not perfectly, and the residual disturbance of function may persist, and even increase in the course of years, in consequence of the degenerative tendency.

One other symptom remains to be considered, which is common to all forms of traumatic lesion of the cord, and is often severe and persistent when other symptoms are slight,—spinal pain. It may continue for years after other symptoms have ceased, and probably depends on a neuralgic state of the nerves of the membranes, or of those of the vertebral column. Often, probably, the pain depends on the nerves of the joints and ligaments of the spine. It thus appears to be essentially a traumatic spinal neuralgia. The pain may be felt at one or more spots; when severe, it often extends through a considerable length of the spine, and sometimes passes up to the occiput. It is occasionally referred to the sacrum, and may there have the character of a sense of weight or more vague discomfort. The pain is associated with tenderness of the spine, usually deep-seated, chiefly developed at the injured part, but sometimes present also at other spots.

The cause of traumatic lesions of the cord often acts also on the brain. A cerebral lesion may occur from the violence which affects the cord, and cerebral symptoms then coexist with those of the spinal lesion, and may mask the latter during the early stage. More common, however, is functional disturbance of the brain, the result partly, perhaps, of the physical concussion, but chiefly of the mental shock, which a serious accident necessarily causes. The resulting condition is favorable to the development and persistence of subjective sensory symptoms. Attention, maintained by concern, has a powerful intensifying influence on all forms of nerve pain, and certainly aids in keeping up the pain in the back, and even the local tenderness, which follows injuries to the spine. So marked is the influence of "nervousness" on the subjective symptoms, that it has been even maintained that, in a large number of cases of concussion of the spine, the symptoms are of hysterical origin.* "Hypochondriasis" would, perhaps, be a more accurate designation for the condition in most cases in which mental influence plays a distinct part in the development or maintenance of the symptoms. But, on the other hand, it is necessary to avoid the danger of over-estimating the effect of mental influence, and of regarding as entirely due to this symptoms which are real, and are merely intensified by attention. The danger is especially great in cases of railway injuries, concerning which an unbiassed judgment is not easy to secure, and in which, when objective symptoms are absent, it is easy to minimize suffering, and attribute too much to the mental condition. The sinister influence of litigation on the intellect may be traced very widely. I believe that it is rare for symptoms to be purely mental. It is often asserted by those employed for railway companies, that subjective symptoms quickly subside when the sufferer's "claims" are settled, but in a good many individuals whom I have had an opportunity of observing long after they had received their "damages" (as the expression curiously runs) this subsidence had not occurred, and even the "sovereign balm" of substantial compensation has appeared to do very little for the relief of the sufferer.

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* Putnam, *Boston Med. and Surgical Journal*, *1883*, Sept. 6th.

Diagnosis.—The chief points in the diagnosis of traumatic lesion to the cord have been already incidentally considered. Immediate symptoms may be due to laceration, hemorrhage, or to simple concussion, and the diagnosis between these is not always possible at first. If there are immediate symptoms of a partial lesion, these indicate direct injury, while the rapid subsidence of the disturbance of function renders simple concussion probable, and excludes any considerable direct injury. The latter development of paralysis indicates myelitis, unless there is evidence of considerable irritation of the nerve roots at a certain level, which suggests inflammation outside the cord, and perhaps even outside the dura mater. The greatest diagnostic difficulty is presented by the cases just mentioned, in which the symptoms are subjective, and anxious attention has been long given to the local discomfort. It is important to search for and to give due weight to any symptoms beyond the simple spinal pain. Slight "tingling" or "creeping" sensations may be of purely functional origin, but a persistent sensation of "pins and needles" rarely is of that nature. A definite sense of constriction is also strongly suggestive of organic disease, and so is a well-marked difference in the power of the muscles on the two sides. The latter is of least significance if the excess is slight, general, and on the right side,—of much greater significance if the diminution is partial, and affects only certain groups of muscles, such as the flexors of the hip and knee, or the peronei. Any impairment of power over the bladder or rectum is of great diagnostic importance; loss of sexual power, on the other hand, is of little value, since this function is readily depressed by mental anxiety and preoccupation. A slight change in reflex action is most significant when it is partial. A foot clonus, or rectus clonus, is strong presumptive evidence of organic mischief; a slight excess of the knee jerk is of little value; although it probably always indicates some changes in the nutrition of the spinal cord, it does not render structural disease even probable. In all cases it should be remembered that the absence of any common symptom is of far slighter significance, as evidence of integrity of the cord, than is the presence of that symptom as evidence of disease. It may seem superfluous to insist on a consideration so elementary, but it is still possible, as experience proves, for a medical witness to assert in a court of law that a claimant's spinal cord cannot have been injured because his legs are not wasted.

Prognosis.—Immediately after an accident a cautious prognosis should be given, even if the symptoms are slight, on account of the possibility that grave disturbance may develop in the course of a few days. In developed cases the prognosis must, in general, be guided by the same considerations as those which determine our estimation of the probable course of symptoms of similar character and severity due to spontaneous myelitis. To this there are, however, two general exceptions. First, the danger of death, if any exists, is greater in traumatic cases than in others. Secondly, if there is no danger to life, or such danger has passed, the prospect of improvement is distinctly greater than in a case of similar features but of non-traumatic origin. If the symptoms are slight or moderate in degree, approximate recovery may be anticipated. Often, however, the recovery, although approximate, is not perfect. The patient is never quite as strong, quite as capable of exertion, as before the injury. A cautious prognosis should be given whenever there is the late and gradual onset or increase of symptoms that suggests a degenerative process. Such degeneration presents far less tendency to arrest or subsidence than do the earlier lesions.

Treatment.—In all cases in which spinal symptoms are present immediately after an injury, however slight those symptoms may be, absolute rest for three or four days should be insisted on. This is necessary on account of the secondary inflammation which, as we have seen, so often occurs. The treatment of developed symptoms must be conducted on the same general principles as in cases of myelitis; the details need not be here repeated. If there is muscular wasting, it is important that the nutrition of the muscles should be maintained by electrical stimulation, since a very considerable amount of ultimate recovery may be anticipated, and it is important to keep the muscular tissue, as far as possible, in a condition to respond to the nervous power when this returns. If there is reason to believe that there is inflammation of the membranes, or inflammatory effusion outside the cord compressing it, mercury may be given, but this condition is probably much more rare than might be anticipated. The influence of mercury on inflammation of the substance of the cord is doubtful. The degenerative sequelæ of injuries to the cord need the same treatment as the similar degenerations that occur apart from traumatic influences.

The treatment of the neuralgic condition of spinal pain and tenderness, which so often succeeds injury, is frequently difficult. Counter-irritation is often useful, either by the actual cautery, blisters, iodine, or repeated sinapisms. Of sedatives, Indian hemp is most effective, next to morphia, which should be used as seldom as possible. Hypodermic injections of cocaine may be tried. When all active mischief is over, and the pain has become purely neuralgic, it is often necessary to encourage the patient to neglect it in some degree, and to exert himself in spite of it, while avoiding whatever increases it in considerable degree and for a considerable time.

FUNCTIONAL AND NUTRITIONAL DISEASES.

FUNCTIONAL DISEASES.

Very little is known, though much is heard, of functional diseases of the spinal cord. It is indeed open to doubt whether there are any morbid states which can accurately be thus designated. Hysterical paraplegia is often regarded as a functional affection of the cord, because the symptoms have the same distribution as those of organic diseases of the cord, but a little consideration will show that, in a case of purely hysterical paraplegia, the morbid functional condition is cerebral; the brain centres which act on the legs are at fault, but the condition of the functions of the cord itself may be absolutely normal. The spinal motor centres are in a state of inactivity, because the related cerebral centres are inactive, but this is no more a diseased condition of the cord than is its corresponding functional state during physiological rest. Hysterical paraplegia will be described, with other palsies of like origin, in the chapter on hysteria.

Isolated disturbance of functional centres in the cord does, however, sometimes occur. It is seen in the conditions of inhibition which are designated "reflex paralyses." Such palsies were once thought to be common and persistent, but it has been proved that many, and it is probable that all, cases of considerable and prolonged palsy, formerly thought to be of reflex origin, are due to organic disease, either primary in the cord or secondary to an ascending inflammation of nerves. But transient paralysis sometimes occurs which cannot be otherwise explained than as an inhibition of a spinal centre due to peripheral irritation. Such, for instance, is the curious inability to pass urine which sometimes follows an operation on the anus, the division of a fistula, for instance, or the removal of hemorrhoids. The inability may continue absolute for several days. Transient weakness of one arm is said sometimes to follow an operation for empyema (Lepine), but the fact that the weakness may be attended by choreoid movements in both arm and leg of one side (Weill) makes it probable that the influence is exerted on a cerebral rather than on a spinal centre. Considerable paraplegia was thought to be sometimes a reflex effect of disease of the bladder or of a calculus or other organic disease in the kidney, but it is probable that such cases are always of organic nature.

Another class of cases which may be regarded as functional are those in which symptoms, commonly subjective in character, result from some morbid blood state. The conditions which most frequently have this effect are gout and diabetes. Occasionally there is definite failure of power, lasting for a few days or weeks, without objective symptoms, and passing away. But the most common symptoms from this cause are sensory and subjective,—feelings of tingling and formication in the legs, dull aching, and sometimes actual pain; this is usually transient, but occasionally continues for some days or weeks, various in position, but in gouty cases often felt in the heels. Such symptoms, due to morbid blood states, occur chiefly during the second half of life. Those of gouty origin, like other symptoms of the same class, are especially common in persons who inherit a tendency to gout but have not suffered from attacks of definite arthritis. The treatment of these symptoms is chiefly that of the blood state which causes them, but they may be to some extent relieved by sedatives, of which bromide of potassium, Indian hemp, and cimicifuga are the most effective.

NUTRITIONAL DISEASES.

Among the cases often classed as functional diseases of the spinal cord are some in which objective symptoms of deranged function, slight in degree but definite in character, persist for months, years, or for life. They occur chiefly in those in the early and middle period of adult life, and are more common in women than in men. Such symptoms are inability to walk more than a short distance without fatigue, impaired nutrition of the legs, slight increase of myotatic irritability, often associated with pain in the back. If the history of such symptoms is traced they will generally be found to date from some definite exciting cause, from an attack of acute illness, such as typhoid fever or acute rheumatism, from pregnancy, a fall, and the like. The increase in myotatic irritability is exceedingly common; it is enough to permit the knee jerk to be obtained by tapping the depressed patella, but there is rarely a distinct clonus,—at most only two or three jerks, quickly ceasing. We can hardly conceive that symptoms which are so persistent can depend on any mere functional derangement; it is probable that they depend upon changes in the finer nutrition of the nerve elements, too slight to be detected by the microscope, causing a corresponding and persistent alteration of function. We have seen (p. 154) that the termination of the upper segment of the motor path is probably that structure of the cord which has least nutritional stability, and is therefore most susceptible of nutritional derangement. Hence we can understand the frequency with which there is an excess of myotatic irritability in these cases. There are probable gradations between such conditions and actual structural disease, in which minute examination reveals visible alteration. We have already had occasion to consider these nutritional changes as probably underlying the condition of arthritic muscular atrophy, and we have seen that the alterations in spinal nutrition in that disease are apparently the result of the impressions on the peripheral nerves. In women such a condition is often associated with uterine or ovarian pain, and with sacral pain, apparently of uterine origin. It is possible that the condition we have been considering is sometimes secondary to uterine pain, which acts in a manner somewhat analogous to that in which joint inflammation acts, and that this is the explanation of some of the cases in which a reflex disturbance of the functions of the cord has been supposed to be of uterine origin. The condition is often associated in women with spinal pain and tenderness, analogous to that which has been already described as secondary to injury, and which we shall have again to consider as a variety of neuralgia. Thus many cases of nutritional and so-called functional derangement of the cord are very complex in character.

It is rare for the indications of nutritional disturbance of the cord to pass away altogether when they have once become established. The treatment of this condition is first the removal of any present cause of depression of function, such as the diminution of uterine or spinal pain; secondly, the improvement of the general health and the avoidance of over fatigue; thirdly, the administration of nervine tonics, such as arsenic, quinine and strychnia. A course of massage treatment is often of distinct service.

PART IV.

DISEASES OF THE BRAIN.

INTRODUCTION.

THE STRUCTURE AND FUNCTIONS OF THE BRAIN.

THE knowledge we possess of the cerebral structure and functions is derived from various sources. Simple dissection of the brain reveals only the coarser outlines of its anatomy, and is apt to mislead the investigator who endeavors, by its aid alone, to unravel the complex connection of the several parts. By the aid of the microscope the structure of isolated portions can be ascertained, but the difficulty and even impossibility of tracing the course of nerve fibres, where thousands interlace, places a narrow limit to the results to be obtained from simple microscopical examination. But the differentiation effected by processes of degeneration on the one hand, and of development on the other, has enabled many facts to be ascertained which are of the highest importance, and which must otherwise have remained unknown. The close correspondence between the results obtained by these two methods, the study of development and of degeneration, not only shows their high value, but also justifies confidence in the indications afforded by either, where the two cannot be combined. The credit of discovering the fact and significance of secondary degeneration belongs to Türck, and Flechsig first showed the value of the structural indications of development, by investigations which have placed many points in the anatomy of the brain on a footing far firmer than any other method could supply.

The functions of the brain have been ascertained by three methods. First, the structural arrangement of its parts affords important suggestions as to their function—suggestions which are valuable in proportion to the simplicity of the structure, and the distinctness of its connections. For instance, a group of nerve cells gives origin to the fibres of a cranial nerve, and from this it is inferred that the function of these cells is to excite the nerve fibres, if the nerve is motor, or to receive the impulses which traverse them, if the nerve is sensory. Another source of knowledge is the result of experiments upon animals, in which portions of the brain are removed, or destroyed, or stimulated, and the effects are observed. The results obtained in this manner by Hitzig, Ferrier, Munk and others, are of very great importance; but so far as the human brain is concerned, they are suggestive, often highly suggestive, but they are not demonstrative, and the conclusions thus reached cannot be accepted as certainly true of man except in so far as they receive confirmation from the third source of knowledge, the experiments wrought upon the human brain by processes of disease or by accidental violence. It is to these that we have to look for exact knowledge and certain proof of the functions of the several parts of the brain of man. Unfortunately, the conditions of disease and injury are usually very complex, and their manifestations are correspondingly involved and difficult to interpret. Hence our knowledge grows but slowly, in spite of the enormous amount of careful observation which, at the present day, is directed to this subject in all parts of the world.

The following account of the structure and functions of the brain does not profess to be complete, even in outline. Its object is to remind the student of the facts he most needs to know, in order to comprehend the symptoms and pathology of disease. Unfortunately, our knowledge is still imperfect, even of the fundamental facts. Many questions of great importance are still unanswered, while to others widely different answers have been given by investigators of equal competence. The statements here made on such points are the result not only of a comparison of the work of others, but also, in many instances, of an independent examination of the point at issue. The functions of the brain will be considered very briefly, since many of them must be discussed more fully in the account of the symptoms of disease.

The few points in the anatomy of the membranes which are of medical importance will be most conveniently considered in the section on their diseases.

THE CEREBRAL CORTEX.

The anatomy of the convolutions of the brain has become of great practical importance, on account of the definite localization of function found to obtain in certain parts. For a full account of the convolutions the reader is referred to works on Anatomy, but the most important facts may be briefly enumerated. The *longitudinal fissure* separates the two hemispheres. In each half of the brain three fissures are of leading importance (see Fig. 172), the fissures of Sylvius and of Rolando on the outer surface, and the parieto-occipital fissure, the extremity only of which appears on the convexity. The *Sylvian fissure*, at the base, separates the prominent temporal lobe from the orbital surface of the frontal lobe, and, on the outer surface, divides into a very short anterior limb, and longer posterior limb; sometimes there are two short limbs, one anterior and the other vertical (see Figs. 172 and 174). The posterior limb separates the temporal lobe below from the parietal lobe above. Just above the bifurcation of the fissure of Sylvius, but not joining it, is the lower extremity of the *fissure of Rolando*, or *central sulcus*, which passes upward and backward to the longitudinal fissure, and separates the frontal from the parietal lobe. The *parieto-occipital fissure* begins at the longitudinal fissure, about midway between the upper end of the fissure of Rolando and the posterior extremity of the hemisphere. It is better marked on the medial than on the outer surface of the hemisphere; on the latter it extends only for a short distance from the longitudinal fissure. It marks the boundary between the parietal and occipital lobes. These three fissures are the chief landmarks on the outer surface, and are our guides in identifying the various convolutions. On the inner or medial aspect (Fig. 173) the parieto-occipital fissure, at its lower extremity, joins another sulcus which extends backward to the posterior extremity of the brain, the *calcarine fissure*, and the two bound a wedge-shaped area, the *cuneus*. In the anterior half of this inner surface, the *calloso-marginal fissure* runs parallel with the corpus callosum, midway between it and the edge of the hemisphere, to which the posterior extremity of this fissure turns up, behind the upper end

FIG. 172.

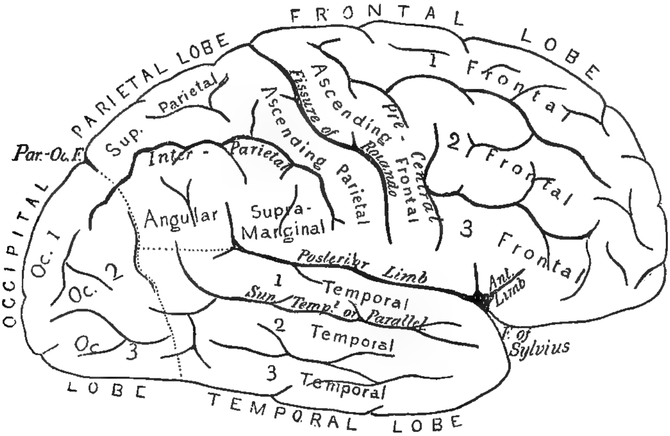

Diagram of the convolutions and fissures on the outer surface of the right hemisphere. The fissures are indicated by italics.

FIG. 173.

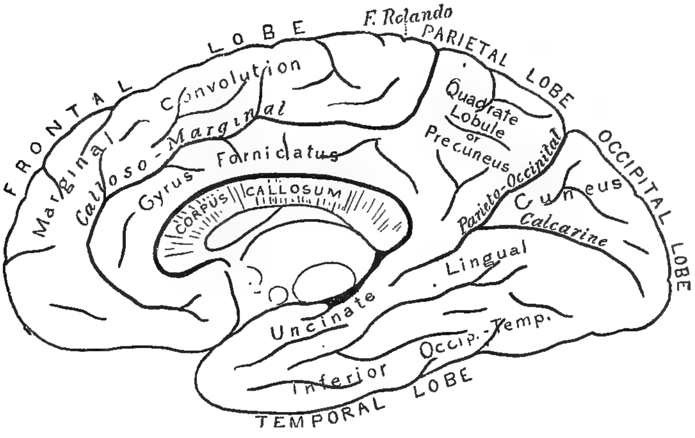

Diagram of the convolutions and fissures on the inner (medial) surface of the right hemisphere.

of the fissure of Rolando. Between this up-curved end and the cuneus is an area termed the *pre-cuneus* or, from its shape, the *quadrate lobule.*

The fissure of Rolando runs between two convolutions which have the same direction as the fissure. These are the *ascending frontal* and *ascending parietal* convolutions. These two gyri are of great importance, because they contain the chief so-called "motor" centres. They are sometimes termed the "central convolutions," "anterior," and "posterior." They unite below the lower end of the fissure of Rolando, and the region that occupies the bifurcation of the fissure of Sylvius is often termed the "operculum." The ascending frontal is bounded by a fissure, the *precentral sulcus*, usually interrupted by the origin of one or two of the remaining frontal convolutions, *upper, middle*, and *lower;* or *first, second*, and *third.* The highest is at the margin of the longitudinal fissure, the lowest curves round the anterior limb of the fissure of Sylvius. The upper part of the ascending parietal convolution blends with the upper part of the parietal lobe in the *superior parietal lobule;* this also forms part of the motor region, and so does the medial aspect of these two central convolutions, the *paracentral lobule* it is termed, in front of the up-curved end of the calloso-marginal fissure. The lower part of the parietal lobe, behind the ascending parietal convolution, is termed the *inferior parietal lobule*, and is separated from the superior lobule by the *inter-parietal fissure.*

The lowest parietal convolution, which bounds, above, the posterior limb of the fissure of Sylvius, is termed the *supra-marginal convolution.* In some brains this constitutes almost the whole of the inferior parietal lobule. By some authorities (Turner, etc.) the supra-marginal gyrus is regarded as ceasing above the extremity of the upturned end of the fissure of Sylvius; by others it is regarded as passing round this, so that the gyrus immediately behind the upturned portion of the fissure is included in the name. Behind the upturned fissure is the *angular gyrus*, which extends back to the occipital lobe, upward to the intra-parietal sulcus, and downward is continuous with the first and second temporal convolutions. Its posterior limit is somewhat arbitrary, corresponding to a line drawn across from the parieto-occipital fissure. Its anterior limit varies according to the area assigned to the supra-marginal convolution.*

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* The importance attached to the angular convolution renders it desirable to point out the different senses in which the word is used. The term was taken from the "*pli courbe*" of Gratiolet, applied by him to the convolution in the brain of the ape which winds round the top of the fissure of Sylvius, and as a simple gyrus, is limited behind by the parieto-occipital fissure, which extends, in the ape's brain, far on the outer surface of the hemisphere. But in applying the term to the more complex brain of man, Gratiolet restricted it to the posterior part of the region between the extremity of the fissure of Sylvius and the occipital lobe. The term is now used in three senses. (1) The whole region from the end of the fissure of Sylvius to the occipital lobe. (2) The posterior two-thirds of this region, a small sulcus, parallel with the upturned fissure of Sylvius, being taken as the anterior limit of the angular and posterior limit of the supra-marginal, which is thus regarded as passing round the fissure of Sylvius. In this sense the parallel fissure passes into the angular gyrus. (3) The pos-

The convolutions passing from the parietal to the occipital lobes on the outer surface are called "annectant;" the highest of these is at the outer extremity of the short parieto-occipital fissure.

In the *occipital lobe* three short convolutions may often be distinguished, *upper, middle* and *lower.* The upper one is continuous with the superior parietal lobule by the first annectant gyrus. These occipital convolutions are often not distinctly separate. In the *temporal lobe* two or three antero-posterior convolutions can be distinguished on the outer surface. Of these the *upper,* or *first,* forms the inferior boundary to the fissure of Sylvius, and is continuous behind with the supra-marginal or the angular convolution, or with both, according to the sense in which these words are used. The fissure below this convolution is termed the *parallel fissure,* because it is parallel with the fissure of Sylvius. The lower convolution or convolutions (if two can be distinguished) are continuous with the occipital lobe.

On the medial surface of the temporal lobe (Fig. 173) two or three similar convolutions are visible; and of these the upper one is the *uncinate* gyrus, so called because its anterior extremity is shaped like a hook. Beneath the calcarine fissure is the *lingual gyrus.* In the rest of the medial surface the *gyrus fornicatus* lies next to the corpus callosum, but only constitutes a well-marked convolution in the anterior half of the brain, where the calloso-marginal fissure separates it from the *marginal convolution,* which is, for the most part, the medial aspect of the highest frontal convolution. The other divisions of the medial surface have been already enumerated. Within the fissure of Sylvius lie the small convolutions of the *island of Reil,* or *insula* (Fig. 174), four or five in number (I to V, Fig. 174), which spread out like a fan. Behind the insula, but still entirely within the fissure of Sylvius, two or three convolutions connect the first temporal with the parietal lobe. They have been termed the *retro-insular* or *temporo-parietal convolutions* (R I, Fig. 174). Neither these convolutions, nor those of the insula, are visible until the lips of the Sylvian fissure are separated. Hence they often escape examination, and this is probably one reason why little is known of the effect of their isolated disease. It will be observed that the boundary between the frontal and parietal lobes also separates the two "ascending" convolutions, which have been found to have functions at once similar, important, and unlike those of most other parts of the brain. Hence it is often convenient to distinguish that part of the frontal lobe which lies in front of the ascending convolution as the "prefrontal lobe."

Relation of the Cortex to the Skull.—The correspondence between certain parts of the surface of the brain and of the skull has been carefully

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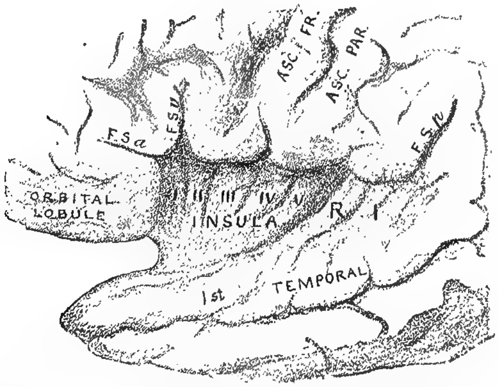
terior third of this region: the anterior limit being the parallel fissure, to which the supra-marginal will then extend. (4) The term is sometimes used (and the corresponding "*pli courbe*" by French writers) to include not only the whole of this region, but also that part of the supra-marginal gyrus which lies in front of the extremity of the fissure of Sylvius. This makes it correspond to the whole region into which the *pli courbe* of the ape has developed in the human brain; but it is inconsistent with the application of the word to the human brain by Gratiolet himself and most other authorities.

investigated by Turner,* Reid,† and Horsley; ‡ by the last two, rules have been formulated by which the position of the various fissures and convolutions may be determined. The general relation of the brain to the bones of the skull is shown in Fig. 175. Beneath the frontal bone lie nearly all the lowest frontal, five-sixths of the middle and about three-quarters of the upper frontal convolutions. The temporal bone covers the temporal lobe except its posterior fifth and anterior extremity. Less than half the occipital lobe lies under the occipital bone. The rest of the cerebral cortex lies beneath the parietal bone.

Horsley's Rules.—My colleague, Mr. Victor Horsley, who has had much practical experience in the surgery of the brain, employs a method of finding the position of the important centres of the brain, of which he has published an account in the *International Journal of Medical Science* for April, 1887. From this the following abstract is taken:—

Fig. 174.



Convolutions within the fissure of Sylvius. F S *a* its anterior and F S *v* its vertical, and F S *p* the extremity of its posterior limb. I–V the short gyri of the insula; R I, retro-insular convolutions connecting the temporal and parietal lobes.

The chief landmarks on the surface of the skull are (1) the parietal eminence and (2) the curved temporal ridge, which is really double, a lower one, which is best marked and limits the attachment of the temporal muscle, and an upper one to which the temporal fascia is attached. The lower one can be best found by making the patient contract the muscle firmly; the upper one marks the sudden change in the slope of the skull to the vertical direction. (3) The coronal suture, which can generally be felt just above the place where the temporal ridge crosses it (this crossing is termed by Broca the stephanion). If the upper part cannot be felt, its position can be ascertained by finding its junction with the sagittal suture.

The parieto-squamosal suture lies beneath the temporal muscle: the highest point of its curve is in a vertical line passing just in front of the articulation of the lower jaw, and it is there two-thirds of the distance from the zygoma to the temporal ridge.

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* *Journal of Anatomy and Physiology, 1873, '74.*

† *Lancet, 1884.*

‡ *Int. Jour. of Med. Science, 1887, April.*

The short junction of the anterior inferior corner of the parietal bone with the wing of the sphenoid (corresponding nearly to the division of the Sylvian fissure) is about half-way between the stephanion and the upper border of the zygoma.

The upper end of the fissure of Rolando is best found by the plan of Professor Thane. Measure the distance along the middle line from the root of the nose to the occipital protuberance, and find the middle point of this distance; half an inch behind this point is the upper extremity of the fissure of Rolando. The fissure makes an angle with the middle line of 67°. Mr. Horsley uses a long strip of soft metal, from which projects another piece at this angle.. When this strip is laid along the middle line of the head, with the junction of the two parts over the spot mentioned, the arm corresponds in direction to that of the upper two-thirds of the fissure of Rolando. In its lower third the fissure is rather more vertical.

FIG. 175.

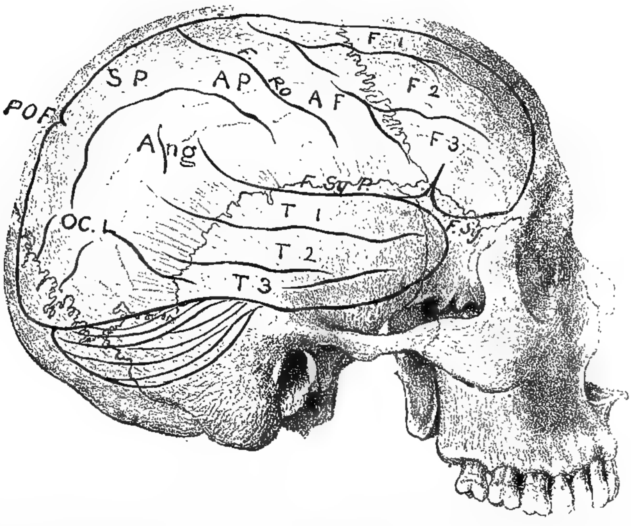


Diagram of the relation of the convolutions to the skull. F 1, 2 and 3, upper middle and lower frontal convolutions; A F, A P, ascending frontal and parietal; S P, sup. parietal lobule; Ang. angular gyrus; Oc L, occipital lobe; T 1, 2, 3, the temporal convolutions; P O F, parieto-occipital fissure; F Sy and F Sy P, fissure of Sylvius and its posterior limb.

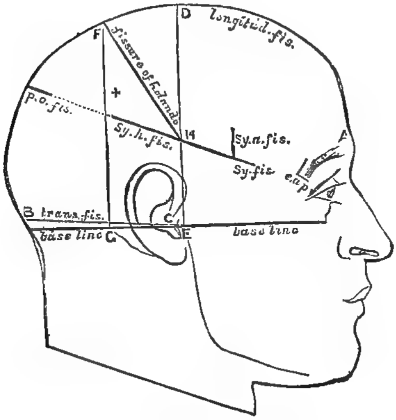
The anterior limb of the fissure of Sylvius runs upward from the parieto-sphenoidal junction above mentioned. The posterior limb passes backward and upward just above the ascending part of the parieto-temporal suture, and from the highest part of this it curves upward toward the centre of the parietal eminence.

The lower half of the precentral sulcus is parallel to and just behind the coronal suture. The inter-parietal sulcus lies, in its ascending part, midway between the line of the fissure of Rolando and the parietal eminence, and then passes backward midway between the latter and the middle line of the skull.

The ascending frontal convolution begins below, beneath the anterior inferior angle of the parietal bone, in front of the prolonged line of the fissure of Rolando, between this and the prolonged line of the lower part of the precentral sulcus; in front of the latter will be the root of the lower frontal; behind the ascending frontal the root of the ascending parietal.

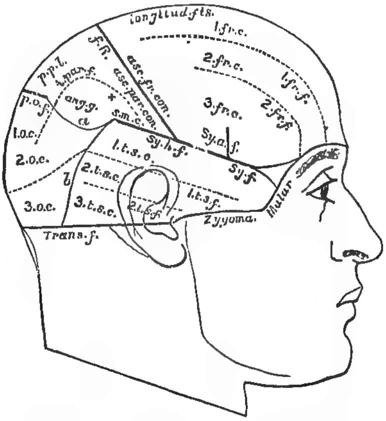
Reid's Rules.—The most important of Reid's rules are the following: some additions are enclosed in brackets. As a base is taken a line running from the inferior margin of the orbit (Fig. 176), through the middle of the auditory meatus. The posterior limit of the fissure of Sylvius lies beneath the hinder three-fifths of a line drawn from the external angular process of the frontal bone to a point three-quarters of an inch below the most prominent part of the parietal bone (this may be termed the Sylvian line). The anterior limb of the fissure ascends from this line above the middle of the zygoma. The temporal ridge corresponds nearly to the fissure between the middle and lowest frontal convolutions. If the Sylvian line is prolonged to the sagittal suture, it will give the position of the parieto-occipital fissure (and the occipital lobe, half-vision centre, lies between this and the base line). A perpendicular from the base line, at the depression in front of the auditory meatus, cuts the Sylvian line where the fissure of Rolando, if prolonged, would join the Sylvian fissure, and the position of the fissure of Rolando is under a line drawn from this point to the place at which the sagittal suture is cut by a perpendicular drawn to the base line drawn from the posterior border of the mastoid process. The central convolutions occupy about an inch on each side of the Rolandic line. The position of the centres for the leg, arm and face can thus readily be ascertained, since they lie on each side of the fissure of Rolando. The angular gyrus lies immediately behind the most prominent part of the parietal eminence. The first temporal convolution lies below the Sylvian line, over the ear and mastoid process.

FIG. 176.



Guiding lines of Reid.

FIG. 177.



Relations of the fissures and convolutions to the guiding lines.*

STRUCTURE OF THE CORTEX.—The nerve fibres of the white substance radiate into the cortex, passing between the nerve cells, and in places separating these into vertical groups. The cells are of various forms and sizes, certain of which predominate at different depths from the surface, thus permitting a distinction into layers. Each layer contains many kinds of cells, but those which are in greatest number give to the layer its distinctive character. Considerable variations exist in different parts of the brain in the number, characters, and relative thickness of the layers, and their precise division has been the subject of much discussion.* There is especially a marked difference between the characters of the cortex in the central convolutions (ascending frontal, ascending parietal, and paracentral lobule) and in the rest of the outer surface of the hemisphere. It may be said generally that the three layers next the surface present nearly the same characters over the greater part of the cortex, varying, however, in thickness. The deepest layer, next the white substance, is also very uniform in its character; the chief variations are in the elements which intervene between the three superficial layers and the deepest layer.

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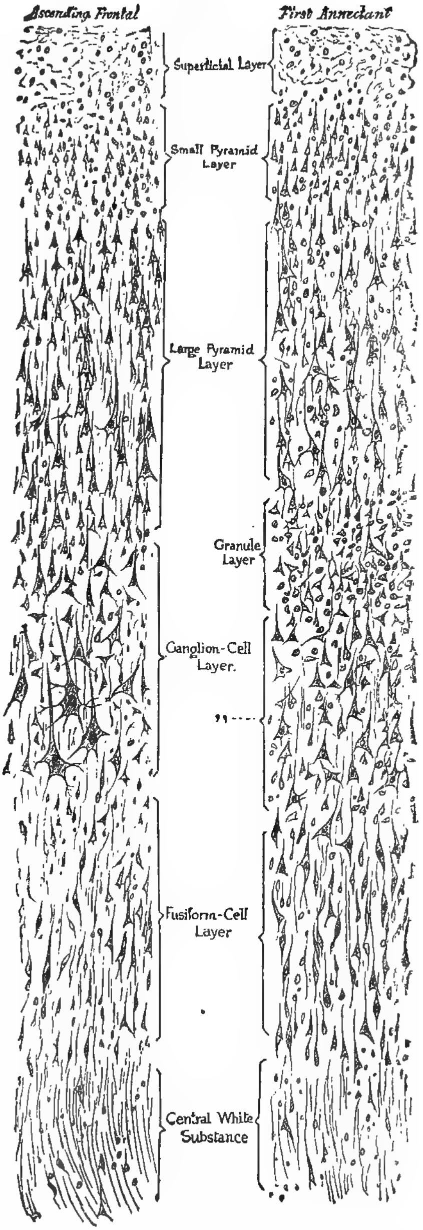
* I am indebted to Dr. Reid for permission to reproduce these figures.

In the central convolutions (as the ascending frontal) the layers present the cell forms shown in the first column in Fig. 178. Above, next the surface, is a narrow *superficial* layer containing few cells, and those of doubtful nature, chiefly small corpuscles, more or less rounded. Similar corpuscles are also scattered through all the layers. This superficial stratum contains also many delicate nerve fibres (Remak, Lockhart Clarke, Exner). Beneath this is a layer of *small pyramidal cells* densely massed, with a few nuclear elements. The cells have the apex of the pyramid turned toward the surface. The next layer is composed of *large pyramidal* cells, less densely massed, and partially separated into columns by the bundles of nerve fibres. The cells in the upper part of the layer are little larger than those of the second layer, but they increase in size in the deeper parts. The apex of each is turned toward the surface and from it a long process may be traced. From the centre of the

FIG. 178.



Diagrammatic sketch of the layers of the cortex cerebri. The drawings were made from sections of the ascending frontal and first annectant convolutions (the latter near the extremity of the parieto-occipital fissure). The sections were kindly furnished me, as representative of the so-called motor and sensory types, by Dr. Bevan Lewis.

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* They were studied, first, by Baillarger, thirty years ago; subsequently by Lockhart Clarke, Meynert, and others; and, more recently, they have been investigated with much care by Betz and Bevan Lewis.

base another process extends, continuous, it is said, with the axis cylinder of a nerve fibre, while from the angles branching processes are given off. The small pyramidal cells occur also, in fewer number, throughout this layer, and at some parts of the brain they are more numerous in the deepest part of the layer. The next layer is characterized by nerve cells which are irregular in shape, and often triangular. Many of them resemble the motor nerve cells of the spinal cord, and hence it has been proposed by Bevan Lewis to call this layer the *ganglion-cell layer.* The cells vary much in size. Some are small angular elements, the size of the small pyramidal cells of the second layer, others are as large as the large pyramidal cells of the third layer, but both differ from the cells of the second and third layer in being very rarely distinctly pyramidal in form and in being less regularly placed. Moreover, in this layer some cells are met with which exceed the size of the largest pyramidal cells; some are even three times the size of the latter. These are the "giant cells" of Betz, and were once regarded as pathological, but every gradation is seen between them and the ordinary ganglion cells of the layer. The largest cells occur in groups of two to five, and are almost confined to the central region, while the smaller ganglion cells of this layer are met with over the greater part of the surface of the brain. Beneath this stratum is a layer in which, with a few angular cells, there are many fusiform cells. Hence it is called the *spindle-cell layer.* It is the deepest layer of the cortex, and beneath it is the white substance of the hemisphere, among the fibres of which are many nuclei and a few angular and spindle cells, especially near the cortex. Thus the type here is five laminated.

In the region of the brain behind the central convolutions (and also in the anterior part of the frontal lobe), the structure of the cortex more or less resembles that shown in the second section in Fig. 178, which is from the first annectant gyrus. It will be seen that the first three layers, superficial, small pyramid, and large pyramidal layers, closely resemble those of the other section, and so also does the deepest or spindle-celled layer. The ganglion-cell layer is rather narrow and contains no very large cells. Above it, and between it and the large pyramid layer, is a stratum of small round or angular granule-like elements with a few rather larger, but still small, angular cells. It has been called the *granule layer.* At the extremity of the occipital lobe this granule layer is considerably larger, and the pyramidal cells are much less developed, while in the cornu ammonis the latter are alone found. The claustrum is composed of fusiform cells, and hence is regarded as a detached part of the deepest layer.

We can merely surmise the functional significance of the several varieties of cells. It is important to note that the transition from one type of structure to another is everywhere gradual, and that (with the exception of the very large ganglion cells) each form of cell can be found in almost all parts of the brain. The localization of the largest ganglion cells in the so-called motor regions, and the analogy between these and the motor cells of the cord, make it highly probable that they are motor in function. It has been conjectured that the large pyramidal cells are also motor, but from the wide extent of this formation it is possible that the function of these cells varies according to their connections. The preponderance of the granule cells at the posterior portion of the brain, and their similarity to the cells of the posterior cornu of the spinal cord, has suggested that they are sensory in function.

FUNCTIONAL REGIONS OF THE CORTEX.—Doubt was formerly entertained as to the existence of differentiation of function in different parts of the cortex, but recent researches have established the existence of a differentiation which has almost revolutionized cerebral physiology, and has vastly extended the range of cerebral diagnosis. The first step of the new discovery was constituted by the clinical and pathological investigations of Hughlings Jackson, which suggested the existence, on each side of the fissure of Rolando, of special centres for the movements of the leg, arm and face. These observations led to the experiments of Ferrier, which resulted in the demonstration of the existence in the cortex of the lower animals of well-defined regions, stimulation of which caused separate movements, or evidence of special sense excitation, while the destruction of the same parts caused indications of a loss of the corresponding function. Hence he came to the conclusion that these regions constitute actual motor and sensory centres. Ferrier had, however, been anticipated in many of these results by two German experimenters, Fritsch and Hitzig, whose results, differing a little in detail, correspond closely in their general significance. Many other investigations of the same character have since been made, of which those of Munk are especially important. The original observations of Hughlings Jackson left little doubt that the general facts, learned from experiments on animals, are true of man; and this conclusion has been to a large extent confirmed by pathological and clinical observations directed to the verification on man of the experimental results. To this verification the labors of Charcot and his coadjutors have largely contributed. But the verification has already made it probable that some differences exist between the brain of man and of other animals (even of monkeys), and that the conclusions from the latter cannot be simply transferred to the former.

CORTICAL CENTRES IN THE MONKEY.

Before considering the indications of the position of the various centres in the cortex of the human brain, it may be well to enumerate the chief functional regions ascertained by Ferrier and others in the brain of the monkey.

MOTOR CENTRES.—*Superior parietal lobule (except the part adjacent to the fissure of Rolando):* movement of the leg and foot, flexion of the hip, extension of the knee, flexion of the ankle.

Highest and adjacent parts of the ascending frontal and parietal convolutions, close to the margin of the hemisphere, together with the root of first frontal: flexion and outward rotation of thigh, rotation inward of leg, flexion of toes, as in scratching abdomen with foot. (Horsley and Schäfer* obtained movements of the leg also from the medial aspect of the convolutions on each side of the extremity of the fissure of Rolando. Horsley and Beevor found movement of the great toe chiefly represented in the upper extremity of the ascending frontal.)

Adjacent parts of ascending frontal and parietal, outside the last centre but still opposite the highest frontal convolution. adduction and extension of arm, pronation of hand.

Ascending frontal and base of the highest frontal: extension of elbow, movement forward at shoulder, and synchronous movement of leg. (Horsley and

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* *Proc. Roy. Soc., 1885.*

Schäfer obtained movements of the arm from the medial aspect of the posterior half of the highest frontal.)

Ascending frontal, opposite upper part of middle frontal: supination of hand and flexion of forearm.

Middle three-fifths of ascending parietal: movements of hand, especially clenching of fist.*

Ascending frontal, opposite lower half of middle frontal: elevation and retraction of angle of mouth.

Ascending frontal, opposite highest part of third frontal: elevation of upper lip and ala of nose.

Lowest part of ascending parietal: retraction of angle of mouth by platysma.

Lowest part of ascending frontal: movements of lips and tongue (and in the anterior part, closure of the vocal cords, according to Horsley and Semon).

Posterior half of upper and middle frontal convolutions: lateral movement of head and eyes, with elevation of eyelids.

(*Medial aspect of top of ascending frontal, i. e.* corresponding region of the marginal convolution, movement of the trunk.—Horsley and Schäfer.)

SENSORY CENTRES.—Stimulation of the angular convolution caused a movement of the eyes to the opposite side, suggestive of a visual sensation. Extirpation of the angular gyrus caused amblyopia of the opposite eye, more or less transient, and destruction of this convolution in both hemispheres caused permanent blindness. (Munk found that hemianopia was produced by destruction of the occipital lobe, and Ferrier found that destruction of the occipital lobe and angular gyrus caused not only transient amblyopia of the opposite eye, but also permanent hemianopia. He failed to obtain any affection of vision by extirpation of the occipital lobe only, even on both sides. Nevertheless, human pathology has fully confirmed the correctness of Munk's conclusion.)

Stimulation of the anterior part of the uncinate convolution caused movement of the nostril suggestive of a sensation of smell on the same side. Isolated extirpation of these regions is impossible, but the destruction of it, together with other parts, always caused loss of smell.

Stimulation of the superior temporo-sphenoidal convolution caused a movement suggestive of an auditory sensation on the opposite side, and destruction of this convolution caused deafness on the opposite side.

Destruction of the hippocampal region (gyrus hippocampi and hippocampus) seemed to cause partial loss of sensibility to touch and pain on the opposite side. (Horsley and Schäfer found that extensive lesions of the gyrus fornicatus had a similar effect. Munk's experiments point to the outer surface, and especially the central region, as that in which cutaneous sensibility is chiefly represented.)

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* Horsley and Beevor (*Proc. Roy. Soc., 1886*) found that the representation of the arm is as follows: In the highest part of the arm region of the two central convolutions, opposite the upper frontal sulcus, the shoulder movements are represented; next, below, come the movements of the elbow behind and of the wrist in front, while in the lowest part the movements of the fingers are represented in front, and of the thumb behind. But this is only the maximum representation of the several parts, in which the special movement is produced by the weakest currents. There is scarcely any part in which movements of the elbow and wrist are not produced, or in which they are produced alone. Hence the experimenters conclude that the representation of the movement of these parts is subordinate to that of the shoulder on the one hand and of the digits on the other.

CORTICAL CENTRES IN THE HUMAN BRAIN.—The evidence we possess regarding the cortical centres in the human brain is derived solely from the comparison of the effects of disease, observed during life, with its position ascertained after death. It is probable, however, that brain surgery may indirectly increase considerably the facts at our disposal. The evidence at present available shows that there is a general correspondence between the cortical centres of man and those of the monkey. At the same time our knowledge of many of the centres of the human brain is not yet precise, and we are not justified in assuming a precision at present unwarranted by the actual evidence.

The evidence of the position of these centres is afforded by cases in which small lesions have caused definite loss of function, motor or sensory, or definite excitation of function. The irritation symptoms are of chief significance in the case of the motor function, and are constituted by local spasm, or more commonly by convulsion beginning in or limited to one part. But such convulsion may be caused by disease adjacent to, as well as by that which is in, a cortical centre, and therefore such cases afford less precise and certain evidence than do those in which there is local destruction of tissue and local paralysis.

It is only under certain conditions, however, that even destroying lesions can be taken as evidence of functional localization. Those conditions have been rightly insisted on by Nothnagel in his work* on this subject, a work which is a model of scientific method. It is only the lasting symptoms which can be regarded as related to the damaged region of the brain, because an acute lesion frequently causes, for a time, symptoms of much wider range than strictly correspond to the destruction. Such wide symptoms are due to pressure, secondary vascular disturbance, or irritative inhibition. Hence sufficient time must be allowed to elapse for these "indirect" effects to pass away before any inference can be drawn; that is, only such symptoms as have lasted for some weeks can be regarded as having real significance, and cases of shorter course are of small value.

Negative evidence is also important—the occurrence of lesions in certain parts without the production of certain symptoms. But another condition must be observed in the reception of this evidence. The nerve structures are remarkably tolerant of morbid processes that develop gradually. Tumors or abscesses may form in the position of structures that are known to have a certain function, and there may be no symptoms of disturbance of that function, or such symptoms may be slight, even when the structures are apparently destroyed by the morbid processes. In these cases the nerve elements are displaced and not destroyed; any damage they have suffered has been so slowly produced that it has not deranged their function. Hence the negative evidence afforded by such cases is of almost no value. On the other hand, these, and other lesions which cause pressure or irritation, often produce symptoms of much wider extent than corresponds to the position of the lesion or to its direct influence,—effects which are analogous in nature to the "indirect" initial consequences of an acute lesion. Hence the positive evidence afforded by irritating and compressing lesions can only be accepted with reserve. Neglect of these considerations has deprived of almost all value one of the most laborious investigations into this problem —that of Exner.*

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* "Topische Diag. der Gehirnkrankheiten," Berlin, 1879.

There are some functions of the cortex that elude localization for another reason, because their loss is quickly compensated by the other hemisphere. It appears that one-sided movements are represented in both hemispheres, and can be excited from either, in proportion as they are habitually associated on the two sides. This law, first stated by Broadbent, is of extreme importance, and we shall have to return to it many times. The above statement of it is perhaps only part of the truth, but it enables us to understand the absence of any evidence as to the position of the centres for certain movements. Before the indirect effects of a lesion have passed away, and the persistent symptoms can be admitted "into court," the loss of the movements referred to has passed away, because the other hemisphere has supplied the lost function. It is so, for instance, in the case of lateral movement of the head and eyes, the movement of the muscles of mastication, and many movements of the trunk.

The evidence at present available consists of a large number of facts, ascertained by various observers, many of which have been collected and carefully compared by Nothnagel, Charcot and Pitres,† and Allen Starr.‡

Motor Centres.—There is conclusive evidence that the two ascending convolutions have the same special relation to voluntary motion as in the monkey. It is also certain that the paracentral lobule on the medial aspect of these convolutions has a similar function, and that the motor region extends through part at least of the superior parietal lobule, and perhaps on to the root of the highest frontal convolution. Destructive lesions in these parts cause paralysis on the opposite side, while irritating disease causes convulsion of corresponding situation. Lesions elsewhere in the cortex cause no persistent paralysis. There is at present no direct evidence that the motor region in man extends beyond the ascending frontal, with the possible exception of the root of the first frontal.

It is from this motor region that the fibres arise which, as we shall see, pass down through the white substance into the anterior pyramids of the medulla, and the pyramidal tracts of the spinal cord. Through these fibres the nerve impulses pass that ultimately excite the muscles. Lesions of the cortex in this region cause a descending degeneration of these pyramidal fibres.

We can distinguish in these convolutions regions related to the leg, arm, face and tongue, which have the same relative position as in animals.

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* "Untersuch. u. d. Localisation der Grosshirnrinde," Wien, 1881.

† In a valuable series of papers in the *Revue de Méd.*, *1883*.

‡ In critical and analytical collections of cases in the *American Journal of Medical Science*, and other American periodicals, during the last three or four years.

The centre for the leg occupies the highest part of the motor region, namely, the ascending frontal and parietal convolutions, adjacent to the longitudinal fissure. We do not at present know how far back it extends in the superior parietal lobule, or how far outward it extends on the ascending frontal. It does not seem to extend further than the upper frontal sulcus, perhaps not so far. There may be individual variations in the area occupied by the leg centre. It certainly occupies also the paracentral lobule on the inner surface. We do not know how far the representation of separate movements corresponds to the arrangement in animals, but in many cases of convulsion beginning in the foot, the disease has been in or near the junction of the highest frontal and ascending frontal convolutions. Moreover, evidence of the representation of movements of the great toe in front of the highest part of the fissure of Rolando is shown by a case in which convulsions beginning in the toe were due to a cicatrix in this situation, and the removal of the part by Horsley left paralysis of the toe only.*

FIG. 179.

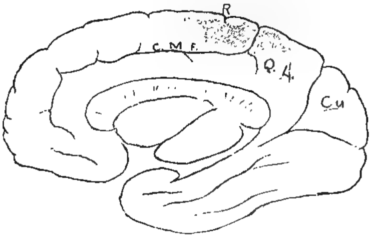
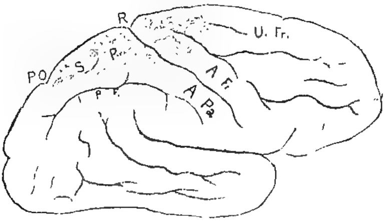
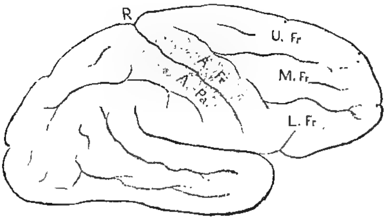


FIG. 180.



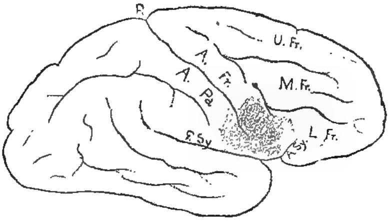
Position of the cortical centre or the leg. It is doubtful, however, whether the centre extends so far back in the superior parietal lobule (S P), as is here represented on the outer surface.

FIG. 181.



Position of the arm.

FIG. 182.



Position of the centre for the face and tongue.

The arm centre (Fig. 181) appears to occupy the middle third of these convolutions, but extends higher up the ascending frontal than the ascending parietal, perhaps reaching almost to the edge of the hemisphere. A small lesion very near the longitudinal fissure has paralyzed the arm. It is probable that this centre overlaps that for the leg.

The centre for the movement of the face (Fig. 182) lies in the lower third of the ascending frontal. It is probable, but not yet proved, that it extends on to the ascending parietal. The centre for the movement of the angles of the mouth lies opposite the fissure between the middle and lower frontal convolutions.* The lips and tongue are apparently represented together in the lowest part of the ascending frontal and perhaps in the adjacent root of the third frontal. The orbicularis oris and the transverse fibres of the tongue habitually act together; hence we cannot separate the centres for the face and tongue.†

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* Horsley, *Int. Journal of Med. Science*, April, 1887.

We have no evidence at present as to the position of the centre for the movement of the head and eyes (said to occupy, in the monkey, parts of the first and second frontal), nor whether the centre for the trunk muscles has the position assigned to it by Horsley and Schäfer on the inner surface of the hemisphere (see p. 456). The reason for this uncertainty has been already mentioned; compensation occurs so readily that the loss of movement quickly passes away.‡ It has been thought that there is a centre for the movement of the upper eyelid in the lower parietal lobule (Landouzy and Grasset), but the evidence of this is at present inconclusive.

There has been much discussion regarding the precise nature of these centres, and opinion is still divided on the matter. It is certain, however, that movements are produced by their stimulation in man as well as in animals.§ It is certain, also, that the fibres which conduct motor impulses to the cord spring from them, and pass directly downward. Hence it is difficult to believe that any considerable amount of error can be involved in designating the region "motor;" especially from the standpoint of practical medicine. We need not, therefore, conceive that these parts subserve no other function (we shall presently see reason for believing that they have sensory as well as motor functions). It is instructive, in this connection, to note that in this part are found the largest ganglion cells met with in the cortex, cells comparable to, though exceeding in size, the certainly motor cells of the anterior cornu of the spinal cord.||

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* See the conclusive case of Dr. Berkeley, of Baltimore, figured in the chapter on facial spasm.

† It is probable that the movements of the jaw are also represented in this region. In the course of an operation on man, Horsley found that the stimulation of the ascending frontal, at the junction of the middle and upper third of the facial region, caused a lateral movement of the jaw, as well as of the angle of the mouth.

‡ It is in the highest degree probable that the position of the centres corresponds to that in the monkey, and this is supported by an interesting case recorded by Horsley (*Int. Jour. of Med. Science*, April, 1887), which is not, however, quite conclusive.

§ First demonstrated by Bartholow, and since abundantly confirmed in the course of surgical procedure.

|| A theory of the nature of the so-called motor centres, which has received wide acceptance in Germany, is that of Munk. This theory regards them as essentially sensory; the movements which result from their activity are due to the fact that the nerve processes in them represent "conceptions of movements" composed of the cutaneous and muscular sensations and sensations of active innervation. These are regarded as essentially sensory conceptions,

Sensory Centres.—Our knowledge of the position of the centres for sensation of the limbs and trunk is far less precise than is that of the motor centres. According to Flechsig the fibres of the sensory path of the internal capsule pass toward the outer surface of the cortex, toward the region which, roughly speaking, lies beneath the parietal bone, *i. e.*, the central convolutions and the parietal lobe. The meagre facts of pathology, as far as they go, are in harmony with this view. One case is shown in Fig. 185. In another case complete left-sided loss of sensibility, including the eye and other special senses, resulted from a layer of softening at and just beneath the surface of the greater part of the convexity of the right hemisphere, the medial aspect and internal capsule being unaffected.* The view that the central convolutions have some sensory function, as well as a motor function, is supported by the facts that there is often slight blunting of sensibility on the extremity of a limb paralyzed by disease in this region, and that in convulsions due to irritating lesions in this situation, a sensory aura very often precedes the motor spasm. It is quite possible that the sensory region extends also to the medial surface of the hemisphere, just as does the motor region, but all the facts hitherto observed are opposed to the inference drawn by Ferrier from the experiments on animals, that any part of the medial surface has an exclusive or even a preponderant relation to cutaneous sensibility. One reason why we have so little evidence of the seat of this function is that extensive compensation is possible. Thus, a unilateral lesion in childhood, however extensive, scarcely ever causes permanent loss of sensation.

Smell.—The indication of experiment is that there is a centre for smell at the anterior extremity of the uncinate convolution on the medial surface of the temporal lobe, related to the olfactory nerve of the same side. This is supported, as regards man, by the facts that some fibres of the olfactory nerve can be traced toward this region, and that olfactory symptoms have been observed in a few cases of disease of this part. Thus, epileptic fits beginning with an olfactory aura were associated in one case with softening in this region.† But it is probable that other fibres, or fibres from this centre, cross the middle line and go to some part of the cortex of the opposite hemisphere, since there are cases in which organic disease of one hemisphere has caused loss of the sense of smell, in addition to that of the other special senses, on the opposite side.‡

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and hence Munk terms this region the "Sense-sphere" (Fuhlsphäre). An objection to this view is that the "sense of innervation," one element of the threefold "conception of movement," is a sense of something which is not itself sensory, and which is commonly termed motor, and that destruction of this region causes loss of this "motor" function out of all proportion to any demonstrable sensory loss.

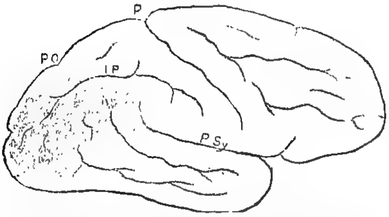
* Demange, *Revue de Méd.*, May, 1883, p. 391. A collection of clinical facts pointing to the parietal and central regions as the chief sensory region, is given by Allen Starr, *Journal of Nervous and Mental Diseases, 1884*, p. 327.

† McLane Hamilton, *New York Med. Journal*, June, 1882.

‡ Such a case, with autopsy, showing softening of the hinder part of the internal capsule, is recorded by Féré, *Arch. de Neurologie, 1885*. I have seen several cases during life in which smell was implicated.

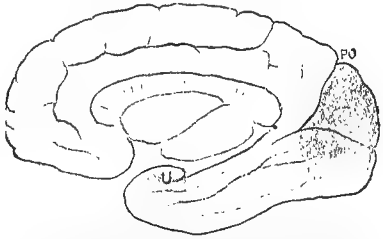
Vision.—Numerous observations have established beyond question the fact that hemianopia results from disease of the occipital lobe, which thus constitutes a centre for the fibres from the same-named half of each retina, and receives impressions from the opposite half of each field of vision. The impressions from the retinæ reach it by the optic tract, and probably by the optic thalamus and by fibres from the thalamus through the white substance of the occipital lobe. Fuller details of this path are given in the account of the origin of the optic nerves. We do not know the exact position of the visual centres in the occipital lobe. Hemianopia has resulted from disease of the apex of the lobe, the outer surface and the medial surface, but in some cases of partial lesion the white fibres of the optic path may have been involved. Complete hemianopia has most frequently been produced by disease of the apex of the lobe and especially of the cuneus. Munk believes that, in animals, there is a serial surface representation of the half-field in the occipital cortex, the anterior half corresponding to the upper quadrant and the posterior half to the lower quadrant. Since small cortical lesions cause incomplete hemianopia, it is probable that, in man also, there is such a projection of the field as Munk describes, but it is not probable that the projection occupies the whole occipital region. There must be a representation of the color fields in the cortex, distinct from that for light and objects, since color hemianopia has been met with as an isolated symptom. (See Diseases of the Optic Nerve.) Wilbrand* assumes that all impressions are conducted first to the apical region, because disease there causes total loss, and that there is a re-representation of the color half-field in front of this.

FIG. 183.



Cortical visual centres on the outer surface of the hemisphere. The darker shading indicates the region of the half-vision centre (the precise limitation of which is not yet known); the lighter shading is that of the supposed higher visual centre.

FIG. 184.



Inner aspect of the right hemisphere. Probable position of the visual centre in the occipital lobe and of the olfactory centre in the uncinate gyrus (U).

But disease sometimes causes, not hemianopia, but "crossed amblyopia," *i. e.*, dimness of sight in the opposite eye, generally with concentric diminution of the field.† The theory which best explains the fact is that on the outer surface, in front of the occipital lobe, there is a higher visual centre in which the half-fields are combined, and the whole opposite field is represented. Such a centre, in animals, is localized by Ferrier in the angular gyrus (in which term he includes the extremity of the supra-marginal convolution). Pathological evidence in man points to some part of the same region as the seat of this centre. In the very few post-mortem examinations in cases with "crossed amblyopia" the posterior and inferior part of the parietal lobe, *i. e.*, the angular gyrus, in its widest sense, has been involved in the disease. In the case mentioned on page 461, recorded by Demange, the loss of sight of the left eye was an early symptom, and the softening of this region appeared older than that elsewhere. Another instructive case, recorded by Dr. Sharkey, is shown in Figs. 185 and 186. It would seem that the field of the same side is also represented in this centre, since the amblyopia in the opposite eye is usually accompanied by a slight restriction of the field of the eye on the same side. Thus the centre must be assumed to represent the whole of both fields, but that of the opposite side in a far higher degree than the field on the same side. This assumption enables us to understand another curious fact, viz.: that the "crossed amblyopia" generally lessens after a time (while hemianopia is usually persistent). If the higher centre in each hemisphere is connected with both retinæ, it is conceivable that the loss caused by disease of one hemisphere may be compensated by the function of the other hemisphere. Hence we can understand that atrophy of this region, congenital or dating from early life, may be unaccompanied by any recog-

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* *Ophth. Beit. zur Diag. d. Gehirnkr., 1884.*

† I have seen several such cases, and others have been recorded, some by Ferrier, *Brain*, vol. iii, p. 456. Of course, all ocular causes of amblyopia are excluded.

FIG. 185.

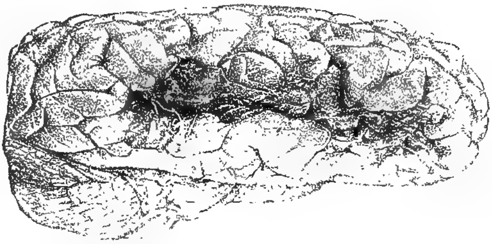


FIG. 186.

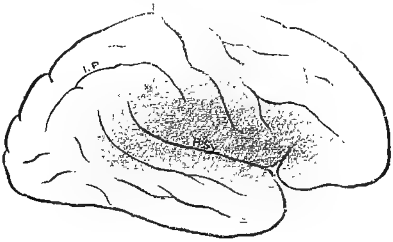
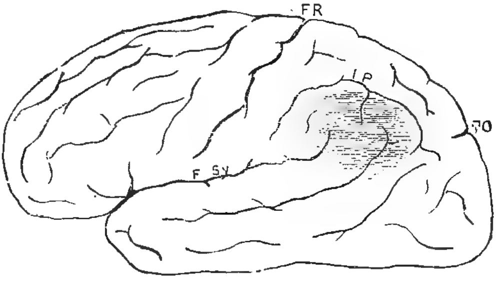


Fig. 185, aspect of right hemisphere, and Fig. 186, probable area of the lesion, in a case of embolism of the right middle cerebral artery (Sharkey, *Med.-Chir. Trans.*, *1884*, p. 265). The immediate effect of the lesion was left hemiplegia and hemianæsthesia, with almost complete blindness of the left eye, and loss of hearing and taste on the left side. Four weeks after the illness there was marked improvement in the special senses, and, a little later, in the hemianæsthesia. Six and a half weeks after the onset the special senses were normal, the leg had regained much power, and a fortnight later sensation was normal. The paralysis of the arm continued until death, seven years later.

FIG. 187.



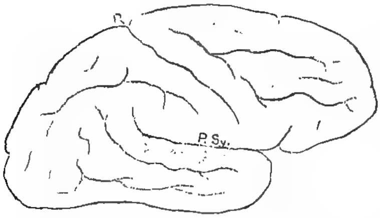
Position of lesion in the left hemisphere (angular gyrus) which caused complete mind blindness during the short time the patient lived. (*Chauffard*, *Rev. de Méd.*, *1881*, p. 940.)

nized loss of sight.* The visual path will be considered in connection with the optic nerves.

Cortical lesions in dogs that do not cause loss of sight may abolish or impair the power of recognizing the nature of seen objects, although they can be recognized at once when some other sense is employed; the condition has been termed mind blindness (Munk †). The power thus lost seems to be subserved by structures in or near the higher visual centre, but the position of the lesion has not yet been ascertained in any case in which the loss was considerable and persistent. The loss, of course, includes the recognition of words, and the latter may occur without inability to recognize objects in general. Mind blindness was produced by the lesion indicated in Fig. 187, but this case, while it agrees with the probable localization, was too brief in duration to constitute actual proof of the seat of the disease on which the symptom depends. The subject is considered further in the sections on Affections of Speech, and Diseases of the Optic Nerves.

Auditory Centre.—Pathology, on the whole, supports the indication of experiment, which places the auditory centre in the posterior half of the first temporo-sphenoidal convolution (Fig. 188). This convolution has been found atrophied in cases of congenital deafness.‡ Destruction of this region has been accompanied by loss of hearing in the opposite ear, although the loss has not been permanent. An instance is the case figured on the last page (Fig. 185). In a case under my observation, of extensive tumor, in which the oldest part was beneath this convolution, convulsions commencing by an auditory aura, referred to the opposite ear, were a very early symptom. In another case a tumor in the position of the letters P Sy in Fig. 188 caused unilateral convulsions, preceded by a loud noise, as of machinery. The convulsion on each side would appear, therefore, to be related to the opposite auditory nerve. It is important to note that the loss is not permanent, and this may explain another fact, that, in many cases in which the symptoms lasted for some time, the absence of deafness has been noted during life, although this convolution was found destroyed after death.§ Hence, it would seem that perfect compensation is possible, presumably by the corresponding centre of the opposite side. The complete deafness present at first in the case shown at Fig. 185 passed away completely at the end of

FIG 188.



Position of the auditory centre in the first temporal convolution.

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* As in a curious case recorded by Dr. Sharkey (*Med.-Chir. Trans.*, vol. lxvi, 1883, p. 293.)

† *Deut. Med. Wochensch.*, *1877*, No. 13, and *Arch. f. Anat. u. Phys.*, *1878*, p. 162.

‡ Fletcher Beach and others.

§ As in a case of softening recorded by Ball, *New York Arch. of Med.*, *April, 1881*.

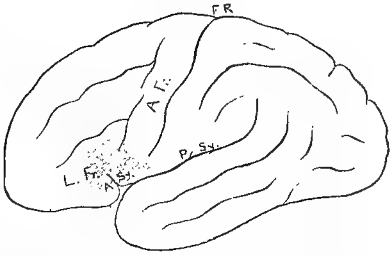
six weeks. Thus each auditory nerve must be structurally connected with both hemispheres, although only the connection with the opposite hemisphere is habitually in functional action.

A condition of "mind deafness" has apparently been caused in animals by destruction of the first temporal convolution (Munk). An analogous condition is met with not infrequently in man when this convolution on the left side is diseased, but it has been observed rather in connection with the perception of words than of sounds in general. (See Affections of Speech.)

Of the cortical representation of taste we know nothing. The perception of flavors is related to the olfactory, not to the gustatory centre.

Speech.—The relation of certain parts of the cortex to speech processes can be better understood when these processes have been considered in detail. The centres concerned in articulate speech are in the posterior extremity of the lowest frontal convolution, and the adjacent part of the ascending frontal, in the left hemisphere. They correspond, in part at least, to the centres for the movements (lips and tongue) by which articulation is effected. The perception of heard words is subserved by the centre for hearing in the first temporal, or by structures adjacent to it. There may be loss of the power of understanding heard words without deafness. The comprehension of seen words is a difficult and complex subject which will be considered in the chapter on affections of speech.

FIG. 189.



Position of the motor speech region in the left hemisphere.

Psychical Processes.—It is presumed that mental processes are subserved by those parts of the cortex that have no known motor or sensory function, and especially by the pre-frontal lobes. Many cases are on record in which considerable mental change was produced by extensive disease in this part, especially great when the disease was bilateral. Small lesions, however, may cause no symptoms, perhaps because there is considerable capacity for functional compensation. It would probably, however, be wrong to regard mental processes as exclusively related to the parts which are not known to have other functions, since the motor and sensory regions must also subserve mental operations.

THE CONNECTING TRACTS, CENTRAL GANGLIA, ETC.

The next subject of medical importance is the course of the fibres that unite the various parts, and establish a connection with the spinal cord. The course of these fibres has been only partially unraveled, and the difficulty of the task is increased by the fact that many of the tracts are interrupted in places by gray matter. This interruption arrests the progress of secondary degeneration, which is so great an aid in tracing the course of interlacing fibres.

The centrum ovale of the hemisphere consists of medullated nerve fibres, of which three classes have been distinguished, according to their course: (1) Fibres that pass between and connect different convolutions in the same hemisphere; (2) fibres that pass inward to the corpus callosum, and for the most part connect corresponding regions in the cortex of the two hemispheres.* (3) Fibres that pass to the central ganglia or crus cerebri. The last named converge from all parts of the cortex to the inner and lower region of the hemisphere, where the central ganglia lie and the crus leaves the brain. If traced upward from the crus and ganglia they radiate toward the cortex, and the radiation of the two hemispheres has been compared to a crown, and termed the "corona radiata." This expression is not now much used, but a limited application of the term is current and convenient: special groups of these fibres are termed "radiations," as the "pyramidal radiation," "optic radiation," etc., while the fibres that thus radiate are sometimes termed "coronal."

The crus cerebri, it will be remembered, enters the inner side of the hemisphere, spreading out beneath the optic thalamus and caudate nucleus, and its fibres ascend in a layer beneath these ganglia on the inner side and the lenticular nucleus on the outer side. This layer is termed the "internal capsule," because it bounds internally the lenticular nucleus. In a horizontal section through these ganglia (Fig. 190) it is seen that the anterior and posterior parts of the capsule have not quite the same direction; the anterior part, between the body of the caudate nucleus and the fore part of the lenticular nucleus, joins at an angle the posterior part between the optic thalamus and the hinder part of the lenticular nucleus. These are called the anterior and posterior "limbs" of the capsule, and the angle at which they join is called its "elbow" or "knee."† The extent of the lenticular nucleus from front to back corresponds to both the caudate nucleus and optic thalamus together, but the slender tail of the caudate nucleus extends as far back as the posterior extremity of

Fig. 190.

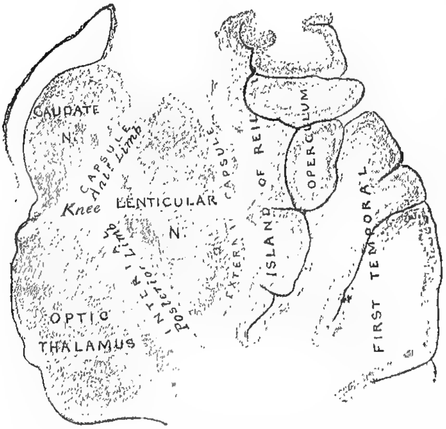


Diagram of horizontal section through the central ganglia and internal caspule.

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* This opinion has been confirmed by almost all modern investigations, with the exception of those of Dr. D. J. Hamilton (*Journ. of Anat.*, xix, p. 385), whose theory is scarcely to be reconciled with the certain facts of pathology, and whose evidence, from comparative anatomy, has been contested by Beevor (*Brain, 1886*).

† Such a bend (as in a pipe) is termed a "knee" in Germany, an "elbow" in this country. It is perhaps better to term the junction the "angle" of the capsule.

the lenticular nucleus, curving down into the roof of the descending cornu of the lateral ventricle. The two parts of the corpus striatum (caudate and lenticular nuclei) are connected at their anterior and posterior extremities by slender tracts of gray matter, which pass between the fibres of the capsule; elsewhere these fibres separate the two ganglia. In the lenticular nucleus, as seen in transverse section, three parts can be distinguished by a difference in tint: the inner, middle and outer, or first, second and third, the last being by far the largest (see p. 479).

A section through the crura cerebri above the pons (Fig. 191) shows them connected above by the corpora quadrigemina. The crus proper is separated into two parts, anterior (ventral) and posterior (dorsal),* by the "locus niger;" the lower or anterior is called the *crusta*, the upper or posterior the *tegmentum*. The crusta is also called the "pes" or "basis," and the former ("Fuss") is the term usually employed in Germany, but neither of these words lends itself to the English system of terminology, and it is better to use the older term "crusta" and its adjective "crustal." The tegmentum is much grayer than the crusta, because it contains many nerve cells mingled with the fibres, while the crusta consists almost exclusively of nerve fibres. The tegmentum varies in tint in different parts of its section. Near the middle line is a round or oval area, grayer than the rest, and often reddish gray. It is the "red nucleus," or "tegmental nucleus." To the outer side of the red nucleus, and just above the outer part of the locus niger, a paler curved band is seen, largest at its inner extremity. This is the *fillet*, an important tract of fibres, which extends up from the lowest part of the pons, and occupies there also the same relative position, the lowest part of the tegmentum. Above the red nucleus is a small triangular white area, near the middle line, the section of a compact bundle of "posterior longitudinal" or "posterior horizontal" fibres.

FIG. 191.

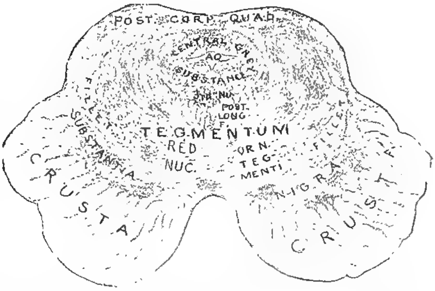

Diagram of section of crura cerebri. Aq, Aqueduct of Sylvius.

In a section through the middle of the pons (Fig. 197, p. 473) the distinction between the two parts, crustal and tegmental, is still indicated by the position of the fillet in the lowest layer of the tegmentum. The crustal portion below (*i. e.*, in front of) the fillet is here increased in bulk by the transverse fibres from the middle peduncle of the cerebellum and by much scattered gray matter. In the tegmentum the posterior longitudinal bundles are still seen near the middle line and close to the floor of the fourth ventricle. Between them and the fillet is an area consisting of interlacing bundles of fibres, transverse and longitudinal, the "reticular formation." In the tegmental part of the pons there are also various collections of gray matter from which certain cranial nerves take origin. These lie chiefly in the upper part, near the floor of the fourth ventricle, and will be subsequently described.

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* Although the direction of the pons and crura is nearer the horizontal than the vertical, it is convenient to retain the terms anterior and posterior in the same significance as in reference to the cord—the more so because the fibres from the crus again assume an ascending course when they enter the hemisphere.

Our knowledge of the course of the motor path is more complete than that of any other set of fibres. The relations of the spinal portion of this path have been described in the account of the anatomy of the spinal cord (p. 128). We have seen that it there occupies the two pyramidal tracts, anterior or direct, and lateral or crossed. The precision with which their limits are indicated by secondary degeneration enables us to trace their course with equal certainty in the brain. The degeneration has revealed also the remarkable fact that the pyramidal fibres extend from the central convolutions to the spinal cord without any interruption by gray matter. Tracing their course up from the cord (Fig. 192) we find that each lateral tract crosses the middle line in the medulla, and, uniting with the anterior or direct tract, forms the "anterior pyramid;" from this the name is taken by which the fibres are known throughout their course. Entering the pons the two pyramids are covered by the superficial layer of transverse fibres, and divide into a series of bundles. Thus divided, they course up through the crustal portion of the pons, lying between the superficial and deep layers of transverse fibres and surrounded by much gray matter, with which, however, their fibres have no connection. Above the pons the bundles again unite, and in the crus cerebri the pyramidal fibres lie together, and occupy the middle two-fifths of the crusta, extending from the surface below almost, but not quite, to the substantia nigra above. Passing beneath the optic thalamus they ascend between it and the lenticular nucleus, as part of the internal capsule. They occupy the anterior two-thirds of its posterior limb, the hinder third containing the sensory path.* Above the lenticular nucleus they radiate through the white substance of the hemisphere to that part of the cortex in which experimental stimulation causes movement in the limbs, viz., the two central convolutions with the superior parietal lobule and para-

FIG. 192.

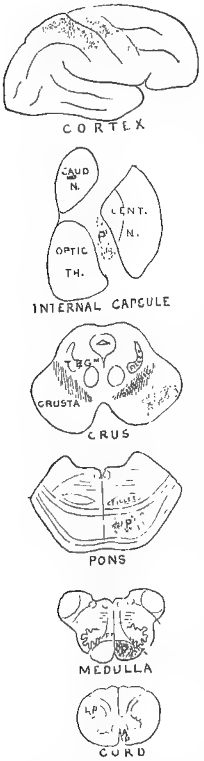

Diagram of the course of the pyramidal tract of the right hemisphere.

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* Perhaps not exclusively, however. A few fibres of the motor path have been found in the hinder third (Mannkopf, *Zeitsch. f. kl. Med.*, *1884*, Sup.). It is possible, however, that these fibres belong, not to the pyramidal tract, but to the fillet (q. v.).

central lobule. The motor impulses originating in these convolutions appear to be conducted directly to the gray matter of the spinal cord by these fibres, without the intervention of any nerve cells, either of the central ganglia or the pons. If these convolutions are destroyed, the fibres degenerate down to the lowest part of the cord. Some fibres from the motor cortex pass into the narrow "external capsule," outside the lenticular nucleus, into which their degeneration can be traced. Their further course is unknown.

The "motor" convolutions contain also centres for the movements exerted through some of the motor cranial nerves—movements of the jaw, face, and tongue. The fibres from these centres pass from the cortex with the pyramidal fibres. The centres for the face and tongue are lowest in the cortex (as we have seen), and their path lies correspondingly below that of the limbs in the centrum ovale. But the change from the vertical to an antero-posterior relation, in the internal capsule, brings the fibres for the face and tongue in front of those for the limbs, and they occupy the bend or angle of the capsule (Fig. 193). In the crusta they occupy a corresponding position on the inner (medial) side of the pyramidal fibres (Brissaud, Raymond and Artaud), the change in direction of the crus

FIG. 193.

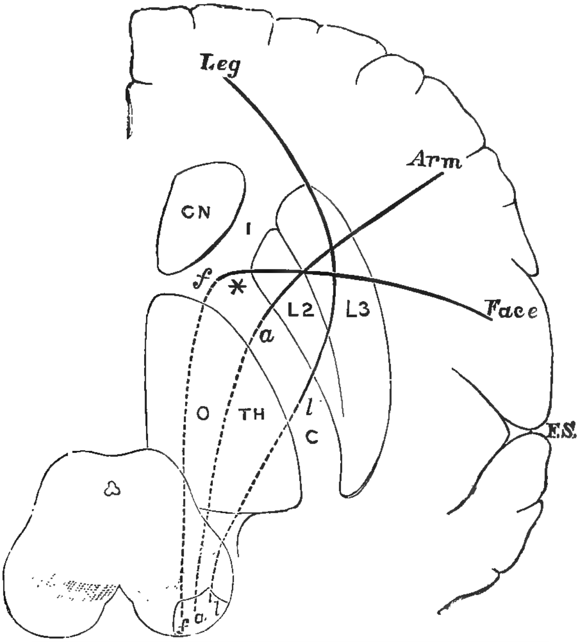

Diagram to show the relative position of the several motor tracts in their course from the cortex to the crus. The section through the convolutions is vertical; that through the internal capsule, I C, horizontal; that through the crus is again vertical; C N, caudate nucleus, O TH, optic thalamus, L 2 and L 3, the middle and outer parts of the lenticular nucleus; *f*, *a*, *l*, face, arm, and leg fibres. The words in italics indicate the corresponding cortical centres.

having brought to the inner side that which was anterior in the capsule and inferior in the cortex. It is probable that, in the pons, the fibres for the cranial nerves continue with the pyramidal fibres, lying on the inner side of the latter; hence the face and limbs may be paralyzed together by a small lesion in the upper part of the pons. When these fibres approach the level of their nuclei, they leave the neighborhood of the pyramidal tract, and, crossing the middle line, pass to the nucleus from which the cranial nerve takes origin.

Thus, of the white fibres that constitute the crusta, or lower half of the crus cerebri, less than half belong to the pyramidal tracts. What are the other crustal fibres? They lie partly on the outer side of the pyramidal fibres (T O C, Fig. 194), partly on the inner (medial) side (F C), partly above (C C), between the pyramidal fibres and the substantia nigra. Only those on the inner (medial) side of the pyramidal tract pass up into the internal capsule. The change in the direction of the crus brings these inner fibres in front of those of the pyramidal tract, and they form the anterior limb of the capsule (Fig. 195). They radiate to the cortex of the "pre-frontal lobe," *i. e.*, that part of the brain which lies in front of the central convolutions. From the crus they pass downward into the pons. They degenerate downward, and have been found degenerated in many cases of disease of the anterior part of the capsule. Hence we may assume that they conduct downward, but their degeneration stops in the pons, apparently because they end in the gray matter which is so abundant in the crustal portion. This gray matter also receives fibres from the middle cerebellar peduncles, and these fibres probably decussate in the middle line before entering the nerve cells (Lallemont). They seem to continue the path constituted by the inner

FIG. 194.

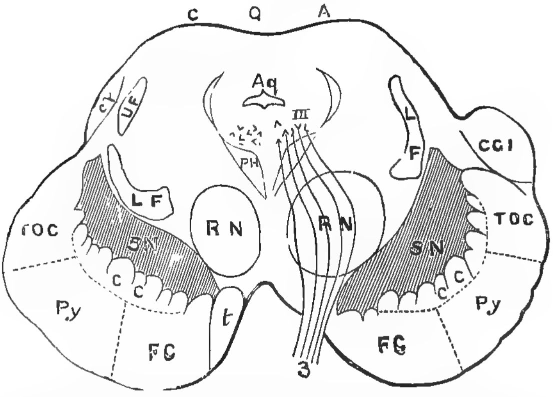

Diagram of section of the crus (modified from Wernicke). L F, U F, upper and lower fillet; C Q A, anterior corp. quad.; Aq, aqueduct; III, nucleus of third nerve (3); P H, posterior horizontal fibres; c p, brachium of the post. corp. quad.; R N, red nucleus; S N, substantia nigra; C G I, internal geniculate body; T O C, temporo-occipital cerebellar fibres; Py, pyramidal fibres; F C, fronto-cerebellar fibres; C C, caudate-cerebellar fibres; t, inner fibres of crusta to tegmentum.

crustal fibres. Thus each pre-frontal lobe is connected with the opposite cerebellar hemisphere, and chiefly with its lateral and posterior regions. When the cerebellum is congenitally absent, these fibres, the crustal gray matter of the pons, and the anterior limb of the internal capsule, are absent (Flechsig). In animals the gray matter atrophies if the cerebellum is excised.*

FIG. 195.

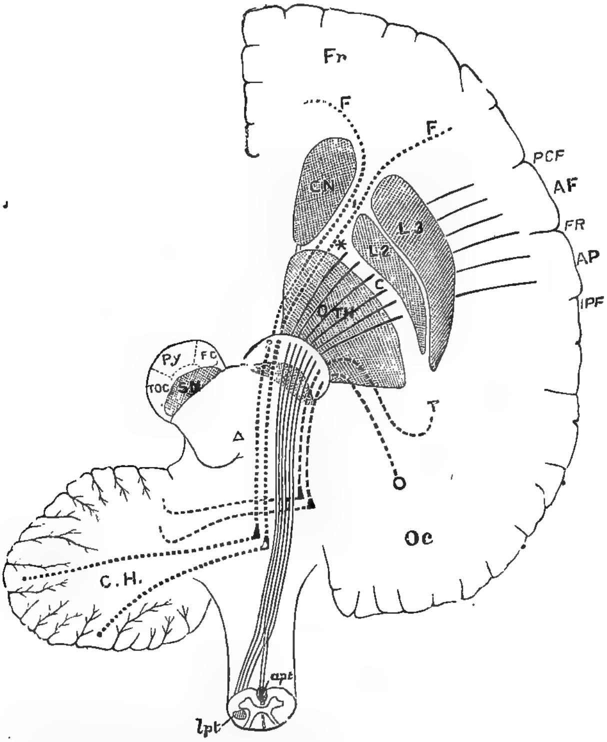

Diagram of the course of the motor tract as shown in a diagrammatic horizontal section through the cerebral hemisphere, pons, and medulla. Fr, frontal lobe; Oc, occipital lobe; A F, ascending frontal, and A P, ascending parietal, convolutions; *P C F*, pre-central fissure in front of the ascending frontal convolution; *I P F*, inter-parietal fissure. A section of the crura is lettered on the left side: S N, substantia nigra; Py, region occupied by the pyramidal fibres (motor tract), which on the right are shown as continuous lines, converging in the white substance of the hemisphere, to pass through the posterior limb of I C, the internal capsule (the elbow of which is shown at *)—through the crus and pons, and to divide in the medulla into the decussating lateral pyramidal tract (*lpt*) and the direct anterior pyramidal tract (*apt*).

One small bundle of fibres in the inner part of the crusta (t, Fig. 194), lying close to the medial surface, differs from the rest. As it descends, it passes backward into the tegmentum, and joins the fillet. Its further relations have not been traced.

The crustal fibres that lie outside the pyramidal tract† (T-O-C, Fig. 196) do not enter the internal capsule. They leave the other fibres when the crus enters the hemisphere, and pass, partly beneath the lenticular nucleus, partly between its posterior extremity and the outer geniculate body, to radiate toward the cortex of the occipital and temporal lobes. Downward they pass into the crustal portion of the pons, and then end in the gray matter, just as do the inner fibres, and are probably, in like manner, connected with the cerebellar hemisphere, chiefly. Flechsig thinks, with the upper surface, near the middle lobe. These fibres are also wanting when the cerebellum is absent. Flechsig thought that they do not degenerate downward, but it appears certain that they do, since they have been found degenerated in several cases of disease of the occipital and temporal cortex.* It was formerly believed that these fibres enter the internal capsule and constitute its posterior extremity, which is known to be the chief sensory path, and this statement is still made in some text-books. But Flechsig, by developmental investigations, has shown that this is an error, and his conclusions have been confirmed by pathology. As the fibres separate from the pyramidal tract, their place is taken by sensory fibres from the tegmentum, and thus the extremity of the capsule is formed.

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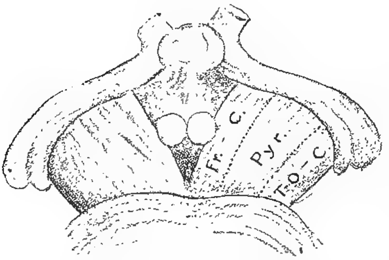
* Marchi, "*Rivist. sperim. de freniat.*," *1886*.

† Sometimes called "Türck's bundle," but to be carefully distinguished from "Türck's column" in the cord.

FIG. 196.



Crura cerebri; position of the fronto-cerebellar (Fr-C), pyramidal (Pyr), and temporo-occipital cerebellar fibres (T-O-C) on the surface.

The last part of the crusta to be considered is the thin layer of fibres which lies above the pyramidal tract, between it and the substantia nigra. According to Flechsig, these arise above, from the corpus striatum (caudate nucleus, and outer part of the lenticular nucleus), and they seem to end below, in the pons, probably in the crustal gray matter, as do the other fibres just described. They degenerate downward, and cannot be traced beyond the pons. They may thus connect the corpus striatum and cerebellum, in the same way as the inner group connects the pre-frontal lobe and cerebellum. Some of them may be connected with the gray matter of the substantia nigra.

Thus, of all the fibres of the spinal cord, the pyramidal tracts alone find a place in the crusta of the cerebral peduncle. It may be noted, moreover, that these are the only long spinal fibres that degenerate downward. The other fibres of the cord are either short fibres or degenerate upward, and are connected with the cerebellum or with the tegmentum of the crus. Their connections are at present very imperfectly known, but are of great importance. The sensory path must be sought in some of these fibres: it certainly lies in the tegmentum of the crus, and probably passes to the crus by the tegmental region of the pons.

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* Betcherew, *Cent. f. Nervenh., 1886*, p. 635. See also Fig. 228, p. 506.

In the tegmental region of the pons, as we have already seen, three chief groups of fibres may be distinguished (Figure 197), the small bundle of "posterior longitudinal fibres," the "fillet," and the extensive "reticular formation." We have to consider the connection of the remaining tracts of the cord with these structures and with the cerebellum, and also the relation of these tegmental fibres to the cerebral hemispheres.

FIG. 197.

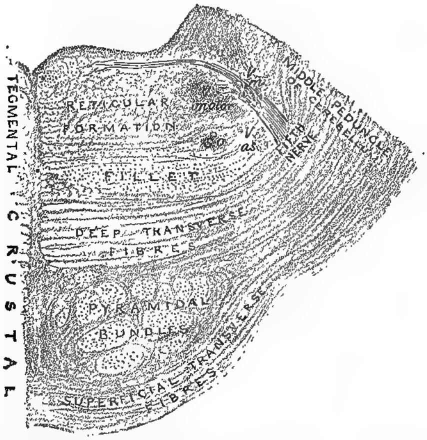

Diagram of one-half of a section through the middle of the pons. *S o*, superior olivary body; *V as*, ascending root, *Vm*, middle nucleus, *V motor*, motor nucleus of the fifth nerve.

Most of the white columns of the cord become occupied by gray matter when they reach the medulla oblongata. Their fibres probably end in the cells of this gray matter, from which other fibres proceed and continue the functional conducting path of the spinal tracts. Nerve cells arrest secondary degeneration; and hence most ascending degenerations of

FIG. 198.

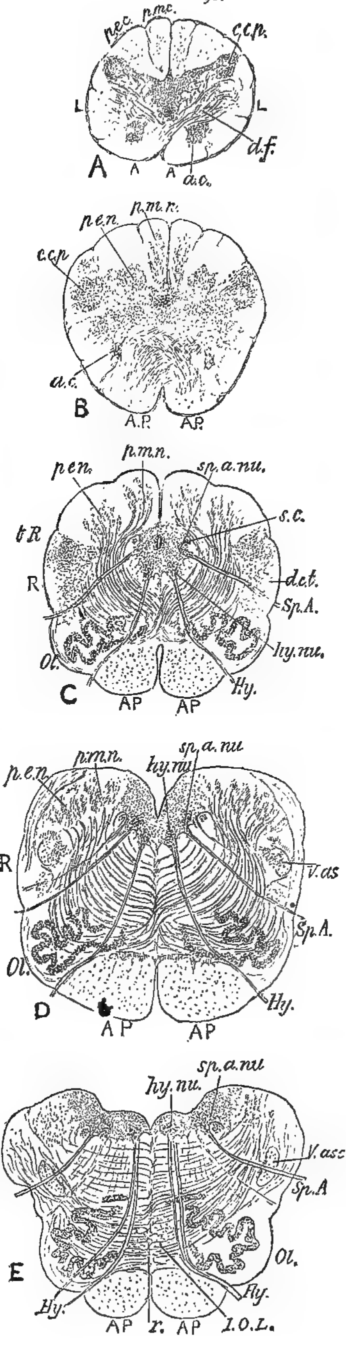

FIG. 198.—Diagrams of the structure of the medulla oblongata. A, lower, and B, upper part of decussation of the pyramids; C, at the lowest of the olivary bodies; D, at the apex, and E, at the middle of the calamus scriptorius. A, anterior, L, lateral column of cord; A. P., ant. pyramid; R, restiform body; a. c., ant. cornu; *t R*, tubercle of Rolando; *c, c, p.*, caput cornu posterioris; *d. c. t.*, direct cerebellar tract; *Hy*, hypoglossal nerve; *hy. nu.*, its nucleus; *Ol*, olivary body; *p. m. c.*, post. med. col.; *p. e. c.*, post. ext. col.; *p. m. n.*, post. med. nucleus; *p. e. n.*, post. ext. nucleus; *Sp. A.*, spinal accessory nerve, *sp. a. nu.*, its nucleus; *s. c.*, slender column; *V. as.*, ascending root of the fifth nerve.

the cord, as, for instance, that of the posterior median columns, stop in the medulla. This very much increases the difficulty of tracing the connection of these fibres.

One tract passes up to the cerebellum without interruption, the direct cerebellar tract. Its fibres pass in the restiform body, and reach the middle lobe of the cerebellum; they cross the middle line, and end in the opposite side of this lobe. They degenerate, and doubtless conduct, upward, and probably conduct centripetal impressions from the muscles of the lower part of the trunk to the coördinating mechanism in the cerebellum (see pp. 128 and 133). A few fibres from the direct cerebellar tract pass up into the pons.

The postero-median column (col. of Goll) is sometimes termed, at the medulla, the "funiculus gracilis," sometimes the "posterior pyramid." Its fibres, or at least most of them, end in the gray matter which occupies this region of the medulla, the "post-pyramidal nucleus," or, better, "postero-median nucleus" (Fig. 198, *p. m. n.*). Here ascending degeneration stops. The postero-external column ("funiculus cuneatus," "col. of Burdach") is also occupied by gray matter, the "postero-external nucleus" (Fig. 198, *p. e. n.*). From the cells of these nuclei other fibres continue the upward path. Those from the two nuclei are so blended that their distinction is scarcely possible, and the difficulty in tracing them is increased by the circumstance that their course is circuitous. Many fibres from both these nuclei course forward to the neighborhood of the olivary bodies. A large number of them cross the middle line, in front of the central canal, forming a decussation analogous to that of the pyramidal fibres, but higher up the medulla; it is of course wholly unconnected with the pyramids, but it has been rather unfortunately termed the "superior pyramidal decussation." These fibres turn upward between the two olivary bodies ("inter-olivary layer," Fig. 198, I. O. L.) and between them and the anterior pyramids. Some are probably connected with the cells of the olivary bodies. From the olivary bodies many fibres pass to the cerebellum by the restiform body, and these may establish a connection between the posterior columns and the cerebellum, perhaps by fibres which are not connected with the cells of the olivary body. The "inter-olivary layer," a little higher up, forms the commencement of the fillet, which has been already mentioned, and the fillet is thus connected with the posterior columns. Other fibres from these posterior nuclei pass to the reticular formation, which increases in size as the posterior columns lessen in size. Thus, the posterior columns are probably connected with the reticular formation, with the fillet, and with the cerebellum.

The olivary body bears a close resemblance in structure to the corpus dentatum of the cerebellum, and many fibres pass between the two. The connection is crossed, congenital atrophy or long-standing disease of one cerebellar hemisphere is associated with atrophy of the opposite olivary body, and experimental removal of one hemisphere in animals causes a similar atrophy.

Although the restiform body (inferior cerebellar peduncle) appears to be formed from the lateral column of the cord, its external relation does not indicate its real connections. Of the elements of the lateral column, only the direct cerebellar tract enters the restiform body. The pyramidal tract leaves the lateral column to cross to the opposite anterior pyramid, and in the remaining part of the lateral column, gray matter appears (just as it does in the posterior columns), the "lateral nucleus." This lies in front of a gray mass into which the caput cornu posterioris (Fig. 198, *c. c. p.*) has expanded, the "gray tubercle of Rolando" (Fig. 198, *t R*). The lateral nucleus higher up is continuous with the reticular formation, most of the fibres of which spring from the nucleus. Fibres also pass from the lateral nucleus and reticular formation to the restiform body and cerebellum, but the direction in which they conduct is uncertain. Flechsig thinks that they may conduct from the cerebellum to the reticular formation. Besides these two sets of fibres, the restiform body contains also the fibres from the opposite olivary body to the cerebellum, which we have already considered.

The antero-lateral ascending tract seems to pass to the reticular formation, but its fibres are most likely interrupted by the gray matter of the lateral nucleus. We have seen that the posterior columns are also connected with the reticular formation. It is probable that the upward sensory path passes, in part at least (and perhaps chiefly), by this structure, which can be traced through the pons into the tegmentum of the crus, in which the sensory path certainly lies.

The *fillet* or *lemniscus*, as we have seen, is a layer of fibres that lies between the crustal and tegmental portions of the pons, on the anterior (ventral) side of the reticular formation. It extends through the crus cerebri, where, however, it moves outward, and lies in the outer part of the tegmentum. There is still much difference of opinion regarding the connections of its fibres, in spite of the fact that these have been the subject of an immense amount of recent investigation.* It is clear, however, it contains several different sets of fibres. Below, the fillet arises, as already stated (p. 474), in the inter-olivary layer, formed of the arciform fibres which proceed from the nuclei of the opposite posterior columns, median and external; part of the fillet thus appears to be a continuation of the path of these columns, and is conjectured by Spitzka to conduct chiefly centripetal impressions from the muscles. Many of the fibres, however, degenerate downward; others seem to degenerate upward. In a case in which the fillet was destroyed on the left side of the pons by an old hemorrhage, Spitzka traced the descending degeneration through the inter-olivary layer to the opposite nuclei of the posterior columns. But in another case of focal lesion an extensive ascending degeneration of the same part of the fillet was found by Meyer.† A few fibres also seem to pass to the lateral column of the cord. Among the different sets of fibres in the fillet we may distinguish the following upward connections: (1) Many fibres pass by the red nucleus, and are associated with fibres from this and from the opposite cerebellar peduncle, in what is termed the "lenticular loop," a set of fibres which, beneath the optic thalamus, turn outward and pass transversely through the internal capsule, to end in the lenticular nucleus* (Fig. 199). (2) Some fibres pass, in the pons, into the reticular formation. (3) Some fibres go to the posterior corpus quadrigeminum. Some of these end below the superior olivary body. (4) Other fibres end in a collection of gray matter lying outside the junction of the two corpora quadrigemina, the "nucleus lemnisci" of Flechsig and Betcherew. (5) Fibres pass up to the posterior part of the internal capsule and radiate with these capsular fibres to the central and parietal cortex. Most of them are said to be connected with the posterior median nucleus. It appears probable, however, that they degenerate downward (Monakow, Spitzka).

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* The most important recent writings on the subject are those of Flechsig ("Plan des Menschlichen Gehirns"); Flechsig and Betcherew (abstract by Flechsig in the *Neurologisches Centralblatt, 1885*, p. 356); Monakow (*ib.*, p. 265); Wernicke ("Gehirnkrankheiten," Bd. i); and Spitzka (*New York Med. Record, 1884*, Nos. 15–18).

† *Arch. f. Psych.*, xvii, p. 439.

The *reticular formation* contains numerous longitudinal fibres, the downward connection of which, with the lateral column and posterior columns, has been already described. It also receives fibres from the nerve nuclei of the pons. At the upper part of the pons it rapidly lessens in size, and many of its fibres go to the upper corpus quadrigeminum,† and from this, again, fibres proceed to the posterior part of the internal capsule and tegmental radiation. The fibres that do not go to the corpus quadrigeminum pass upward to the optic thalamus and perhaps to the posterior part of the internal capsule and tegmental radiation; a few go to the gray matter lining the third ventricle.

The third group of longitudinal fibres in the tegmental portion of the pons is that termed the *posterior longitudinal fibres*, lying near the posterior surface and middle line (Figs. 194, 199, 209). This group contains fibres of different size. The finer are continuous below with some of those of the anterior column of the cord, and above pass to the central gray substance that lines the third ventricle (Flechsig). The coarser fibres extend only from the nucleus of the third and fourth nerves above, to the level of the nucleus of the sixth below, and connect these nuclei. Some of the fibres decussate, and they no doubt subserve the complex associated action of the eyeball muscles, their relation to which will be considered later.

In the lower part of the pons, above the olivary body, is a small body, with somewhat sinuous outline, the *superior olivary body*. It lies in the anterior part of the tegmentum, and its important connections have been traced by Betcherew.‡ According to him, fibres pass up from it to the fillet and central gray substance of the posterior quadrigemimal bodies; other fibres pass to the nucleus of the sixth nerve, the anterior auditory nucleus, the cerebellum (roof nucleus in the middle lobe), and to the lateral column of the spinal cord. This connection suggests that the body has important central functions, and it may be through it that an auditory impression causes a lateral movement of the eyes and head, the former through the nucleus of the sixth, the latter through the lateral column of the cord. The connection with the cerebellum may be one path by which disease or stimulation of the cerebellum influences ocular movements. The corpora quadrigemina are probably concerned in the movements of eyes, and the fibres from them to the superior olivary body belong to that part of the fillet which, according to Flechsig, degenerates and conducts downward.

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* In a case of absence of the cerebellum, examined by Flechsig, the red nucleus and fibres from the superior cerebellar peduncle were absent, and the fillet fibres in the lenticular loop were very distinct.

† A few of these reticular fibres, that lie close to the fillet, are by some authorities regarded as belonging to this structure.

‡ *Wratsch*, No. 32, 1885; *Cent. f. Nervenheilk.*, *1886*, p. 587.

Beneath the corpora quadrigemina the tegmentum receives a considerable accession of fibres from the passage into it of the superior cerebellar peduncle, the fibres of which come chiefly from the dentate nucleus. They may be connected, through this, with the fibres from the olivary body, and also with the cortex of the cerebellum. In the tegmentum these fibres cross the middle line and are connected with the opposite red nucleus, which is absent, with the fibres of this peduncle, in congenital absence of the cerebellum (Flechsig). Thus the red nucleus may be connected with the olivary body of the same side, by means of the opposite dentate nucleus. Proceeding upward from the red nucleus, the fibres divide into two groups; one passes forward and outward to the lenticular loop and thus to the lenticular nucleus (Fig. 199); the other courses backward and outward, partly into the basal part of the optic thalamus, partly into the posterior part of the

FIG. 199.

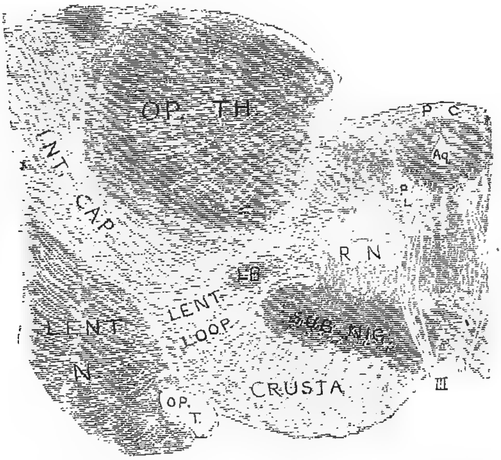

Diagram of a section through the crus, etc., in front of the corp. quad. P C, posterior commissure; Aq, aqueduct of Sylvius; P L, posterior longitudinal fibres; III, third nerve; L B, Luy's body; OP T, optic tract. (*Modified from Wernicke.*)

internal capsule and tegmental radiation. Those fibres which enter the thalamus perhaps merely pass through it to the capsule.

Thus the chief destination of the tegmentum, including the superior cerebellar peduncle, is twofold: the lenticular nucleus, and the cortex of the hemisphere by the tegmental radiation. Fibres go to each of these from the fillet and the cerebellar peduncle, and other fibres of the fillet, together with those of the reticular formation, ultimately reach the tegmental radiation. As before stated, the sensory path certainly lies in the tegmental radiation where this begins in the hindmost region of the internal capsule. It is also practically certain that most forms of sensation from the limbs pass through the tegmentum of the pons, and it is probable that the path is different for the different forms of sensibility. It may be assumed that no sensory impressions pass in the larger division of the fillet (which is connected above with the lenticular nucleus) since the fibres degenerate downward, nor by the posterior longitudinal bundles, which are not connected with the tegmental radiation. There remain, then, as possible paths only the upper smaller division of the fillet and the longitudinal fibres of the reticular formation. At the same time it is possible that some forms of sensibility, *e. g.*, visceral sensibility and muscular sensibility, may pass through the cerebellum and superior cerebellar peduncles.

The posterior third of the internal capsule contains not only the sensory path from the limbs, but also the optic path, and it also contains the paths for the other special senses; those for hearing and taste ascend to it from the pons in the tegmentum. How the path for smell reaches it is at present unknown. This is the "sensory crossway" of Charcot, in which a lesion causes hemianæsthesia, hemianopia, and loss of the other special senses, all on the side opposite to the lesion.

Central Ganglia.—The corpus striatum and optic thalamus present considerable differences in their connections, and these probably indicate fundamental differences in function, although we are still almost entirely in the dark as to the nature of their function.

The gray matter of which the *optic thalamus* is composed consists of fine nerve cells, among which narrow tracts of fibres pass. At the surface is a layer of white fibres, the "zonal stratum," and outside this again a very thin layer of gelatinous gray matter, continuous with that lining the third ventricle and surrounding the central canal of the cord. It is the union of this layer on the two thalami that constitutes the posterior commissure. The posterior extremity of the thalamus is distinguished as the "pulvinar," and between the extremities of the two thalami lie the anterior corpora quadrigemina. The gray matter of which the thalamus is composed is divided by a thin stratum of white fibres (internal medullary lamina) into an *external* and an *internal* nucleus, and the lamina divides anteriorly into two, which join the capsular layer, and with it enclose a small *anterior* nucleus. The most important connections of the thalamus are with the cortex, the optic tracts, and the tegmentum of the crus. It is connected with all parts of the cortex by fibres that pass from it to the internal capsule and corona radiata, of which they form a large part. They pass to all parts of the cortex; those from the pulvinar go to the occipital lobe and constitute part of the "optic radiation" of Gratiolet. It is probable that most of these fibres conduct from the thalamus; nevertheless, some fibres from the central convolutions to the thalamus have been found to degenerate downward.* Fibres also pass between the thalamus and lenticular nucleus, but we do not know whether they end in the lenticular nucleus, or merely pass through it to the cortex.

The connection of the thalamus and optic tract will be described in the account of the latter. The chief downward connection of the thalamus is with the tegmentum. This may be said to end beneath the posterior part of the thalamus, in what has been termed the "subthalamic region," in which are certain collections of gray matter. Many fibres pass to the thalamus from the superior cerebellar peduncle, going through the red nucleus. The extent of the connection of the thalamus with the fillet and the reticular formation is a subject on which there is much difference of opinion. Wernicke finds the chief origin of the fillet in the thalamus, while Flechsig could trace no connection between them.

Corpus Striatum.—The caudate nucleus is more uniform in structure than is the lenticular nucleus. In the latter two narrow laminæ of white fibres, almost vertical in direction, divide the gray substance into three zones, inner, middle, and outer (Fig. 199). In consequence of the shape of the nucleus, the outer zone is the most extensive, both in the antero-posterior and vertical direction; and it is also darker in tint than the other parts. Each part of the corpus striatum consists of nerve cells, large and small, and interlacing fibres. The fibres are far more numerous in the lenticular than in the caudate nucleus; but it is probable that many of these merely pass through the former, while the fibres that enter the caudate nucleus are connected with its cells. Moreover the outer part of the lenticular nucleus actually blends with the caudate nucleus in front by tracts of gray matter which pass through the internal capsule. To the outer side of the lenticular nucleus is the white layer of the "external capsule," and the gray lamina of the "claustrum."

FIG. 200.

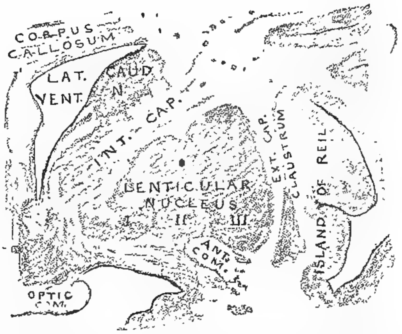

Diagram of a transverse section of the lenticular nucleus and internal capsule. I, II, III, indicate the three parts of the nucleus; ANT. COM., section of the bundle of fibres of the anterior commissure to the temporo-sphenoidal lobe.

It is doubtful whether the corpus striatum has any connection with the cortex, and the old hypothesis that its cells interrupt the fibres which conduct motor impulses seems to be altogether wrong. Meynert thought that many fibres pass from the caudate nucleus to the cortex; but the researches of

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* Mannkopf, *Zeitsch. f. kl. Med.*, *1884*, Bd. vii, Sup.

Wernicke and others make this connection very doubtful. It is extremely difficult to ascertain whether the lenticular nucleus is connected with the cortex, on account of the number of fibres that pass through it and do not end in it. Fibres, however, pass from the caudate nucleus to the internal capsule, and others pass to the crus through the lenticular nucleus. These caudate fibres seem to end, as we have seen, in the pons, and to be connected, through the pontine gray matter, with the fibres of the middle cerebellar peduncles. The lenticular nucleus differs from the caudate nucleus in being extensively connected with the tegmentum of the crus cerebri by many fibres, and especially by those of the lenticular loop. It is also connected, in a similar manner, with the superior cerebellar peduncle. The connection of the corpus striatum with the cerebellum is thus very considerable. When the cerebellum is absent, the corpus striatum is reduced to a third of its normal size. Flechsig and Wernicke consider that it must be regarded as a central organ, analogous to the cortex. The latter has also pointed out that—the lenticular nucleus being continuous with the gray matter of the anterior perforated spot, and this being continuous with the cortex—the gray matter of the corpus striatum may even be conceived to be homologous with that of the cortex. The "amygdala" of the temporo-sphenoidal lobe is an instance of the development of the cortical gray matter into the white substance to such an extent as to appear, in some sections, as if it were a central mass.

The *Corpora quadrigemina* are masses of gray matter mingled with fibres, which lie over the aqueduct of Sylvius and the tegmentum of the crus cerebri. A process or "brachium" extends forward from each, and contains white fibres, which proceed from a superficial layer immediately beneath the convex surface. The process from the anterior tubercle sends fibres to the external geniculate body, and, by the posterior portion of the internal capsule, to the tegmental or optic radiation. That of the posterior tubercle goes to the internal geniculate body, and is thus connected, according to Von Gudden, with the commissural fibres of the optic tract, which, at the optic chiasma, turn back, along the opposite optic tract, to the opposite internal geniculate body and corpora quadrigemina.

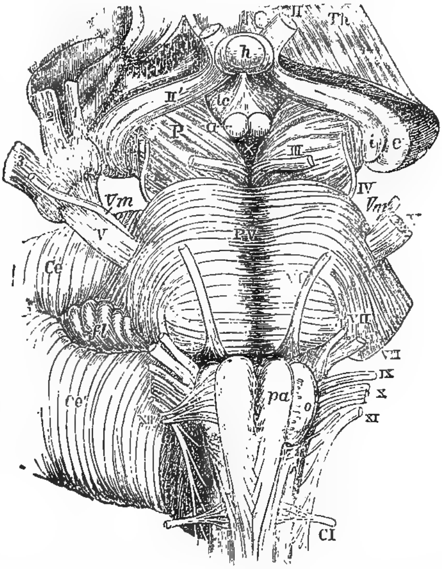
The quadrigeminal bodies receive many fibres from the tegmentum, chiefly from the reticulate formation, and, according to some authorities, from the fillet. From the gray matter of the corpora quadrigemina fibres pass directly to the subjacent nucleus of the third nerves (Meynert).

We have no direct evidence of the function of these ganglia. The results of experiment are difficult to interpret, and these bodies appear to have a higher relative importance in animals. In man they are scarcely ever the seat of isolated disease. It is not probable that they are directly concerned in the function of vision. Stimulation of either the anterior or posterior causes dilatation of the pupils, first of that on the side opposite to the tubercle stimulated; while further stimulation causes tonic and tetanic spasm, ending in opisthotonos, and stimulation of the posterior causes the animal to cry out (Ferrier and others). The relation to the optic nerve and oculo-motor nuclei, and likewise some of the results of experiment, strongly suggest that these bodies are concerned in the adjustment of ocular movements to visual impressions. The spasm produced by their stimulation must be of reflex origin, and suggests important connections; but of the nature of these we have at present no evidence.

ORIGIN OF THE CRANIAL NERVES.

The surface attachment of the cranial nerves is too well known to need repetition here. Their relative position is shown in the accompanying figure (Fig. 201), and is of considerable importance because it determines the grouping of nerve palsies in diseases of the base of the brain. The nerves that arise nearest the middle line, and are therefore most readily affected on both sides by a single lesion, are the third nerves. The two sixth nerves are also near together, and so are the two hypoglossal nerves, but the latter are seldom both affected by disease outside the medulla, because each passes outward, and they are separated by the prominent anterior pyramids. The two sixth nerves suffer, on the other hand, with great frequency, because they have a long course, not far apart, over the most prominent part of the pons, and they are readily affected by distant pressure. The nerves that are farthest apart, and are least frequently damaged together by a basal lesion, are the two fifth nerves.

FIG. 201.

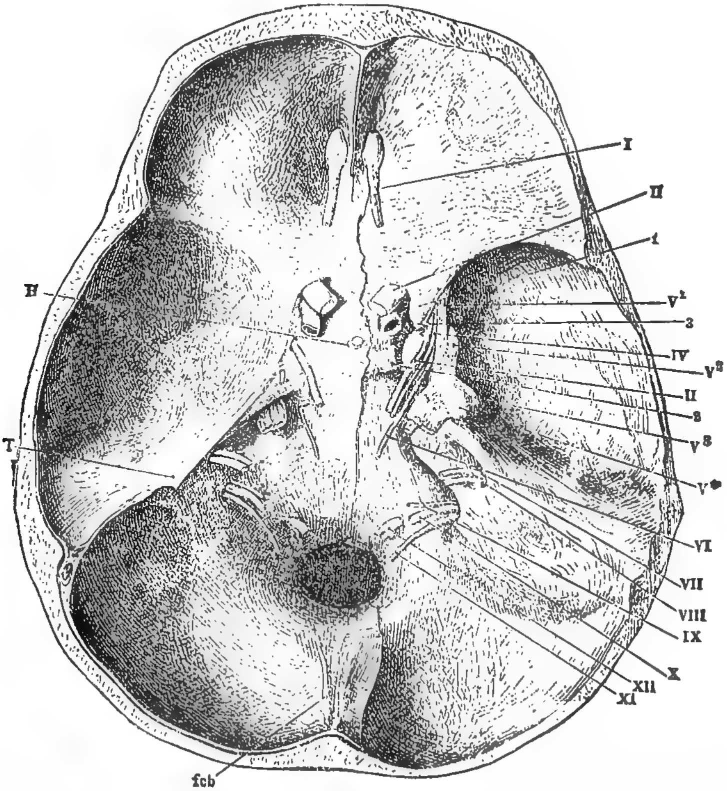


Origin of the cranial nerves (indicated by the Roman numerals). Th, thalamus; t c, tuber cinereum; h, pituitary body ("hypophysis cerebri"); P. peduncle; P V, pons Varolii; a, corpora albicantia; Ce, cerebellum; pa, anterior pyramid; o, olivary body. (*After Henle.*)

With regard to the relative position of the nerves, the fifth occupies a comparatively isolated position at its surface attachment: the sixth nerve is the nearest, and is most frequently associated with it in paralysis. As the fifth nerve enters the dura mater the third and fourth nerves are also near it (Fig. 202). The contiguity of the facial and auditory, and also that of the glosso-pharyngeal, vagus, and spinal accessory, are well known. It should be especially noted that the hypoglossal passes outward close to the spinal accessory, and hence these two nerves not unfrequently suffer together, and palsy of the tongue is then conjoined with that of the vocal cord on the same side.

The deep origin of these nerves (with the exception of the first two) is from a series of tracts of gray matter which are situated in front of the central cavity of the mesencephalon, and extend from the level of the anterior corpora quadrigemina above to that of the decussation of the pyram below. Above and below, these tracts lie near the middle line, but bene the floor of the fourth ventricle they extend almost from one side of the fl to the other. Most of the nuclei are situated between the reticular format and the floor of the ventricle, but some lie on the outer side of theretict formation. It is probable that the upward path from those nuclei that sensory in function lies in the reticular formation, while that from the br to the motor nuclei runs chiefly with the pyramidal tracts, as we have alrea

Fig. 202.



Base of the skull, showing the relative positions of the nerves (I–XII) as they enter the dura mater, which has been removed on the right side from the Gasserian ganglion, V*; f c b, cut edge of falx cerebelli; H, pedicle of pituitary body or hypophysis cerebri; T, cut edge of tentorium. (*After Henle.*)

seen. The tracts of gray matter which constitute these nuclei are, for t most part, of small transverse sectional area, but of considerable length.

The *hypoglossal* nucleus (Figs. 198, 204, 205) is situated close to the mi dle line, and extends from just above the decussation of the pyramids to tl calamus scriptorius of the fourth ventricle, at the point of which it li beneath the prominence that adjoins the raphe. Below it is situated front of the central canal. The nerve fibres pass from it through the inn part of the reticular formation and olivary body, and then curve outward between the latter and the anterior pyramid.

Accessory, vagus, and glosso-pharyngeal.—A group of nerve cells behind the hypoglossal nucleus, and behind the level of the canal, is the lower part of the nucleus of the *spinal accessory* nerve[*] (Fig. 198, *sp. a. nu.*) and from it the fibres course outward (*Sp. A*), through the lateral part of the medulla. As the canal opens out into the fourth ventricle, this nucleus passes to the outer side of the hypoglossal, and the fibres pass forward between the reticular formation and the restiform body. The nucleus is the lowest part of a tract of gray matter, the upper part of which lies beneath the floor (at the "ala cinerea") outside the eminentia teres, and gives origin, above the upper fibres of the spinal accessory, to the fibres of the pneumogastric, and then to those of the glosso-pharyngeal. The filaments of origin of these nerves form a continuous series, and it is scarcely possible to say where one ends and another begins. They all have a similar course to the surface, emerging beside the prominence of the restiform body, and all pass through a group of longitudinal fibres, more or less oval in transverse section, the ascending root of the fifth nerve (Fig. 204, V asc). No other nerve roots pass through this bundle. It may be noted that the fibres of the spinal accessory which arise from the medulla are those that innervate the muscles of the larynx, and are thus associated, in origin, with the fibres of the great respiratory nerve, the vagus.

FIG. 203.

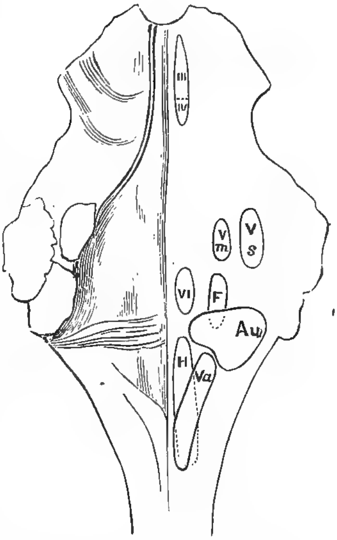


Diagram of the relative position of the nerve nuclei beneath the floor of the fourth ventricle. III, third nerve nucleus; IV, fourth; V, s, middle sensory nucleus of the fifth; V m, motor nucleus of fifth; VI, sixth; F, facial; Au, auditory; H, hypoglossal; V a, vago-accessorial nucleus, the upper part giving origin to the pneumogastric, the lower to the highest fibres of the spinal accessory. Where one nucleus lies beneath another its outline is indicated by a dotted line.

Moreover, in this relation, another connection of these nerves is important. To the other side of these nuclei, and of the fibres proceeding from them, is another bundle of longitudinal fibres, rounded in section and small in size. It is the "*slender column*" of Lockhart Clarke (Fig. 204). It extends upward as high as the highest part of the glosso-pharyngeal nucleus, while downward it passes into the deep part of the lateral column of the cord; its fibres have been traced as far as the middle of the cervical enlargement, and may extend lower still. Some fibres of the glosso-pharyngeal, pneumogastric, and spinal accessory nerves pass into it, or rather arise from it, and hence it has been termed their ascending root.[†] It is supposed to be connected with the process of respiration, to which the spinal accessory and vagus have such important relations. Hence Krause termed it the "respiratory column." Division of it on both sides is said to arrest all movements of respiration.*

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[*] The lower fibres of the nerve arise, not from the spinal accessory nucleus but from the anterior cornu of the upper cervical cord. These fibres supply the muscles of the neck.

[†] "Common ascending root of the lateral mixed system," by Meynert.

The nucleus of the *pneumogastric* and *glosso-pharyngeal* nerves is thus a tract of gray matter which lies beneath the outer half of the floor of the fourth ventricle, having the hypoglossal nucleus on the inner side, and on the outer side the restiform body below (where the pneumogastric arises, Fig. 204) and the lower part of the auditory nuclei above (where it gives origin to the glosso-pharyngeal, Fig. 205). Some fibres arise from groups of large nerve cells in the deeper part of this tract, and these are probably the motor fibres of the nerves; such cells are especially conspicuous in the glosso-pharyngeal portion (Fig. 205). Other fibres arise from smaller nerve cells in the more superficial gray part of the nucleus. Some fibres seem to come from the "slender column," as already stated, and others have been traced inward to the raphe. According to Meynert, some fibres of the pneumogastric pass to the cerebellum; the gastric functions of the nerve are readily deranged by disturbance of the equilibration, as in the vomiting of vertigo. Lastly, from the neighborhood of this nucleus, where the nerve enters it, fibres pass forward toward the deep part of the tegmentum (*x x*, Figs. 204 and 205). These have been thought to arise from a small collec-

FIG. 204.

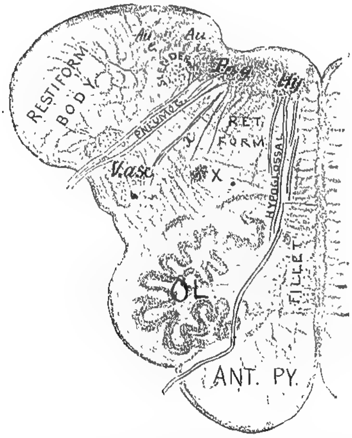


FIG. 205.

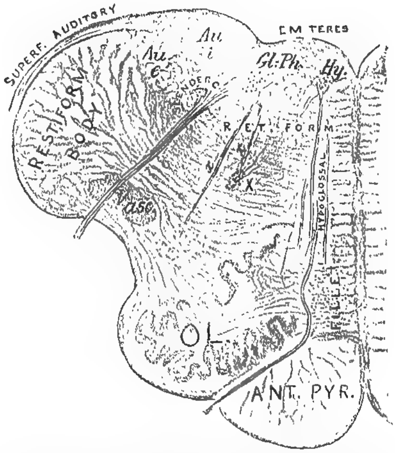


FIG. 204.—Origin of pneumogastric. Hy, hypoglossal nucleus; Png, chief nucleus of pneumogastric; Au, i e, lowest part of internal and (so-called) external auditory nuclei; V asc, ascending root of fifth; X, nucleus of unknown function in front of reticular formation (RET. FORM.); *x*, fibres passing forward from the neighborhood of the vagal nucleus.

FIG. 205.—Diagram of half-section of pons at the level of the glosso-pharyngeal nucleus, *Gl.Ph.*; *Hy*, hypoglossal nucleus. The other lettering is the same as in Fig. 204.

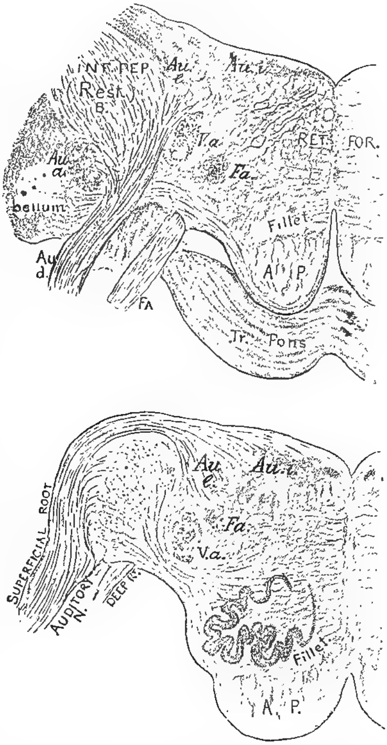
---

* Gierke. At the same time the division of this tract without injury to adjacent structures, on a living animal, is manifestly impossible. The connection with the vagus has been described by almost all investigators, but has lately been contested by Spitzka (*New York Med. Record, 1884*).

tion of large nerve cells (X), but most of them certainly pass by this nucleus* to nerve cells near the surface.

The deep origin of the *auditory* nerve is still involved in some uncertainty. Its attachment to the medulla (at the junction of this with the pons) is by two roots, one of which (Fig. 207) winds round the restiform body (inferior cerebellar peduncle), while the other (Fig. 206) passes into the substance of the medulla. The former is termed the superficial root, and the latter the deep root. The superficial root is a little posterior to the other, so that the two are not shown in the same section. The lowest superficial fibres are on a level with the glosso-pharyngeal nucleus (Fig. 205), but the nuclei of the auditory nerve extend still lower in the medulla, as far as the middle of the vagal nucleus (Fig. 204). The superficial root, in coursing round the medulla, contains some gray matter mingled with its fibres. Some of the fibres are continuous with the auditory striæ, which seem to pass to the middle line and opposite side of the medulla. Many of the fibres end in a thick tract of gray matter, which occupies the inner two-thirds of the floor of the fourth ventricle at this level, the "inner auditory nucleus" (*Au i*, Figs. 204–207), and is commonly regarded as the chief nucleus of the nerve. Outside this is a smaller area containing many groups of longitudinal fibres, which is commonly regarded as the external auditory nucleus (*Au e*); to it some fibres of the superficial root seem to go, but it is probable that they merely pass through it, and it is doubtful whether this external nucleus is really connected with

FIGS. 206, 207.



FIGS. 206 and 207.—Origin of auditory nerve. Fig. 206 shows the origin of the deep (Au. d.), Fig. 207 of the superficial root. *Au. a, i,* and *e*, anterior, internal and so-called external, nuclei; *Va*, ascending root of fifth; Fa, nucleus of facial nerve; A P, anterior pyramid; Tr Pons, transverse fibres of pons.

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* The nucleus has been termed the "anterior nucleus" of these nerves by some, by others the "nucleus ambiguus." It is not certain that the fibres that go toward it are root fibres. I believe they are connected with scattered nerve cells which lie among the arciform fibres, between the ascending root of the fifth, and the olivary body. I have many times traced the fibres to this part, and observed that these cells send a process in the direction of these fibres. The nucleus X is not, as might be imagined from its position, continuous with that of the facial nerve.

the auditory nerve.* As the nerve courses round the medulla many fibres curve inward into the restiform body; some may change their direction and join the restiform fibres, but others appear to pass transversely through the restiform body, and to be continuous with the transverse fibres of the tegmental portion of the pons. What their destination is we do not know. Meynert thinks that they may pass to the opposite restiform body and the cerebellum.

The deep root (Fig. 206) passes inward and upward on the inner side of the restiform body, at the level of the lowest fibres of the pons. It has been supposed to go to the external auditory nucleus above described. But many fibres curve outward into the restiform body and pass toward the middle lobe of the cerebellum. These are probably the fibres that come from the semicircular canals, and subserve, not auditory sensations, but impressions from the canals determined by the position and movement of the body. A few fibres may reach the internal auditory nucleus, while others turn outward before reaching the restiform body to a collection of gray matter which lies in front of the latter, the "anterior auditory nucleus" (*Au a*, Fig. 206). Lower down, this anterior nucleus lies in the angle between the two roots of the nerves.

Of the auditory path to the hemisphere we know only that it passes by the posterior part of the internal capsule. It probably passes in the most superficial layer of the tegmentum of the crus.† Meynert thinks that it passes through the cerebellum and not through the pons, and improbable as this may seem, it cannot be said that we have any facts at present inconsistent with the hypothesis.

Above the auditory striæ the most prominent part of the eminentia teres marks the position of the nucleus of the *sixth nerve* (Fig. 208), to which the fibres pass through the inner part of the reticular formation. They enter the nucleus on its inner side. The fibres of the *facial nerve* pass outside the reticular formation to the neighborhood of the sixth nucleus, where they form a loop and turn downward, most of them in a compact bundle around the upper extremity of the nucleus, but some through the nucleus itself. They then radiate downward, forward, and outward to a column of nerve cells (Fa, Figs. 206, 207), which lies above the outer part of the fillet, near the gray tract known as the "superior olivary body." There is a very close physiological connection between the muscles of the lip and tongue, and the facial nucleus extends down almost to the level of the hypoglossal nucleus, but the two are some distance apart, and we do not know whether the connection between the lips and tongue is subserved by fibres that pass between the two nuclei, or whether (as is possible) the nerve fibres for the lips actually arise from the hypoglossal nucleus. Many of the fibres of the facial nerve, turning downward at the loop, have a longitudinal course in the inner part of the reticular formation, and may readily reach the hypoglossal nucleus.*

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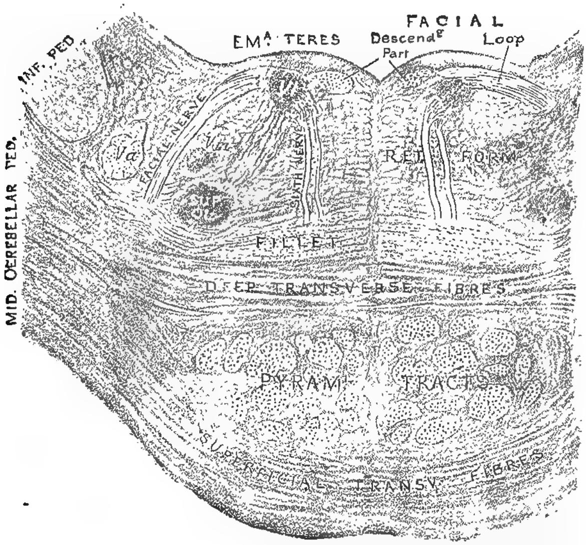
* Monakow found it atrophied after hemisection of the lower part of the medulla (*Arch. f. Psych.*, Bd. xiv). See also, on this subject (Onafrowicz, *ib.*, Bd. xvi). This nucleus has been also termed "Deiter's nucleus," as an indifferent designation, but the name has not come into general use, and it is marked, *Au e*, in the diagrams, in accordance with prevailing usage.

† See a case published by me, *Lancet*, March 15th, 1879, in which bilateral deafness was caused by a tumor of the corpora quadrigemina, damaging the superficial layer of the tegmentum on each side.

The origin of the *fifth* nerve (Fig. 209) is very extensive, its deep connection reaching from the level of the anterior quadrigeminal tubercle to the lowest part of the medulla. This is not surprising when we reflect that this nerve represents the sensory roots of all the motor cranial nerves, from the third to the hypoglossal. The two parts of the nerve pass backward and inward, through the outer part of the pons, or rather through the inner part of the middle peduncle of the cerebellum. The smaller, motor root is a little higher up than the other. The sensory root reaches the outer part of the tegmentum, beneath the outer edge of the floor of the fourth ventricle, and there some of its fibres terminate in small collections of gray matter, which are collectively termed the *middle nucleus,* or "chief nucleus." It is certainly not the chief origin of the nerve, for by far

FIG. 208.



Transverse section of the pons at the level of the nuclei of the sixth nerves (VI); RET. FORM., reticular formation; Vm, motor nucleus of fifth nerve; *Va*, its ascending root seen in section; SUP. OL., superior olivary body.

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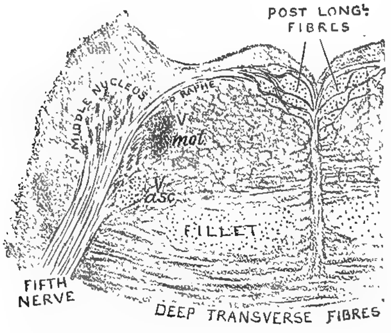
* The closeness of the central relation between the lips and tongue is shown both by their simultaneous affection in disease, such as labio-glossal paralysis, and by the fact that the transverse fibres of the tongue and the orbicularis oris can only contract together.

the largest number of the fibres turn downward and descend the pons and medulla, as a compact collection of bundles, lying in the same relative position, on the outer side of the tegmentum. This is termed the *ascending root.* It is a conspicuous object in all sections, and is shown in most of the preceding figures (*V asc.*). It becomes smaller as it passes down the medulla, but the ultimate origin of its fibres is not known. It terminates near the upper extremity of the gray tubercle of Rolando, into which the caput cornu posterioris of the cord develops, but its connection with the tubercle is not proved. (3) Some fibres of the nerve course inward, beneath the floor of the fourth ventricle; passing through the posterior horizontal fibres, they reach the raphe and probably go to the opposite half of the pons: their precise origin is not known. This is termed the *medial root.* It is easily mistaken for the loop of the facial nerve, which has a similar position a little lower down the pons. A few fibres are said to turn outward to the cerebellum. The *descending root* of the nerve consists of a series of bundles which pass upward* and lie outside the aqueduct of Sylvius, where they are arranged somewhat in the form of a crescent (Figs. 210 and 211).† They can be traced as far as the upper part of the corpora quadrigemina, but lessening in number, and they apparently rise from large rounded nerve cells that lie adjacent to the root. The root has commonly been regarded as one origin of the sensory portion of the nerve, but it has been stated (by Henle and Forel) that most of its fibres enter the motor division.‡ Almost all the fibres of the *motor root* end in a large collection of nerve cells (*V mot.*), which lies on the inner side of the sensory root, in the outer part of the tegmentum, and extends for a short distance down the pons.

FIG. 209:



Origin of fifth nerves. *V mot*, motor nucleus; *V asc.*, ascending root.

Each *fourth nerve*, arising from the valve of Vieussens (superior medullary velum), decussates within the substance of the valve with its fellow (Fig. 211). It is the only nerve that decussates between its surface origin and nucleus, with the exception of the optic. In its course in the velum, the nerve divides into a series of bundles, which curve forward and upward, round the central gray matter lining the aqueduct of Sylvius, to a collection of nerve cells at the hinder and outer part of the posterior longitudinal fibres (Fig. 210). The nucleus lies beneath the junction of the anterior and posterior quadrigeminal bodies, so that the fibres pass the whole length of the nates before reaching the nucleus. Hence, according to the position of the section, the bundles are divided transversely, obliquely, or longitudinally (see Figs. 210 and 211). Some fibres seem to pass to the posterior longitudinal bundles.

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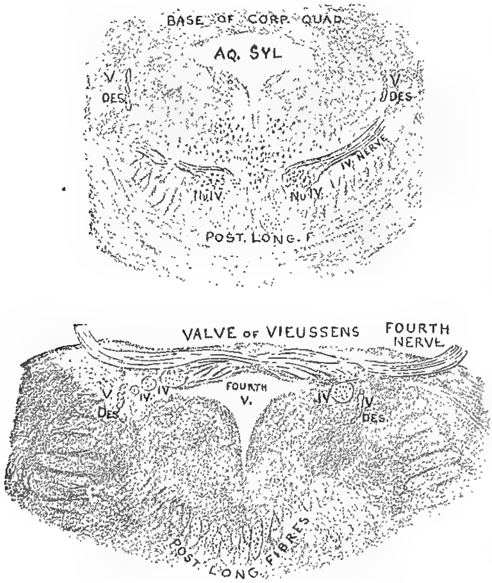
* The terms ascending and descending have been adopted, apparently under the influence of the word "root," but in tracing the fibres it is convenient to adopt the opposite mode of expression. It should be remembered, also, that the ascending root conducts downward.

† See also Fig. 194 (p. 470), in which the crescent on each side of the aqueduct represents the descending root.

‡ In spite of the high character of these authorities, the statement is so opposed to the opinion of other investigators that the point must be regarded as at present unsettled.

The fibres of the *third* nerve arise from a column of nerve cells that occupies the anterior part of the central gray matter below the aqueduct of Sylvius, beneath the anterior quadrigeminal bodies and the posterior commissure (Fig. 194, p. 470). The cells from which the fourth nerve arises are in the lowest part of this column. The fibres of origin of the third nerve pass forward, curving outward through the red nucleus, and then inward, to the side of the crus, where they emerge. On leaving the nucleus they pass through the posterior horizontal bundles, which, as we have seen (p. 476), are connected with this nucleus, and seem to establish an association between it and the sixth nucleus, through which the external and internal recti act together. The researches of Hensen and Voelcker show that the nucleus of the third nerve in dogs contains a series of centres, corresponding to the several functions subserved by the nerve, the most anterior being related to

Figs. 210, 211.



Figs. 210, nucleus, and 211, origin of fourth nerves. v. des., descending root of the fifth nerve; iv, iv, sections of fasciculi of the fourth nerves.

accommodation, the next to the reflex action of the iris, while the remainder of the nucleus, comprising its chief part, subserves the external ocular muscles in the following order, from before backward: Internal rectus, superior rectus, levator palpebræ superioris, inferior rectus, superior oblique. It is highly probable that the same arrangement obtains in man, and that the serial disposition of the filaments of origin of the nerve corresponds to the series of functions and of centres. It is to be noted, in this arrangement, that the centres are contiguous for those muscles that habitually act together, the superior rectus and levator, the inferior rectus and superior oblique. A much more complex arrangement subserves the association of the internal and external recti. When acting together, both these muscles are innervated through the nucleus of the sixth, disease of which causes loss of at least the associated action of the internal rectus. The path from the sixth nucleus to the origin of the internal rectus is doubtless by the posterior longitudinal fibres; and there is some reason to regard the superior olivary body as concerned in the action. The subject is considered more fully in the account of diseases of the oculo-motor nerves. Fibres pass to the third nuclei from the gray matter of the quadrigeminal bodies, and may subserve the relation between visual impressions and ocular movements.

The origin of the *optic nerves* is complex and still imperfectly understood. At the optic chiasma a partial decussation takes place, the fibres from the nasal half of each retina cross, and as the temporal half of each field of vision (subserved by the nasal half of the retina) is the larger, more fibres cross than pass uncrossed. Thus each optic tract contains the fibres from the same-named half of each retina, and conduct impressions from the other-named half of each field. Fibres also pass from one tract to the other by the posterior portion of the chiasma. The old opinion that fibres pass from one optic nerve to the other, by the anterior part of the chiasma, seems to be erroneous. In the tract, the fibres from the opposite eye lie chiefly on the inferior (basal) surface; the direct fibres lie in the superior part of the tract; and the fibres from the opposite tract are on the inner side. (V. Gudden.) The fibres from the lower half of each retina lie outside to those from the upper half (Marchand). Beneath the posterior extremity of the thalamus the optic tract divides into two parts. The outer is the larger, and goes to the optic thalamus, external corpus geniculatum, and anterior quadrigeminal body. All these structures atrophy after extirpation of the eye of young animals (v. Gudden). From each of these bodies fibres pass to the posterior (sensory) portion of the internal capsule and thence to the white substance of the occipital lobes, in the "optic radiation" of Gratiolet. Some fibres of the tract have been said to pass directly to the hemisphere, but it is doubtful whether any have this direct course. Of these intermediate stations, the external corpus geniculatum has been commonly regarded as that which is of chief importance in connection with the visual fibres since its atrophy has been frequently observed in cases of long-standing atrophy of the tract. But many recent observations establish the fact that disease limited to the posterior extremity of the optic thalamus may cause hemianopia, and it seems doubtful whether the symptom is caused by disease of the external geniculate body.* Hence we must regard the pulvinar as the intermediate visual centre, and the precise function of the external corpus geniculatum becomes again mysterious. The inner root of the optic nerve goes to the internal geniculate body, and is said to contain only those fibres which at the optic commissure turn back to the opposite optic tract (v. Gudden). Through this body the nerve also appears to be connected with the posterior

FIG. 212.

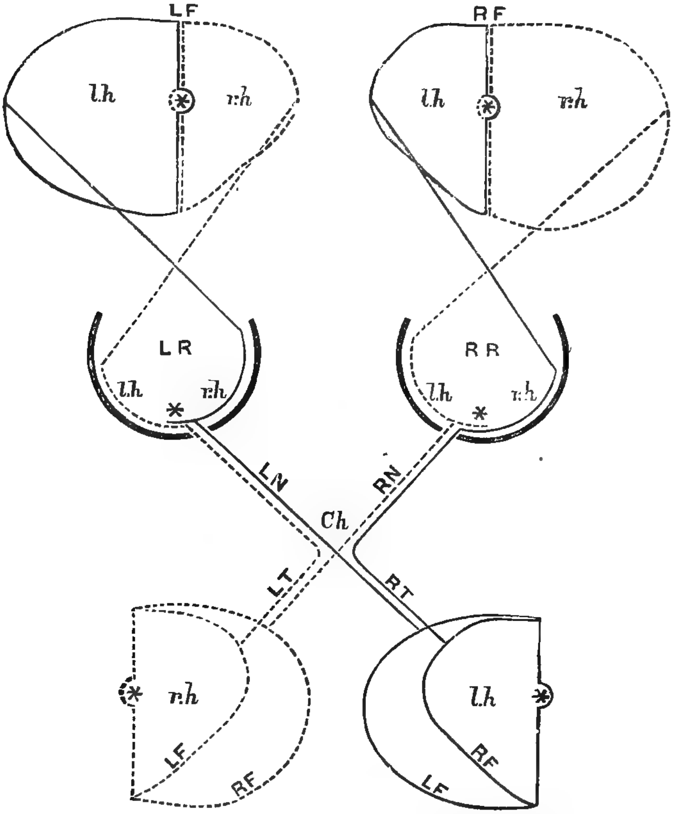


Diagram of the relation of the fields of vision, retina, and optic tracts. R F, L F, right and left fields—the asterisk is at the fixation point; R R, L R, right and left retina—the asterisk is at the macula lutea; *l h*, *r h*, left and right half of each retina, receiving rays from the opposite halves of the fields; R N, L N, right and left optic nerves; *Ch*, chiasma; R T, L T, right and left optic tracts; below are the superimposed halves of the fields from which impressions pass by each optic tract.

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* It is commonly believed that the fibres are interrupted by nerve cells in these ganglia, but Darkschewitsch (*Arch. f. Anat. u. Phys.*, *1886*) has recently asserted that they pass through without interruption, a fact that can scarcely be proved by the method of investigation employed by him. Secondary degeneration from a lesion of the occipital lobe causing hemianopia has been traced into the pulvinar, but no farther (Richter, *Arch. f. Psych.*, xvi, 638).

corpus quadrigeminum. The connection of the optic nerves with these bodies probably subserves the relation between visual impressions and ocular movements. The relation of the cortex to vision has been already mentioned, and some other points in connection with the function of the nerves will be described in the account of their diseases.

The central connections of the *olfactory nerve* are still but little known. The external root, crossing the fissure of Sylvius, passes to the temporal lobe—toward the anterior part of the uncinate convolution. There is some evidence, as we have seen, that an olfactory centre exists in this locality. Some fibres (according to Obersteiner) pass to the optic thalamus on the same side. The fibres of the inner root go to the anterior commissure, one part of which, in animals, is large in proportion to the size of the olfactory nerves, and is regarded as commissural between the two olfactory bulbs. But there is evidence that the path from one olfactory nerve reaches the cortex of the opposite hemisphere, since the sense of smell has been affected, together with the other special senses on the same side, by disease of the posterior part of the opposite internal capsule, and also by disease of the cortex (see p. 461). It is possible, as Charcot suggests, that the path damaged by disease involving the internal capsule is that which crosses by the anterior commissure.*

CEREBELLUM.

The division of the cerebellum into two hemispheres and a middle lobe, founded on external conformation, is borne out by the little we know of its function. Nevertheless, all parts consist of similar structural elements. Branching folia everywhere constitute the superficial layer; the gray substance which envelops these folia, and, following all the involutions of their surface, is of enormous superficial extent, has an identical three-layered structure in all parts. Below a superficial "gelatinous stratum" is a layer that contains nuclei and branching fibres, which are the processes of the large "cells of Purkinje." These cells lie at the junction of this and the deepest layer of the cortex; the latter is composed of nuclear bodies similar in aspect to those that constitute the granule layers of the retina. The gray matter is not confined to the cortex. It exists also in the white substance, as a series of masses or "nuclei." Of these the corpus dentatum lies in the inner part of the hemisphere, and closely resembles the olivary body of the medulla. In the white substance of the middle lobe is the "roof nucleus,"

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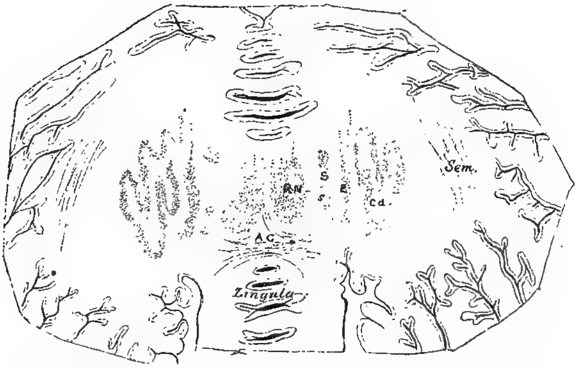
* But the subject is involved in great obscurity. The other fibres of the anterior commissure pass into the temporal lobe, but they have not been traced to the uncinate gyrus. Meynert says that fibres pass back from the anterior commissure, and thus the olfactory path (whether from the nerve by the one part of the commissure, or from the opposite uncinate centre by the other part of the commissure) may reach, as it certainly does, the internal capsule, but Ganser denies that fibres pass backward from the anterior commissure. As a further instance of the uncertainty as to the precise relations of the anterior commissure, most of its fibres have been found secondarily degenerated in a case of bilateral softening of the region of the lingual gyrus, by Popow, who regards it as a commissure between these convolutions (*Wratsch, 1886,* and *Cent. f. Nervenh., 1886,* p. 684).

RN, Fig. 213, so called by Stilling because it lies just above the roof of the fourth ventricle. At the junction of the hemisphere and middle lobe are two other gray bodies, the "spherical" and "emboliform" nuclei (Stilling). The latter is the nearer to the dentate body.

The course of the fibres in the white substance is still imperfectly known. The difficulty of tracing them is extreme, unaided as it is by secondary degeneration, and the conclusions reached by different investigators do not altogether correspond. Although much has been written on the subject, the facts are so uncertain, and their application is so meagre, that a lengthy discussion of the subject is unprofitable. There are few well-marked tracts of fibres, and of those that can be distinguished, such as the semilunar tract (*Sem*, Fig. 213) in the outer part of the hemisphere, the connections are doubtful. Some fibres of the hemisphere appear to pass in various directions between the different folia of the cortex; others pass inward, or toward the middle line. Of the latter, some go to the dentate nucleus, and interlace around it in a felty capsule, the "fleece" of Stilling; others pass to the middle peduncle and end in the gray matter of the pons, and through this are connected with the cortex of the cerebral hemispheres and with the corpus striatum, as we have already seen (p. 472). Other fibres pass to the middle lobe, part of which may have a commissural function. Flechsig states that many fibres connect the cortex of the superior vermiform process with that of the cerebellar hemispheres.

FIG. 213.



Nuclei of the cerebellum. C d, corpus dentatum; E, emboliform nucleus; S *s*, parts of the spherical nucleus; AC, anterior commissure; *Sem*, semilunar tract.

Of the fibres of the *inferior peduncle* many course to the neighborhood of the dentate nucleus; some enter this nucleus, and apparently connect it with the olivary body. The destination of those that pass by the dentate nucleus is very uncertain. Stilling thinks that most go to the cortex of the hemisphere and a few directly to the superior peduncle. Another and very important series of fibres of the inferior peduncle pass to the middle lobe. Among these are the fibres of the direct cerebellar tract, and probably fibres from the posterior pyramids and from the auditory nerves and nuclei. Mc of these fibres are believed to cross the middle line, and end in the gr matter of the opposite side of the middle lobe; some in the roof nucleu others probably in the gray cortex of the superior vermiform process. Tl fibres of the *middle peduncle* connect the gray matter of the pons with tl cortex of the cerebellar hemisphere. Most of the fibres of the *superi peduncle* go to the neighborhood of the dentate nucleus, but some are sa by Stilling to radiate to the cortex of the anterior and outer part of tl hemisphere, others to pass to the inferior peduncle, and others again to tl middle lobe. The fibres of the superior peduncle cross the middle lir beneath the corpora quadrigemina, and pass through the opposite re nucleus.

The precise functions subserved by the cerebellum have been long mystery. There is, however, abundant evidence, experimental and path logical, to show that this part of the brain is in some way connected wit the coördination of movement, and especially with those muscular actio which maintain the equilibrium of the body. It appears, however, that th function is confined to the middle lobe. As Nothnagel first showed, tl function is not impaired by disease of the hemispheres, unless such disea compresses the middle lobe. We do not know to what extent this functio is subserved by the roof nucleus, or by the gray matter of the middle corte We may reasonably assume that the fibres which go to the middle lobe fro the cord, those of the direct cerebellar tract and (probably) from the pos pyramidal nuclei, are connected with this function, since we have seen reaso (in the account of the spinal cord) to believe that these fibres constitu the path of centripetal impressions from the muscles, interruption of whic causes incoördination. We may likewise assume that this function is als subserved by the fibres which pass from the auditory nerves or nuclei to tl middle lobe, since that part of the auditory nerve which comes from tl semicircular canals conducts impressions determined by the position an movement of the body (acting on the endolymph), and disturbance of the fibres deranges the maintenance of equilibrium. The only effect of electric stimulation of the middle lobe obtained by Ferrier was a movement of tl eyes. It is not probable that there are centres for the ocular movements i the cerebellum, but a connection between the centres for these movemen and the middle lobe is highly probable, since the relation of seen objects t the body is estimated unconsciously from the position of the eyes; the degre of innervation of the ocular muscles must influence the mechanism for mai taining equilibrium. It is to be noted that most of these impressions, *e. g* from the muscles of the legs and from the semicircular canals, do n influence consciousness directly, and so cannot, strictly speaking, be calle "sensory." It may be that in the middle lobe of the cerebellum we have mechanism by which the various centripetal impulses are combined an harmonized, and that an influence is thence exerted on the motor centres i the cerebral hemispheres, from which the muscles are excited to the necessar contraction.

The function of the hemispheres of the cerebellum is still mysterious. They lessen in size as we descend the scale of animals, until they disappear in birds, in which the whole cerebellum corresponds to the middle lobe of man. They are connected chiefly with those parts of the cortex of the cerebrum which chiefly subserve psychical processes. With these parts, moreover, the cerebellar hemispheres have this in common, that simple loss of substance causes no definite and recognizable loss of any function of the brain. The loss can apparently be compensated by other parts. Hence it seems possible that the old theory may be correct which assumes that the cerebellar hemispheres are in some way connected with psychical processes.

BLOOD VESSELS OF THE BRAIN.

The blood supply to the brain is a subject of great importance, because many diseases are due to the rupture or obstruction of vessels, and the distribution of the vessels (especially that of the arteries) explains the incidence of a large number of cerebral lesions.

Distribution of the Cerebral Arteries.*—The blood supply to the brain comes from the carotid and vertebral arteries. The mode of origin of the two carotids presents an important difference, which explains why embolism is more frequent on the left side than on the right. The left carotid arises directly from the highest part of the arch of the aorta, and its course is thus almost a direct continuation of the direction of the current of blood in the aorta, whereas the right carotid comes from the innominate, which arises from the aorta at an angle with the course of the aortic blood. Hence clots, washed from the cardiac valves, pass into the left carotid more readily than into the right.

There is a similar difference in the mode of origin of the two vertebrals. The left, usually the larger, arises from the left subclavian in the ascending part of its course, while the right subclavian gives origin to its vertebral when horizontal. Hence a plug can enter the left vertebral more readily than the right, because the direction of this vessel is that of the blood current, and this is probably also the reason why the left vertebral is usually larger than the right. But this does not determine any difference in the frequency of embolic obstruction in the two posterior cerebral arteries, because, to reach these, the plug has to pass through the common basilar.

The "circle of Willis," it will be remembered, is formed by the two posterior communicating arteries, which pass, one on each side, from the internal carotids to the posterior cerebrals into which the basilar divides, and by the anterior communicating artery, between the two anterior cerebrals of the internal carotid. The usual arrangement is subject to occasional varieties, which are important because they explain some of the anomalous facts of disease. The vertebrals often present abnormalities which will be described further on. One posterior communicating artery is sometimes very small, and occasionally one, usually the right, is abnormally large, and through such a vessel the posterior cerebral may even arise from the carotid, a minute branch from the basilar representing the usual origin of the vessel. Duret has even seen the chief blood supply to the basilar come from the carotid, by a large artery which entered the basilar in the middle of its course, the vertebrals and the lower part of the basilar being very small. Occasionally one anterior cerebral, almost always the right (Duret), is very small at its origin, and the anterior communicating artery is abnormally large, so that the right anterior cerebral virtually arises from the left. It is to be noted, further, that there are usually communicating branches between the posterior cerebrals and the anterior cerebellar arteries, which, with the posterior communicating arteries, constitute an important connection between the carotid and the basilar systems.

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* Our knowledge of the distribution of the vessels within the substance of the brain is largely due to the researches of Duret, *Arch. de Physiologie, 1873 and 1874*, and Heubner, *Centralblatt f. m. Wissensch., 1872.*

The internal carotid, on each side, divides into the anterior and middle cerebral arteries, and from these, and the posterior cerebral, the blood supply of each hemisphere is derived. From the circle of Willis, and the commencement of the three cerebral arteries, small branches arise which supply the central ganglia of the hemisphere and the adjacent white substance, while the three arteries ramify over the surface of the brain, and supply the gray cortex and the greater part of the white substance of the hemisphere. Between these central and cortical systems there are no anastomoses, nor do the central branches communicate with each other. The anastomoses between the several cortical branches seem to vary in different persons. In many, the communications are too slight to permit of the establishment of a collateral circulation. In others they appear to be sufficient to maintain nutrition. Moreover, similar differences seem to exist among the cortical branches in the same individual and even among those of the same vessel; hence the effects of obstruction of these vessels vary much.*

The *central arteries* may be divided into six groups, two medial and four lateral, two on each side.

The *anterior medial group* consists of a few twigs, inconstant, which are given off from the anterior cerebrals and anterior communicating artery. They supply the anterior extremity of each caudate nucleus. The *posterior medial group* consists of twigs given off by the posterior cerebral arteries near their origin from the basilar; these twigs pass into the posterior perforated spot, and supply the inner parts of the optic thalami and the walls of the third ventricle. The supply by these groups is insignificant as regards extent, but important in regard to position, on account of its proximity to the ventricles, into which blood, escaping from one of these vessels, may readily burst.

The lateral groups supply the chief part of the central ganglia. The *antero-lateral* group arises from the middle cerebral near its origin (in the first inch of its course) and consists of a number of small arteries which, coming off at a right angle, pass into the "anterior perforated space," and supply the caudate nucleus (except its head), the lenticular nucleus, the internal capsule, and part of the optic thalamus. These arteries are of great importance, and may be divided into two sets (Fig. 214): an *internal* set, the vessels of which pass directly through the internal segment of the lenticular nucleus to the internal capsule, and an *external*, consisting of vessels that course upward for a short distance outside the lenticular nucleus, and then pass into its third segment, and through this to the internal capsule. The vessels extend through the internal capsule, the anterior to the caudate nucleus (except its head), the posterior to the optic thalamus (except its inner and hinder part). Hence the anterior have been called the *lenticulo-striate*, and the posterior the *lenticulo-optic* arteries. These vessels are prone to rupture, and one of the former group, which runs at the outer side of the lenticular nucleus (Fig. 214) from its size and tendency to give way, has been called by Charcot "the artery of cerebral hemorrhage." The vessels burst chiefly outside the lenticular nucleus, and the hemorrhage separates the nucleus from the external capsule.

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* The conclusions of Heubner and Duret differ regarding the anastomoses of the cortical vessels; the latter could not find the anastomoses described by the former. The facts of pathology are strongly in favor of the view that anastomoses between the cortical branches occur often, but not invariably. An injection often passes beyond the limit of the distribution of the artery injected.

Fig. 214.

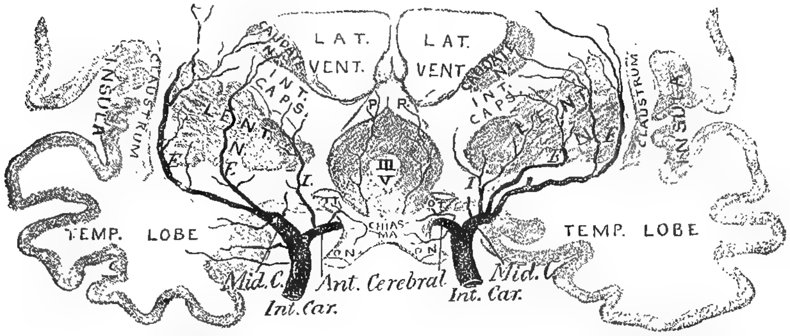
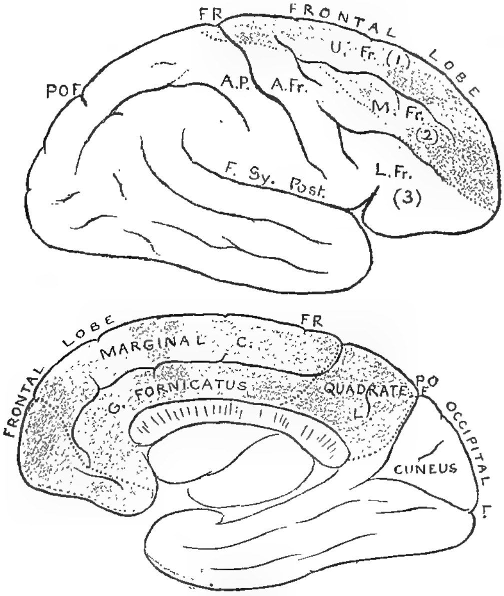


Diagram of the blood supply to the central ganglia by the lenticulo-striate arteries, external (*E*) and internal (*I*). III V, third ventricle; P P, pillars of the fornix; *Mid. C*, middle cerebral artery. (*After Duret.*)

Figs. 215, 216.



Figs. 215 and 216.—Area of the cortex on the outer and medial aspects, supplied by the anterior cerebral artery. The supply of the several branches is indicated by the dotted lines.

The *postero-lateral group* arises from the posterior cerebral, after it has passed round the crus, and supplies the hinder part of the optic thalamus. Hemorrhage from the rupture of these vessels is apt to invade the crus on the one side and the hinder part of the internal capsule on the other. The posterior cerebral gi branches also to the crus and to the corpora quadrigemina.

Of the supply to the *cortex* from the three vessels, that from the middle cereb is the most extensive and the most important, embracing, as it does, the cent (motor) convolutions. The general plan of distribution of each artery is the san Each divides into certain branches, and these again divide and ramify ; from t branches and the ultimate ramifications (in the pia mater) twigs are given off to t cerebral substance. These are of two kinds, long and short. The short are *cortic* supplying only the gray cortex, in which they form a capillary network, m abundant in the middle layers which contain the large nerve cells. The lo are *medullary*, and, passing through the cortex, penetrate the white substance various depths. The distribution of the longest reaches the neighborhood that of the central vessels, but the two do not blend, and at the confines of t two areas small cavities often form in old age. The cortical moreover do n communicate with each other, and, although there is a scanty communicati between the areas supplied by the larger trunks, it is often insufficient to establ a collateral circulation if one is obstructed. Hence the obliteration of branch usually (though not invariably) causes softening limited to the area supplie This is most extensive on the surface, and lessens in extent toward the cen of the brain.

The *anterior cerebral* curves round the corpus callosum and supplies part of t orbital lobule, the inner surface as far as the quadrate lobule, and the frontal lol with the exception of the inferior and ascending frontal convolutions. Its ch branches are three, and supply the following parts: (1), the supra-orbital lob (as far as the orbital sulcus) and the inner aspect of the anterior extremity of t hemisphere ; (2) the anterior half of the inner surface, in front of the quadr lobule, also the first and second frontal, and the highest part of the ascendi frontal convolution ; (3) to the quadrate lobule (precuneus).

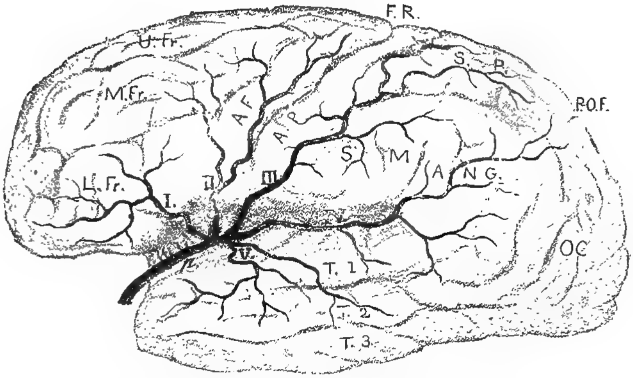
The *middle cerebral* divides, opposite the island of Reil, into four branch marked I, II, III, IV, in Fig. 217. These lie in the sulci of the insula, and th passing on to the surface of the hemisphere, have the following distribution (Fi 217 and 218): (1) to the third (inferior) frontal convolution ; (2) to the lower tv thirds of the ascending frontal and the root of the middle frontal ; (3) to the wh ascending parietal, superior parietal lobe, and adjacent part of the inferior parie lobule, often also to a small part of the ascending frontal ; (4) to the convolutic about the posterior limb of the fissure of Sylvius, viz., part of the inferior parie lobule (supra-marginal and angular gyrus), posterior extremity of the super parietal lobule, and the hinder part of the first two temporal convolutions. Fr this, near its origin, one or two large branches arise (5) which supply the anter part of the first and the greater part of the second temporal convolutions. Th are individual variations in the precise area supplied by the branches, and also the posterior limit of the distribution of the middle cerebral. This is of consid able importance, because the posterior part of the angular gyrus often escapes t softening caused by obstruction of the middle cerebral.

The *posterior cerebral* supplies the greater part of the inferior aspect of the br and also the occipital lobe, inner and outer surfaces, by three branches. The v sel divides into these branches after winding over the crus, and giving off its cent branches and twigs to the tegmentum of the crus, and to the corpora quadrigemi Its branches supply (1) the lower part of the uncinate convolution ; (2) the infer part of the temporal lobe ; (3) the cuneus, lingual convolution (*i. e.*, the poster part of the uncinate gyrus beneath the calcarine fissure), and also the outer surf of the occipital lobe.

Thus the middle cerebral supplies the chief part of the motor convolutions and the motor central structures, as well as the cortical auditory centre, and the greater part of the angular region. The posterior cerebral supplies the optic ganglia (optic thalamus and corpora quadrigemina and geniculata), and in the cortex the visual region in the occipital lobe.

The blood supply to the pons, medulla oblongata, and cerebellum, is derived from the vertebrals and basilar. The arrangement of the supply to the medulla and pons is very important on account of the centres which are here so closely grouped together.

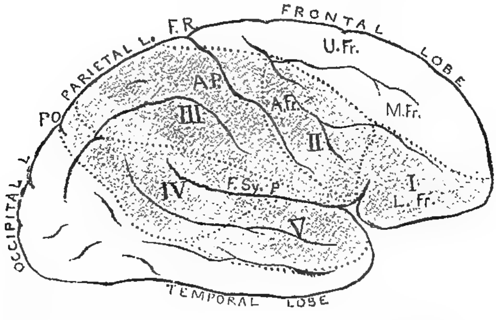
FIG. 217.



Course of the branches of the middle cerebral artery.

It will be remembered that each vertebral, before it joins its fellow to form the basilar, gives off an inferior cerebellar artery and an anterior spinal artery. From all these vessels small branches enter the pons and medulla. The cerebellum is nourished, not only by the inferior cerebellar from the vertebral, and superior cerebellar from the anterior extremity of the basilar, but also by a large branch, the middle cerebellar artery, which comes off from the basilar, about the middle of its course, and passes outward over the pons and middle cerebellar peduncle.

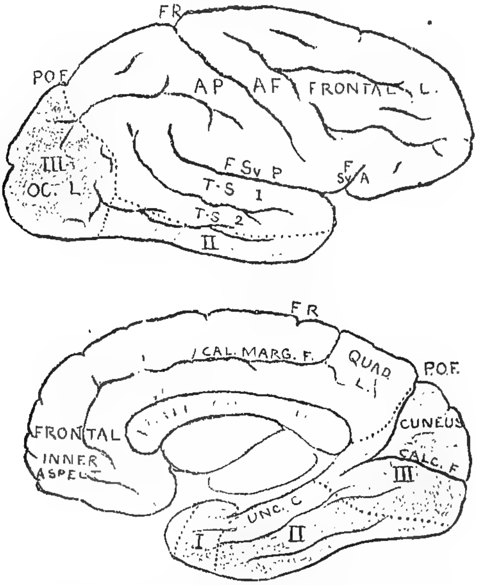
FIG. 218.



Area of distribution of the branches of the middle cerebral artery.

The arterioles for the pons and medulla consist of two sets. (1) A series of *medial* vessels enter near the middle line in front, and pass directly back, close to the raphe, to the nerve nuclei, which receive from these arteries the chief part of their blood supply; (2) *lateral or radicular* vessels pass to the nerve roots, and on each root an artery divides into a descending branch, which extends along the nerve, and an ascending branch, which enters the medulla with the nerve fibres, and accompanies them as far as their nucleus, giving off branches as it proceeds. But the blood supply to the nucleus derived from the radicular branch is far less than that from the median. The vertebral supplies the radicular branches to all the bulbar nerves, except a branch to the spinal accessory nerves which comes from the inferior cerebellar, and to the hypoglossal, from the anterior spinal artery. The branches to the facial and auditory nerves may come from the vertebral or basilar, or both. All the other radicular branches are derived from the basilar, but the middle cerebellar artery sometimes gives branches to the auditory and fifth nerves.

FIGS. 219, 220.



FIGS. 219 and 220.—Area of the cortex supplied by the branches (I II III) of the posterior cerebral artery.

The median branches form a continuous series, but are divided by Duret into four sets, termed, from their position, *bulbar*, *sub-pontine* (at the lower edge of the pons), *medio-pontine* (along the pons), and *supra-pontine* (at the upper edge of the pons). The lower bulbar branches come from the anterior spinal, and supply the nuclei below the calamus scriptorius—the hypoglossal and accessory. The sub-pontine, from the basilar, supply the pneumogastric, glosso-pharyngeal, and auditory nuclei, while the medio-pontine and supra-pontine branches supply the nuclei in the upper half of the floor of the fourth ventricle, as well as the sixth. The anastomoses of these vessels are unimportant.

It is necessary to remember that the larger trunks about the medulla present frequent variations, which entail corresponding variations in the origin of the nutrient arteries. The left vertebral is usually larger than the right, and the latter may be very small, so that the basilar is practically a continuation of the left vertebral only. There is usually only one anterior spinal artery, arising from both vertebrals, or arising from the left only. If there are two, they usually communicate, but the left occupies the medial furrow and furnishes the median nutrient arteries. These variations explain why obstruction of one vertebral sometimes affects the nuclei on one side, sometimes on both, and sometimes on neither.

Branches to the olivary body and anterior pyramids come from the vertebral or anterior spinal; those to the restiform body arise from the inferior cerebellar, and this also gives off the posterior spinal arteries, from which branches pass to the posterior pyramids and to the upper part of the central canal, chiefly at the level of the decussation of the pyramids.

The chief *cerebellar arteries* are (1) the *posterior* from the vertebral (sometimes from the basilar) which winds round the medulla between the hypoglossal and pneumogastric nerves, and, after giving origin to the posterior spinal and branches to the choroid plexus of the fourth ventricle, supplies the inferior vermiform process and the posterior region of the hemisphere; (2) the *median* (Duret) which leaves the basilar at the middle of the pons and passes outward over the middle peduncle of the cerebellum to ramify on the under surface; (3) the *superior cerebellar*, arising from the basilar close to its bifurcation, curves round the pons, and supplies the anterior and upper parts of the hemisphere. These cerebellar arteries communicate with each other, and the superior is often connected by one or more twigs with the posterior cerebral.

VENOUS CIRCULATION.—The veins of the convexity of the hemisphere, and of its inner surface, ascend and open in a forward direction into the superior longitudinal sinus. One of the veins of the convexity courses at first along the fissure of Sylvius, and afterward ascends across the hemisphere: it has been termed the "Sylvian vein" by Duret. The course of the surface veins is important because it helps to explain the frequency with which clots form within them. Elsewhere the blood from ascending arteries passes into descending veins, so that the feeble pressure through the capillaries is supplemented by the influence of gravitation. Elsewhere ascending veins convey blood that has been brought by descending arteries, and the venous pressure is aided by the "hydrostatic pressure," which tends to make the blood rise in the veins to the level of the heart. But on the brain, blood from ascending arteries passes into ascending veins. The openings of these veins into the longitudinal sinus being directed forward, the entering blood is opposed in direction to the current in the sinus, and the effect must be to retard the flow in both veins and sinus. Moreover, in the erect posture, the anterior part of the longitudinal sinus has also an ascending course, while the trabeculæ that occupy the lumen of the sinus must offer some hindrance to the movement of the blood. These circumstances help us to understand the readiness with which clots form in the cortical veins and longitudinal sinus, when other circumstances favor the coagulation of the blood. Indeed, the marvel is that thrombosis is not more common than it is.

The veins of the inferior surface of the brain enter—the anterior the cavernous sinus, the middle the petrosal sinuses, the posterior the lateral sinus. The veins of Galen, from the ventricles, receive not only the blood from the ventricular walls and choroid plexuses, but also from the corpus callosum. The veins of the upper surface of the cerebellum open into the veins of Galen or the straight sinus in which the veins of Galen end. Those of the inferior surface open—the posterior into the lateral sinus, and the anterior into the occipital sinuses. The individual veins have not, as a rule, free communications with those adjacent, although differences exist in this respect. But the system of the convexity (superior longitudinal sinus) has three important communications. A large vein always passes from the Sylvian vein to the superior petrosal sinus (Trolard), and another, at the base, connects the commencement of the Sylvian vein with the basilar vein, and thus with the straight sinus. The vein of the corpus callosum (going to the veins of Galen) often communicates freely with the veins on the inner surface of the hemisphere, which go to the superior longitudinal sinus. The communications between the various sinuses, with the exception of the superior longitudinal, are free. Thus the two cavernous sinuses are connected by the circular sinus around the pituitary body, and each cavernous sinus has a double communication (by the two petrosal sinuses) with the lateral sinus and jugular vein, while the two inferior petrosal sinuses are connected by the "transverse sinus" across the basilar process of the occipital bone. The cavernous sinus receives the ophthalmic vein, the superior petrosal sinus receives veins from the internal ear, and into the lateral sinus some mastoid veins open. The occipital sinus receives the spinal veins, and many sinuses receive veins from the diploë of the skull.

There exist certain communications between the cerebral sinuses and veins outside the skull. The following are the chief. Veins from the nose enter the anterior extremity of the superior longitudinal sinus. The ophthalmic vein (cavernous sinus) communicates freely with the facial vein. By the veins of the mastoid cells the occipital vein communicates with the lateral sinus. Moreover, small "emissary" veins pass through minute foramina in the cranium and connect certain sinuses with external veins. The most important of these are between the superior longitudinal sinus and the veins of the scalp, between the cavernous sinus and the pterygoid plexus of the internal maxillary vein, between the inferior petrosal sinus and the deep veins of the neck. A further communication, very variable in degree, is established by the veins of the diploë. Lastly, the spinal system, which has free external communication, is connected by six veins with the sinus system of the brain. The cerebral veins and sinuses have no valves. These facts are important in regard to the external signs of thrombosis in the sinuses within the skull.

SECONDARY DEGENERATION IN THE BRAIN.

We have already seen that some tracts of fibres in the brain undergo degeneration after a lesion in their course or in the gray matter from which they spring. In the latter case the whole length of the fibre degenerates, in the former only that portion which lies on the distal side of the lesion; *i. e.*, on the side farthest from the nerve cell which gives origin to the fibre. This secondary degeneration proceeds until the fibres are again interrupted by gray matter, and there it ceases. The nature of secondary degeneration was considered on page 60, where we saw that the axis cylinder of each nerve fibre must be regarded as the prolonged process of a nerve cell, depending on that nerve cell for its nutritional integrity, and that the nerve fibre therefore undergoes degeneration when the cell is destroyed or the fibre is cut off from the cell. As a general rule the direction in which a fibre degenerates is that in which it conducts; that is, the cell on which the nutrition of the fibre depends, the cell of which it is the prolonged process, is the cell from which the nerve impulses originate that traverse the fibre, or, in other words, it is this cell only which excites the functional activity of the fibre. But this rule of the identity of the direction of conduction and degeneration does not seem to be true of all nerve fibres. As far as is yet known, it is true of all motor fibres, but there is a conspicuous exception to it in the case of the fibres of the peripheral sensory nerves. These conduct centripetally, but degenerate centrifugally from the ganglion on the posterior root. Above the ganglion they conform to the general law. In the central nervous system we may take the direction of degeneration as presumptive but not as absolute proof of the direction of conduction. It is doubtful whether all nerve fibres undergo secondary degeneration. The fibres of the corpus callosum apparently do not. Possibly some fibres pass between two nerve cells, and are so related to both (connecting, for instance, undivided processes) that either cell is able to maintain the nutrition of the fibre.

FIG. 221.

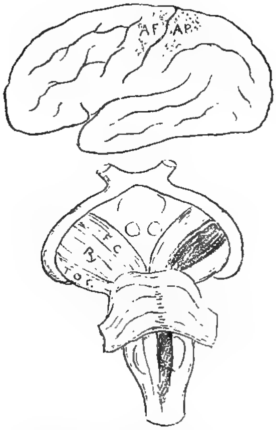


FIG. 222.

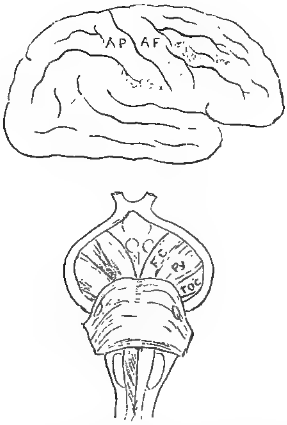


FIG. 221.—Lesion of the upper part of the central convolutions, causing descending degeneration of the pyramidal tract in the crus and the anterior pyramid of the medulla. (*After Brissaud.*)

FIG. 222.—Partial disease of the central convolutions, causing partial degeneration of the pyramidal fibres in the crus and medulla. More degenerated fibres come to the surface near the pons than near the tract.

The most important and extensive secondary degeneration is that of the fibres of the pyramidal tracts, proceeding from the central convolutions. The course of these fibres has been already fully described (p. 468). The lesion causing their degeneration may be in the cortex, or anywhere in the course of the fibres. If it is above the crus, the degeneration appears at the under surface of the crus, in its middle two-fifths (Fig. 221), and is wider above, near the optic tract, than below, near the pons, where the pyramidal fibres are overlapped by those on each side. The pons is reduced in size on the side of the degeneration, and the anterior pyramid of the medulla is small and gray. To cause extensive degeneration of these tracts, a lesion of the cortex must be also extensive, such as that shown in Fig. 221. A smaller lesion (Fig. 222) causes partial degeneration, some fibres are involved and others escape.

A lesion within the pons causes degeneration only of the fibres below the lesion, as in the case shown in Fig. 229. The effects of the area of softening in the crustal half of the pons, shown in Fig. 223, present a good illustration of the downward course of the degeneration through the medulla and the decussation of the pyramids. In Fig. B the degenerated bundles of the pyramidal tract are separate, but in C and D they are united in the anterior pyramid, which is completely degenerated. The passage of the degenerated fibres through the decussation of the pyramids is shown in F and G; in the latter most of them have reached the opposite lateral column of the cord, and occupy the position of the lateral pyramidal tract, while the degeneration in the anterior column is reduced almost to the small dimensions which it presents in the lowest figure (of the cord). The descending changes in the spinal cord have been described on page 129, and are shown in Fig. 224.

In some cases of disease of the motor path in one hemisphere, there is a double degeneration in the cord, a slight degeneration in the lateral pyramidal fibres on the side of the cerebral lesion, as well as considerable degeneration in the lateral tract of the opposite side. Attention has been called to the frequency of this double degeneration by Pitres.* An example of it is shown in Fig. 225. It is, perhaps, related to the double representation of the leg in both hemispheres of the brain, which will be mentioned in the account of the symptoms of brain disease. It is not present, however, in all cases.

Another frequent secondary degeneration is that of the fibres which course from the pre-frontal lobe of the brain (in front of the central convolutions) through the anterior limb of the internal capsule, and the inner part of the crusta of the cerebral peduncle to the gray matter of the pons. In the pons their degeneration ceases, but the evidence that they are connected, through this gray matter, with the cerebellum is very strong (see p. 470). Hence they are termed the fronto-cerebellar fibres (F C, Fig. 226). Their degeneration is visible in the most internal portion of the crus, and may be produced by disease either of the frontal lobe in front of the ascending frontal convolution (see Fig. 226) or of the anterior limb of the internal capsule (Fig. 227).

Degeneration of the most external fibres of the crusta, those that pass between the gray matter of the pons and the temporal and occipital lobes, has been met with in rare cases. It has been caused by extensive damage to the cortex (Betcherew) or by a lesion in the vicinity of the hindmost fibres of the internal capsule, as in the case shown in Fig. 228. It is not the disease of the capsule itself which causes the degeneration, for the fibres that run in this part of the capsule proceed from the tegmentum (p. 472), and probably degenerate, as they certainly conduct, upward. The degeneration is due to the damage to the adjacent fibres, which, as they leave the crus, do

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* See also Hadden and Sherrington, *Brain*, *1886*.

FIG. 223.

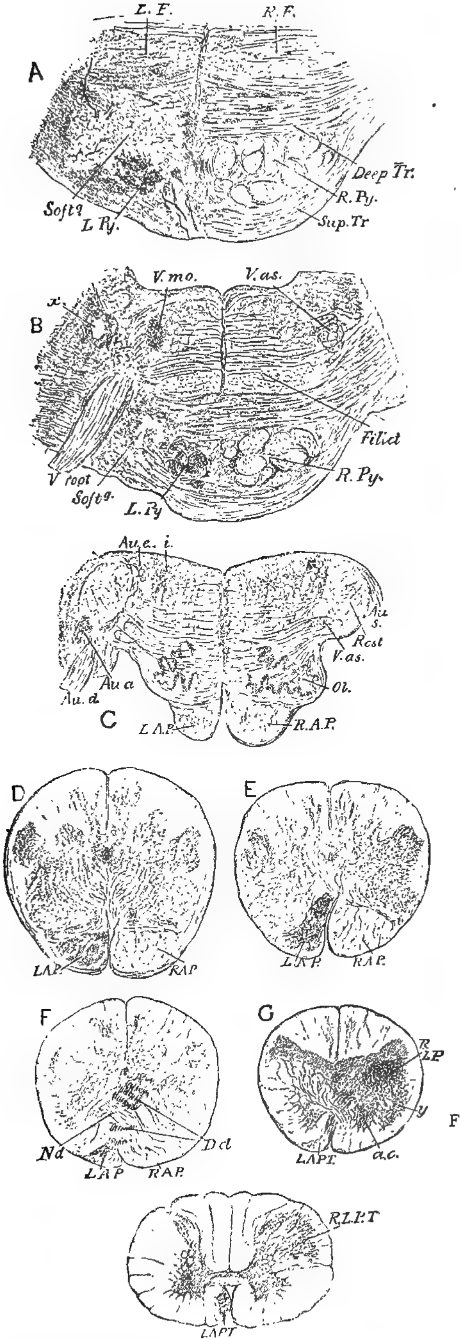

FIG. 224.

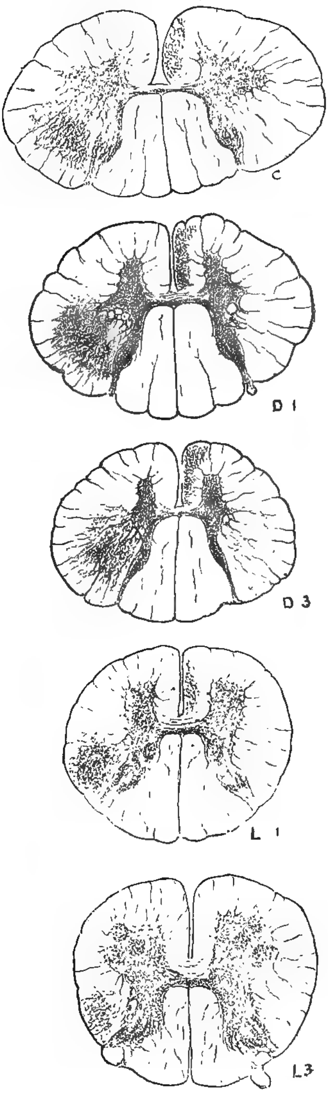

FIG. 223.—Softening in the left side of the pons in the crustal portion (A), and near the nucleus of the fifth nerve (B *x*). The other figures represent sections through various parts of the medulla and decussation of the pyramids; the last is of the spinal cord. *L Py*, *R Py*, left and right pyramidal tract; *L F*, *R F*, left and right fillet; *Deep Tr*, *Sup Tr*, deep and superficial transverse fibres of the pons; *V mo*, *V asc*, motor nucleus and ascending root of the fifth nerve; *Au d*, *Au s*, deep and superficial roots of the auditory nerve; *Au e*, *i*, *a*, its external, internal and anterior nuclei; *Rest*, restiform body; *L A P*, *R A P*, left and right anterior pyramid; *D d*, degenerated (left), *N d*, not degenerated (right) pyramidal fibres at the decussation of the pyramids; *L A P T*, left ant. pyr. tract; *R L P T*, right lateral pyr. tract; *a c*, anterior cornu; *y*, slight degeneration in the anterior part of the lateral column.

FIG. 224.—Degeneration of the right anterior and left lateral pyramidal tracts in the spinal cord, consequent on a lesion in the right hemisphere of the brain, causing left hemiplegia.

not enter the capsule, but pass close by it, to radiate to the temporal and occipital cortex.

The transverse fibres of the pons are said to degenerate when interrupted by a lesion in their course. Of adjacent fibres, some seem to degenerate in one direction, and other fibres in the opposite direction, so that the degeneration extends on each side of the lesion, as in Fig. 229. In the case shown in Fig. 223 such degeneration could not be distinctly traced.

FIG. 225.

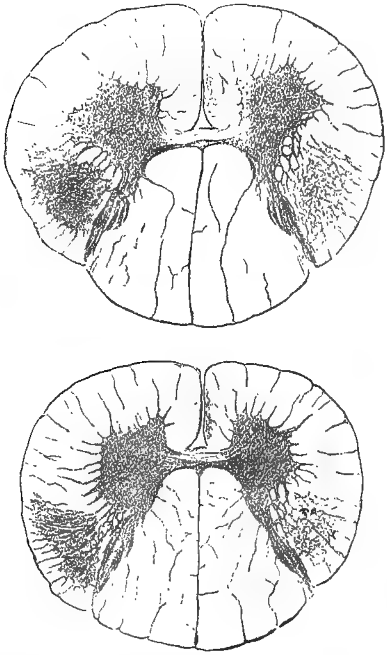

FIG. 226.

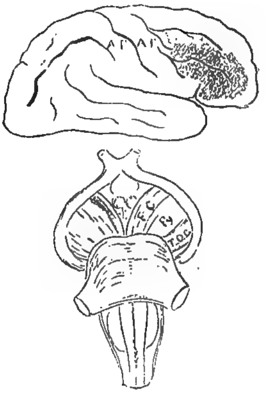

FIG. 227.

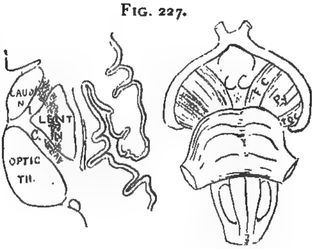

FIG. 225.—Bilateral degeneration of the pyramidal tracts secondary to a lesion in the right hemisphere. (*From sections by Prof. Pitres.*)

FIG. 226.—Area of disease of the right pre-frontal lobe, causing partial degeneration of the inner fibres of the crusta.

FIG. 227.—Lesion of the lenticular nucleus and of both limbs of the capsule, causing partial degeneration of the inner and pyramidal fibres of the crusta.

FIG. 228.

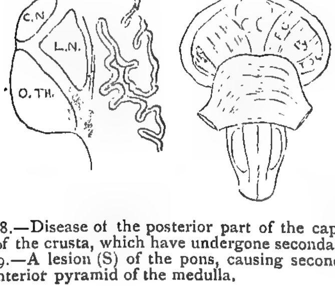

FIG. 229.

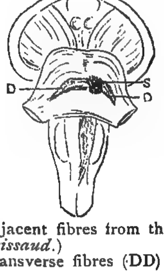

FIG. 228.—Disease of the posterior part of the capsule, damaging also the adjacent fibres from the outer part of the crusta, which have undergone secondary degeneration. (*After Brissaud.*)

FIG. 229.—A lesion (S) of the pons, causing secondary degeneration of the transverse fibres (DD) and of the anterior pyramid of the medulla.

The above are the chief secondary degenerations that are of medical significance. Some other degenerations have been met with, such, for instance, as that of the fillet, but they are not yet of sufficient importance to warrant their detailed description.

THE SYMPTOMS OF BRAIN DISEASE.

The symptoms produced by diseases of the brain are, for the most part, the same in character, whatever be the morbid process that causes them. They depend on the seat of the disease, and on the nature of the change that the nerve elements undergo, rather than on the pathological character of the primary lesion. In most diseases that we term "organic," the primary morbid process is outside the nerve elements themselves; the changes these elements undergo are secondary, and are few in kind, while the diseases by which they are produced are numerous. In the maladies that we term "functional" (which are better conceived as "nutritional," see p. 26), there is no lesion outside the nerve elements; the change which these undergo, and on which the symptoms immediately depend, is primary. This is probably the case also in the degenerations of the brain, although the changes in the nerve elements, and those in the interstitial tissue, often seem to occur almost at the same time.

The symptoms of disease are the manifestations of disturbance of function, objective and recognizable by another person, or subjective and perceived only by the sufferer himself. The disturbance of function is of two kinds: (1) a diminution of action, which may present every degree, down to abolition, and (2) an increase of action. These two effects are often combined. Disease may lessen the possible amount of functional action, and yet render that which remains too readily excited. This is the "irritable weakness" of old writers. The increase of action caused by disease is commonly called "irritation." But irritation sometimes lessens function by the process that physiologists term "inhibition." Thus loss of function may be due to structural damage or to irritation; from the latter the loss is transient, from the former it is permanent.

Structural damage to the nerve elements is produced in various ways by organic disease of the brain. The following are the most important mechanisms: (1) Mechanical interruption or destruction, as when nerve fibres are torn across, or nerve cells are separated, by an extravasation of blood. This involves, of necessity, entire abolition of function; (2) mechanical compression, which may be sudden or slow in its production, and varies in its influence on function according to its rapidity. Very slight compression, if suddenly or rapidly induced, arrests function; on the other hand, a considerable amount of pressure produces little or no effect, if it be very slowly induced. We do not know whether pressure acts on function by a mechanical influence on the nerve elements, or by an arrest of the blood supply, due to compression of the capillaries. The difference between the effect of slow and sudden pressure suggests that its influence on the nerve elements is direct, since the effect on the capillaries must be the same in each case. Pressure does more than arrest function; it causes structural disintegration of nerve elements, and this also is greater when the pressure is rapidly produced.. (3) Arrest of the blood supply causes arrest of function, and diminution of the blood supply causes impairment of function. Here also we may trace a relation to rapidity of production, although slighter than in the case of pressure, and perhaps due to the fact that slow obstruction gives time for the establishment of a collateral circulation. The immediate arrest of function that follows a sudden arrest of blood supply is very remarkable. If the heart fails, for instance, consciousness is instantly lost. It is probable that the influence is in part mechanical, due to the sudden diminution in the pressure to which the nerve elements are normally exposed. It is probable, therefore, that a decrease, as well as an increase, of pressure interferes with function. (4) Inflammation—which, in the brain as in other tissues, involves the interstitial tissue and the proper elements of the part, as well as the blood vessels. (5) Molecular disintegration or wasting of the nerve elements. This constitutes the primary and chief element in many degenerative processes, in which it is associated with changes in the interstitial tissue. It occurs also, in rapid form, in consequence of many of the other morbid processes above described.

Some symptoms, such as local paralysis, are due to and indicate interference with the function of a definite part of the brain. They are often termed *focal* symptoms. Other symptoms, such as loss of consciousness or delirium, indicate a widespread interference with function, and are called *diffuse*. The distinction, although useful, must not be conceived as absolute. Some symptoms may be at one time diffuse, at another focal. The same terms are also applied to the morbid processes, but the two applications do not altogether correspond. Diffuse processes may cause focal symptoms, as when hemiplegia results from an inflammation extending over the whole of one hemisphere. On the other hand, a focal lesion may cause a diffuse symptom, as when a small hemorrhage produces loss of consciousness.

It is necessary, in the first place, to describe the characters of the several symptoms of organic disease of the brain. Some of these symptoms consist of disturbance of the functions of the cranial nerves, and it will be convenint to describe them in connection with the diseases of those nerves, before we consider the general rules of diagnosis and the special diseases of the brain.

MOTOR SYMPTOMS.

MOTOR PARALYSIS—HEMIPLEGIA.—Loss of voluntary power over the muscles is one of the most frequent and important effects of brain disease. A distinction is often drawn between complete and partial loss of power, and the former is termed "paralysis," the latter "paresis." The two differ only in degree, and the term "paralysis" is also applied to all grades of deficiency. The loss is due to interference with the motor centres or the motor path, which, as we have seen, passes from the central convolutions down through the centrum ovale, the internal capsule, the crusta of the cerebral peduncle and the corresponding region of the pons, to the anterior pyramid of the medulla. The disease that causes paralysis may be situated in any one of these regions. Disease elsewhere does not cause loss of motor power, unless it exerts pressure on the motor tracts. The paths from the two sides are near together in the pons, and still nearer in the medulla; hence a lesion in either of these situations may paralyze both sides of the body. Even in the pons, however, a single lesion often affects one side only, while in the crura the paths diverge, and both suffer only when they are damaged by a large tumor. In the cerebral hemispheres a single lesion always affects only one motor path. The unilateral paralysis that results from disease of the tract on one side is termed "hemiplegia." The paths decussate in the medulla, and therefore the limbs are paralyzed on the side opposite to the lesion in the brain. In the pons the motor paths, before crossing the middle line to the hypoglossal and facial nuclei, are associated with the path from the limbs, and hence a lesion anywhere above the middle of the pons usually causes also paralysis of the face and tongue, which is on the same side as the palsy of the limbs. The motor fibres diverge in the internal capsule, and still more as they pass through the white substance to the cortex, where, as we have seen, are centres, to some extent separate, for the leg, arm, face and tongue. Hence lesions of the cortex, or of the white substance just beneath it, may paralyze one of these parts without the others; but such partial palsy seldom results from a lesion in the internal capsule, because, to have so limited an action, a lesion there must be minute.

To the rule that the paralysis is on the side opposite to the cerebral lesion, apparent exceptions have been met with, in which hemiplegia was on the same side as the lesion. Morgagni suggested that in these cases the ordinary decussation of the motor tracts does not occur, and the discovery by Flechsig that there are great variations in the decussation of the pyramids in the medulla, has been regarded as confirming the hypothesis of Morgagni. But there is strong reason to believe that when the paths do not decussate in the medulla they cross lower down the spinal cord, and, if so, this explanation falls to the ground. Much more frequent than hemiplegia on the same side as the lesion, is hemiplegia without any discoverable lesion to account for it. The nature of these cases is mysterious, but most pathologists have met with instances. In the cases in which the paralysis is on the same side as the lesion it is probable that undiscovered disease existed in the other side of the brain. It is in harmony with this explanation that most of the instances of paralysis on the same side as the lesion were observed before the advance in brain pathology that has occurred during the last generation.

In a case of severe "complete" hemiplegia, the arm and leg are powerless; the face is paralyzed, chiefly in the lower part, and moves almost as well in the upper part as on the unparalyzed side; the tongue, when protruded, deviates toward the paralyzed side, being pushed over by the opposite unopposed genio-hyo-glossus. But the muscles of mastication contract equally in ordinary action, and the two sides of the thorax move equally in ordinary respiration. Some of the trunk muscles may be weakened; the patient cannot sit up. If he takes a deep breath, and brings into action the extraordinary muscles of respiration, the half of the thorax on the paralyzed side often expands less than the other. Thus some muscles are completely paralyzed, others are merely weakened, others are apparently unaffected. This difference in the condition of the muscles becomes greater after the lapse of a few months. Even when the motor path in one hemisphere is completely interrupted by the disease, and the interruption is permanent, the muscles that were merely weakened improve in strength, and in some others, that were at first totally paralyzed, a certain amount of voluntary power returns. Some movement of the leg returns, especially at the hip and knee joints; in the arm the shoulder joint can be moved a little, and often also the elbow, although the hand may remain motionless. Such recovery, the lesion persisting, occurs to some extent at all ages, but much more readily in the young than in the old. In children the recovery of the leg is so great that in time there is ability to walk long distances, in spite of the destruction of the motor path. In adults the palsy of the hand may remain almost absolute; often, however, slight power of flexing the fingers returns, and in children some recovery of movement in the hand is invariable, and it may ultimately be considerable.*

This difference in the initial palsy, and the return of power in certain muscles, are both explained by an hypothesis first stated (in a somewhat different form) by Broadbent. Some muscles of the body, as the intercostals and masseters, are used only with their fellows of the opposite side; others are often used with their fellows, but often also alone, as the zygomatici, the trapezii, and the leg muscles; others are chiefly used alone, as the muscles of the hand. Movements are represented exclusively in the opposite hemisphere in proportion as they are unilateral, in both hemispheres in proportion as they are bilateral. In other words, either hemisphere can excite the bilateral movements, but only the opposite hemisphere can excite the unilateral movements. The muscles that are sometimes used with their fellows of the opposite side and sometimes alone, which recover partially with a persistent lesion, are connected with both hemispheres, but are habitually excited from the opposite hemisphere; they recover some power because the hemisphere on the same side gains the influence over them for which structural arrangements previously existed, but had not been used.

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* Thus, in one case of severe hemiplegia in early life, the patient became able to walk long distances, and had some power of (disordered) movement in the hand. After death in adult life the opposite internal capsule was found destroyed. A large cavity occupied the whole of the central ganglia.

We do not yet know the nature of the connection between the hemisphere and the muscles on the same side. Broadbent thought it was by a connection of the lower (spinal and bulbar) centres on the two sides, which enabled the bilateral muscles to be excited together from one hemisphere. It is highly probable that such a connection does exist, and it may be actually effective in the case of the bilateral use of muscles; but such a connection cannot subserve the unilateral use of such muscles, as, for instance, the isolated movement of the leg. This implies a separate path to the lower centre; a mere connection of the lower centres would not suffice. We do not know whether this separate path is by pyramidal fibres that do not decussate (as by the anterior pyramidal tract, or by the passage of some fibres into the lateral column of the same side), or whether it is by a recrossing in the cord. We must, however, associate this relation with the fact that degeneration may be found in both lateral columns when the disease is on one side of the brain (see p. 504); but there is a difference of opinion as to the way in which this degeneration arises, whether the fibres that degenerate come down from the medulla, as is suggested by Pitres' observations on man, or whether they are due to a recrossing in the cord, as Sherrington concludes from his experiments on animals.*

But the retention of some movements and the return of others suggests another correlated fact. If bilateral movements are excited from both hemispheres, disease of one hemisphere should lessen the power on both sides. In proportion as muscles escape or recover on the side opposite to the lesion they should be weakened on the side of the lesion. As a matter of fact this is so. Most of the bilateral muscles are, however, so placed that their strength cannot be measured, but many muscles of partly bilateral use can be tested, and these are always found to be weakened on the side of the lesion. The weakening of this side was pointed out long ago by Brown-Séquard, and has been lately investigated by Pitres† and Friedländer.‡ It is greater when the lesion is in the left hemisphere than when the right is diseased. In the leg the loss is always considerable, and is indeed conspicuous, even on a cursory examination. It exists also in the trunk muscles, where it is less readily detected, and it exists also in the arm in a degree for which theory scarcely prepares us. The power of the hand is often reduced to not much more than one-half of the normal. Such a loss is not adequately explained by the hypothesis above stated, and seems to show that this hypothesis, however true and important, does not express the whole truth, and that there is some representation on the same side, even of muscles of purely unilateral use. We have seen that some power may return in the hand with a total lesion of the motor path, but the difference between the trifling movement in the paralyzed hand and the considerable weakening of the other, is greater than can be explained by any hypothesis hitherto put forward. Perhaps in the two hemispheres there is a mutual interaction and support of which we have at present no distinct conception.

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* *Journ. of Phys.*, vi, Nos. 4 and 5.

† *Arch. de Neurologie, 1882.*

‡ *Neur. Centralbl.*, *1883*, p. 241.

Some other features of hemiplegia remain to be described. Certain movements toward one side are produced by the action of muscles situated on both sides. Such, for instance, is the lateral movement of the eyes, by the external rectus of one side and the internal rectus of the other. Such, also, is the rotation of the head; the face is turned toward one side by the opposite sterno-mastoid, acting with other less important muscles of the side toward which the head is turned. In hemiplegia these movements toward the paralyzed side may be impaired. The head and eyes cannot then be turned toward the affected side; and the unopposed antagonistic muscles may even turn the head and eyes toward the unparalyzed side. This is termed "conjugate deviation of the eyes." The symptom usually passes away in the course of a few days. From this we learn two facts. First, that movements, rather than muscles, are represented in the cerebral hemispheres and are lost in disease. Secondly, that these lateral movements by muscles of both sides are represented in both hemispheres, but that in a normal state they are chiefly effected by the opposite hemisphere. When this is diseased they are impaired, until the hemisphere on the same side has acquired functional power over them through mechanisms before existing but unused.

A sudden cerebral lesion, such as causes hemiplegia, is often attended by loss of consciousness. We are then deprived of the direct evidence of loss of voluntary power, and a slight degree of weakness cannot be detected. But the existence of considerable paralysis can often be ascertained. Sometimes a pinch will cause a movement of the sound limb, but not of that which is paralyzed. There is greater flaccidity of the paralyzed limbs. The face is smoother on the paralyzed side. The lips are less closely approximated on that side, and the paralyzed cheek may flap with respiration. The passive mobility of the limbs is greater than on the other side, and, if raised and allowed to fall, the paralyzed arm falls like a dead weight, influenced only by gravitation, while the other arm, although it also falls, does so less suddenly. We are also helped in this condition by the state of reflex action, which is often, though not always, diminished or abolished. This is best seen in the abdominal, cremasteric, and plantar reflexes, when these are compared with the corresponding actions on the opposite side. Moreover, during the period of unconsciousness, the conjugate deviation of the head and eyes is often distinct; if the head be placed in the mid position, the deviation slowly returns. A slight degree of ptosis may also sometimes be noticed on the paralyzed side.

If the patient is conscious, the power of voluntary movement can be directly tested, and if the paralysis is not absolute, the several movements of each joint should be separately examined, and the force with which passive movement can be resisted should be ascertained. The patient is almost always able to close the eye, but often a strong contraction of the orbicularis is less on the paralyzed side than on the other. When there is no difference in the strength of contraction of the orbiculares palpebrarum, the patient, if previously able to shut the eye on the hemiplegic side without the other, is usually no longer able to do so. In the early stage the patient may be unable even to close the eye on the hemiplegic side. Sometimes there is even slight weakness of the frontalis, and the forehead may be smoother on the paralyzed side than on the other. In the lower part of the face there may be a marked difference in the degree of affection of the voluntary, emotional, and associated movements (Figs. 230–232). The former is tested by making the patient raise the upper lip, and move the mouth from side to side; emotional movement is seen in a smile, and the associated movement is obtained by causing the patient to grasp strongly with the sound hand. This difference is occasionally of considerable diagnostic importance. In some cases, in the early stage, the eye cannot be closed, and there may be a doubt whether the paralysis is due to disease of the nerve or of the motor tract in the hemisphere. In the former case all emotional movement is lost, as well as that which is voluntary. In the latter the smile may be equal on the two sides, although voluntary movement is lost on one. The movement of the tongue within the mouth should be observed, as well as its deviation on protrusion. The base of the tongue is usually higher on the paralyzed side than on the other.

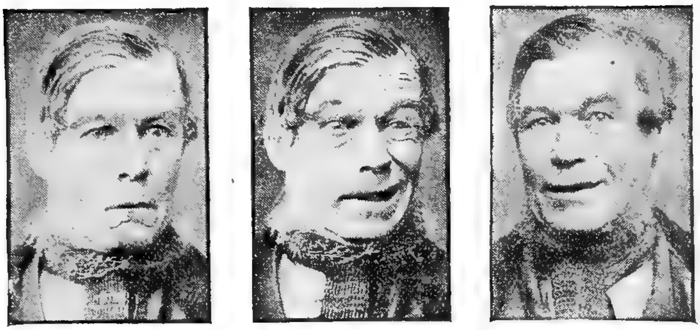
FIG. 230. FIG. 231. FIG. 232.

FIGS 230–232.—Different affection of voluntary and emotional movements of the face in a case of right hemiplegia. The appearance of the face at rest is shown in Fig. 230; an attempt to raise the upper lip on both sides in Fig. 231; a smile in Fig. 232. The last was much more nearly equal on the two sides than the figure suggests. (*From photographs.*)

If hemiplegia is complete in extent, but imperfect in degree, the condition is similar to that of perfect hemiplegia which is in process of recovery. The loss of power is greater in the arm than in the leg, and greater toward the extremity of each limb than in the part nearest the trunk. The muscles moving the hand are weaker than those of the shoulder, and in the leg the most affected movements are those of the foot, especially flexion of the ankle. Hence, in walking, the foot is swung round (or, less commonly, the knee is unduly flexed) in order to prevent the contact of the toes with the ground when the foot is brought forward. There is often more power of using the leg with the other, as in walking or standing, than there is in the separate movements of the limb.

To the rule that the leg recovers before the arm, and the proximal parts of the limbs before the distal, apparent exceptions are sometimes met with. The arm may recover earlier and more than the leg, and sometimes the extremities recover before the upper parts of the limbs. These are always cases of imperfect hemiplegia, in which the damage to the motor path or centres is unequal, and the less affected parts recover first. In comparing the two modes of recovery, we are or may be comparing things in their nature unlike—the return of power due to compensation by the other hemisphere, with that due to the recovery of the damaged hemisphere.

Varieties of hemiplegia depend, for the most part, on differences in the seat of the lesion. These differences may be spoken of as transverse and vertical. The transverse variations determine differences in the distribution of the palsy on the affected side, and are greater the higher the lesion is in the motor path, reaching their maximum at the cortex. The vertical differences determine chiefly the associations of the hemiplegia, although, as we have just seen, they are not without influence on its distribution, on account of the different compactness of the path, and consequent differences in the scope for transverse variations in the lesion. Where the path occupies a small transverse area, a single lesion can hardly fail to influence the whole of it.

The varieties determined by the relative amount of damage to the several parts of the motor path or centres depend on the distribution of the palsy, whether this is "complete" from a total lesion, or "incomplete" from a partial lesion. The latter occurs chiefly when the disease is in the white substance of the hemisphere or in the cortex. Such limited palsy is termed "monoplegia."* The arm only, the leg only, or the face and tongue may be affected. The two last named are usually involved together in consequence of the proximity of their centres and path. These varieties have been termed *brachial*, *crural*, and *facio-lingual monoplegia*. The face and arm may also suffer together (brachio-facial monoplegia). The paralysis is never absolute in these cases of limited range, and the condition of the limb, as regards loss of power, usually resembles that of hemiplegia which is in process of recovery. The coarse movements of the upper part of the limb are preserved, while the movements of the hand may be lost.

The second class of varieties depend on the vertical position of the lesion. If this is in the medulla, the face escapes altogether or only the lips are involved. Disease at the level of the hypoglossal, may, by damaging the fibres of this nerve, cause its paralysis on the same side as the lesion, and a deviation of the tongue toward the unparalyzed side. Theoretically, hemiplegia without paralysis of face or tongue may result from a lesion of one pyramid below the hypoglossal nuclei, but such cases are extremely rare.

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* Strictly, "monoplegia" ought to be used to designate double hemiplegia, but this is commonly called diplegia. We have thus the anomalous nomenclature (too firmly rooted to be disturbed) that two half palsies make a double palsy, and that a one palsy is part of a half palsy!

In the lower part of the pons the facial nerve is damaged on the side of the lesion, and therefore on the side opposite to the palsy of the limbs. In the upper part of the pons the fibres from the opposite facial nucleus have joined the motor tract, and the hemiplegia does not differ from that produced by disease of the internal capsule unless the fifth nerve is involved on the side opposite to the limbs. If the lesion is in the crus, the fibres of the third nerve may be damaged on the side of the lesion, and there is, in addition to the hemiplegia, palsy of the third nerve opposite to the hemiplegia, usually complete, sometimes incomplete, and affecting especially the levator. The impairment of sensation that often accompanies hemiplegia will be described in a separate section.

Associated movements sometimes occur in the paralyzed parts. A strong effort to grasp with the unaffected hand will sometimes cause a movement in the paralyzed hand. More common still is a movement of the paralyzed arm during the act of yawning or stretching, or even during coughing. When partial recovery of power has taken place, voluntary movements with the affected limb may cause an associated movement of the unaffected limb. The explanation of these associated movements is doubtless to be found in the connections of the subsidiary centres through which they are produced.

The diminution in the superficial reflex action already mentioned is almost invariable at first. Sometimes it passes away in the course of weeks or months; in other cases it persists. In rare cases an increase in the superficial reflex action has been met with. This difference bears no relation to the change in sensibility. We do not yet know the significance of the difference. Some physiological researches suggest that there is a cerebral centre which normally restrains the activity of the spinal reflex centres, and that this is, in its turn, controlled by a higher centre, the influence of which may be cut off by the lesion. In consequence, the reflex inhibitory centre is unrestrained, and lowers reflex action on the paralyzed side. This loss of reflex action is of diagnostic importance, because it often occurs when the motor paralysis is slight.

In rare cases, considerable movements of the limbs, even of the arm, similar to the associated movements, may be produced by cutaneous stimulation. The effect may not be confined to the paralyzed side. Thus, Nothnagel mentions a case in which a moderate pinch of the hemiplegic arm caused muscular contractions in the other arm; on a stronger pinch the contractions extended to the other leg, and, on a still stronger pinch, to the leg also on the same (hemiplegic) side, while the arm that was pinched always remained still.*

Muscular Rigidity.—At some period the paralyzed limbs usually present rigidity of the muscles, which stiffens the limbs in a certain posture, and opposes passive movement. The attempt to overcome the rigidity by forced extension of the muscles causes pain. Todd first drew a distinction between two forms, "early" and "late" rigidity, and to these we must add two others. *Initial rigidity* comes on immediately after the onset, and often lasts for a few hours only. It is due to the irritation of the fibres by the process that interrupts their continuity, and is often absent. The *early rigidity* comes on within a few days of the onset, and lasts for a few weeks. The posture which the limbs assume is that of rest. It is apparently due to the irritation of the fibres of the motor tract by the inflammation that the lesion sets up. It is usually slight in degree, but sometimes considerable, when there is an unusual amount of irritation. If the initial rigidity is prolonged, it may pass into the early rigidity. The *late rigidity* comes on in the course of a few weeks, and lasts for months or years; in cases of enduring paralysis it may be permanent. This rigidity occurs when there is descending degeneration in the motor (pyramidal) tracts, and seems to be influenced in its degree by the irritative nature of this degeneration (Charcot). In the upper limb the position is that of adduction of the shoulder, flexion of the elbow, flexion and pronation of the wrist, and still greater flexion of the fingers, especially of the distal phalanges, by the contraction of the long flexor (Fig. 233), the interossei, which flex the metacarpo-phalangeal joints, being little affected in this form of contracture. When the wrist is passively flexed the fingers can be extended freely, but if the wrist is extended they return to the state of flexion, because extension of the wrist lengthens and flexion shortens the course of the flexor tendons (Fig. 234). Although the contracture preponderates in the flexor muscles, the extensors present also some rigidity, as passive movement readily demonstrates. Very rarely the fingers are extended at all joints (including the metacarpo-phalangeal), but in these cases there is never complete paralysis, and the rigidity also is slight. In the leg the rigidity is more nearly equal in the two groups of

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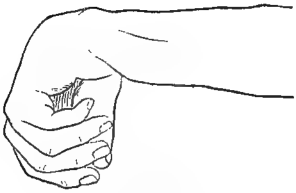
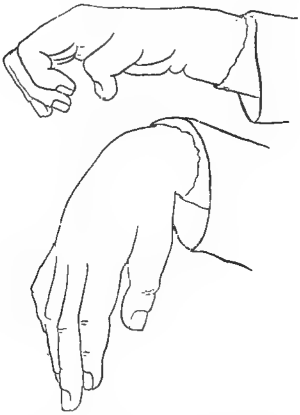
* *V. Ziemssen's Handbuch*, Bd. xi, 1, ii Auflage, p. 110.

FIG. 233.



Late rigidity in hemiplegia five months after the onset.

FIG. 234.



Late rigidity in hemiplegia, showing the flexion of the middle and distal phalanges when the wrist is extended, and their extension when flexion of the wrist shortens the course of the tendons.

muscles, and produces extensor contracture, so that the leg is straight, and the foot tends to assume the position of talipes equino-varus. The face does not participate in this form of rigidity.

The late rigidity depends on an active muscular contracture. It lessens very much during sleep, and when the limb is placed in warm water. It can also be overcome by passive extension, most readily when this is gentle and long continued. The relaxation is facilitated by rubbing the muscle. Faradization of the opponents of the most contracted muscles also lessens the rigidity. When late rigidity has continued for a considerable time, tissue changes sometimes take place in the shortened muscles, in consequence of which their passive elongation is no longer possible. Thus we must distinguish from the active late rigidity an ultimate *structural contracture.*

The rigidity, early and late, evidently depends on the over action of the spinal centres. The centres which thus overact are probably those on which normal muscular "tone" depends, and the rigidity is an excessive degree of this "tone." With it is associated an excess of the peculiar muscular irritability on which the so-called "tendon reflexes" depend, due, probably, to a muscle-reflex action (see p. 35). The excess is not usually evident until a week or ten days after the onset of the hemiplegia. In consequence of this increased irritability there is, in most cases of hemiplegia with persistent weakness, an excessive knee jerk, and a foot clonus can generally be obtained. Sometimes a rectus clonus can be elicited. The same conditions may be observed in the arm. A tap on the front of the wrist causes a contraction in the flexors of the fingers; one on the radius produces a contraction in the biceps, one on the ulna a contraction in the triceps, and this can be still better obtained by a tap on the tendon of the triceps just above the olecranon. A clonus can often be obtained in the flexors of the fingers by putting sudden tension on them. I have even obtained a similar clonus in the trapezius by sudden depression of the shoulder.

The increase of myotatic irritability, which depends on degenerative changes in the pyramidal tracts, comes on, as has been said, about ten days after the onset of the hemiplegia. But the myotatic irritability sometimes presents variations before the degenerative increase is established, and these are probably due to the cerebral irritation, which may increase or inhibit the action of the spinal centres on which the phenomena depend.

Immediately after the onset of hemiplegia the knee jerk may be absent, and it may return in a few hours. When it is thus lost there is perfect relaxation. More frequent than this initial loss is an early increase; a day or two after the onset the knee jerk is increased, and a foot clonus can be obtained. This early increase may or may not pass away before the degenerative excess is developed.

Muscular Irritability and Nutrition.—In cerebral disease there is rarely any considerable change in the irritability of the muscles. It may be the same as that on the unparalyzed side, or it may present a slight change, increase or diminution. The change is never considerable, and is the same to both Faradaism and Voltaism. The increase is the earlier change; a decrease is not usually present until some months after the onset. In the cases in which there is an alteration of muscular irritability there is generally wasting of the muscles, sometimes considerable, but never reaching the degree seen in progressive muscular atrophy. Both the wasting and the alteration in irritability are probably due to the irritative character of the descending degeneration in the pyramidal tracts. Although the degeneration does not spread to the motor nerve cells as a destructive process, it seems, when irritative in character, to influence their nutrition and the nutrition of the motor nerve fibres proceeding from them, and, through these, that of the muscles.

Vasomotor and Trophic Changes.—The paralyzed limbs (1) may present no vascular changes; (2) they may be warmer by half a degree or so than those of the opposite side; (3) they may be colder, pale, or livid. Eulenberg and Landois have ascertained that the cortex contains, near the motor centres for the limbs, other centres that influence the vascular state of the limbs. Irritation of these centres causes pallor and coldness, while hyperæmia and increased warmth are probably the result of a loss of the central influence, and may be due to disease of the cortex or of the path from it, which seems to pass in the posterior limb of the internal capsule. The elevation of temperature may be from 2° to 1.5° F. above that of the unparalyzed side. It is often uniformly raised for ten days or a fortnight, and then presents variations, sometimes an elevation, sometimes a depression. The elevation of temperature may be accompanied by redness of the skin; when the temperature falls the vessels may remain distended, and the limb then has a bluish-red aspect. Occasionally there is increased sweating. There may be some œdema of the subcutaneous tissue, most marked toward the extremity of the limb. This, in slight degree, is very common, and may come on at the end of one or two days and persist for many weeks. It is especially great when there is kidney disease, and may be very marked in the paralyzed limb and absent elsewhere. In some cases there is a tendency to acute trophic changes. Slight pressure, continued for a few hours, may raise a blister, and even cause the skin to slough; sometimes, indeed, the vesication and sloughing seem spontaneous. Bed sores thus readily occur. Their most common seat in hemiplegia is the gluteal region, while in paraplegia it is usually over the sacrum. The skin over the trochanter, malleolus, and heel is also apt to slough, manifestly because these points are the most exposed to pressure. A marked tendency to such sloughing is of bad prognostic significance.

More chronic trophic changes are sometimes observed. Disseminated neuritis, with considerable thickening of the nerves at certain spots, has been met with (Leubuscher), and is said to be independent of degeneration in the pyramidal tracts (Charcot). Inflammation of the larger joints, attended with redness and swelling, has been occasionally observed during the latter part of the period of inflammatory reaction, one to four weeks after the onset. It is limited to the hemiplegic side and is evidently analogous to the similar inflammation that is secondary to acute myelitis. This condition is met with more frequently in cases of cerebral softening than of cerebral hemorrhage.

In some cases there are symptoms that suggest paralysis of the cervical sympathetic (Nothnagel). These are contraction of the pupil, slight drooping of the eyelid (not due to paralysis of the levator, because the eyelid can be raised as high as on the paralyzed side), narrowing of the palpebral fissure, retraction of the eyeball, and an increased secretion of tears and of nasal mucus. The pulse on the paralyzed side is often smaller than on the other, and sphygmographic tracings are said to show a lessened contractility of the wall of the vessel (Wolff and Eulenberg).

Occasionally, the nutrition of the nails is changed, and they become more curved and brittle. Rarely there is an increase in the growth of hair and a thickening of the skin. When hemiplegia comes on in childhood the growth of the limbs is usually retarded, and they never attain the normal size. The difference is more marked in the upper than in the lower limbs, and affects all parts, including the bones; in the scapula it is often very conspicuous.

Disorders of Movement after Hemiplegia.—Besides the rigidity already mentioned, the affected limbs sometimes present other forms of spasm—tremor, rhythmical movements, and especially irregular movements, occasionally quick, more often slow. These occur only when there is some return of voluntary power, not when the paralysis remains absolute. The spasm is especially conspicuous on voluntary movement, although it may also occur, in slighter degree, when the limbs are at rest. All forms are more frequent and more considerable in the arm than in the leg. The spasm does not manifest itself until some months after the onset of the hemiplegia, coming on with the return of voluntary power; once established, it usually persists, although it may lessen somewhat in the course of years.

Simple tremor is not common. It is usually fine, and occurs chiefly during movement, rarely when the limb is at rest. It is confined to the arm. Rhythmical movements are also rare, and likewise occur only in the upper limb. There may be alternate flexion or extension of the fingers or wrist, or pronation and supination of the hand, continuous or only on movement.

The most common form is that in which there is tonic spasm, slowly varying in relative degree in different muscles, and thus causing slow, irregular movements, chiefly conspicuous in the hand, and slow, irregular incoördination. From this character it may be conveniently termed "mobile spasm." It is commonly conjoined with more or less permanent rigidity, which tends to fix the limb in a certain posture. This fixed rigidity is generally proportioned to the amount of paralysis. If the loss of power is slight, and there is no fixed rigidity, the movements may extend in range. In some cases the movements are quick instead of slow. In rare cases, this condition comes on without preceding hemiplegia; such a condition was called by

Hammond "athetosis" (= without fixed position). Such primary spasm is similar in characters to that which may succeed hemiplegia, and every gradation is met with between such cases of extensive movement without rigidity and the more frequent cases in which the movements are slighter, and are combined with fixed contracture. The condition has also been termed "spastic hemiplegia" and "post-hemiplegic chorea." This latter term is objectionable, because the condition has nothing to do with chorea, and, except in the very rare cases of quick movement, the resemblance to true chorea is remote.

In this condition the upper arm is usually adducted; the elbow joint is commonly flexed, sometimes extended, and occasionally the extended arm is carried backward by the spasm and rotated inward, so that the hand is held against the lumbar region with the palm turned outward. In the same patient, the arm may be sometimes extended and sometimes flexed. If there is fixed rigidity this usually affects chiefly the flexors of the wrist, so that this joint is bent at a right angle. If there is no fixed rigidity, the wrist is sometimes flexed, sometimes over-extended. In the hand (Figs. 235 and 236) the spasm has almost always a peculiar distribution. It affects especially the interossei and lumbricales, which flex the metacarpo-phalangeal and extend the phalangeal joints; sometimes it affects also the long extensor, but never the long flexors of the fingers, which are the especial seat of the ordinary "late rigidity." Hence, the hand is usually in the "interosseal position," (such as the interossei produce) with flexion of the proximal and extension of the middle and distal phalanges, but the amount

FIG. 235.

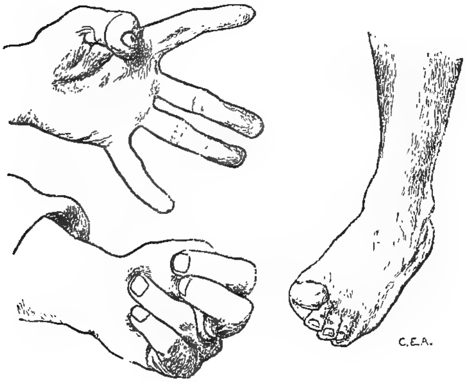


FIG. 236.

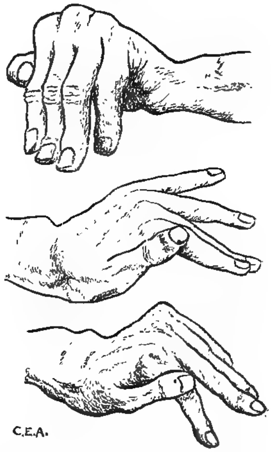
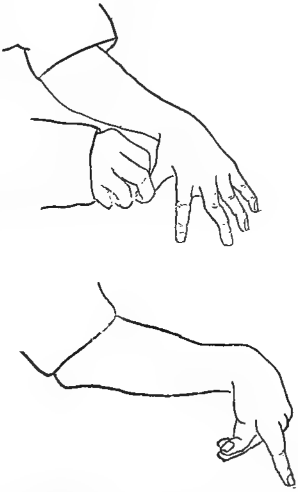


FIG. 235.—Continuous mobile spasm (athetosis) after slight hemiplegia. (Patient aged 24; onset of hemiplegia at 23; of spasm four months later. Previous syphilis.) The hand was in continuous movement between the two positions shown. The foot was habitually inverted, and the great toe often over-extended.

FIG. 236.—Post-hemiplegic mobile spasm. (The hemiplegia came on at 23, probably from embolism, and was severe. Spasm commenced a year after the onset, at the same time as slight return of voluntary power. The figures show some of the postures of the hand six years after the onset, especially the predominant spasm of the interossei.)

of spasm varies from time to time in the muscles of the different digits; now one is slowly extended, then another; and the thumb is sometimes pressed against the forefinger, sometimes over-extended. At one time all the fingers may be extended and separated, then one or another is adducted or flexed, the wrist being sometimes flexed and sometimes extended, and the slow, irregular changes in position suggest the movements of the tentacles of a cuttle-fish. The constant spasm of the interossei may, after a time, so affect the middle and distal phalangeal articulations that they become over-extended and undergo a subluxation, so that the head of the nearer phalanx is prominent on the palmar aspect of the finger.* The exercise of the muscles by the continuous activity may lead to their overgrowth, so that the affected arm may be larger in circumference than the other, and this even in cases in which growth has been arrested and the limb is shorter than the other. When the hand is at perfect rest, the movements become slighter and often cease, but they are renewed by any attempt at voluntary action, and even by attention. In more severe cases, such as those to which the term "athetosis" is strictly applicable, the movements continue during rest, and may even persist during sleep. On the other hand, in many cases the spontaneous movements are trifling, and close observation may be necessary to detect them. In all cases voluntary movement is irregular and difficult, the spasm excited by the attempt producing a peculiar slow incoördination.

Fig. 237.



Mobile and fixed spasm in hand after hemiplegia in early life. In the upper figure the thumb is held so as to show the over-extension of the middle phalanges of the fingers. As soon as the thumb was released, the hand assumed the position shown in the lower figure.

The leg is always affected in much slighter degree than the arm. The spasm is extensor, and in the foot tends to cause elevation of the heel and inversion of the foot—talipes equinovarus. Some fixed rigidity in these muscles usually coexists. There is often also a marked over-extension of the great toe. The mobile spasm in the leg is rarely spontaneous, but is usually at once excited by movement, especially by attempts to walk.

The face, in these cases, presents no spontaneous spasm, but the same spasmodic tendency is seen here also, and causes a slight movement to be greater in degree on the affected side than on the other. In a slight smile, for instance, the angle of the mouth is drawn outward more, and often sooner, on this side than on the other, although there may be a manifest deficiency of power on a stronger movement. The slight over action in the face is often a most valuable diagnostic indication in cases of old infantile hemiplegia, of which little trace may remain elsewhere.

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* This occurs chiefly when the spasm comes on early in life. In children over-extension of the phalangeal joints can readily be produced, and is kept up and increased by the spasm.

In about half the cases in which this condition follows hemiplegia in the adult, there is impaired sensation on the affected side, hemianæsthesia. But in the cases which date from childhood, sensation is always normal. (A lesion of the brain in childhood seldom, if ever, causes persistent loss of sensibility.)

It has been mentioned that movement intensifies the spasm and is disordered by it, being, in consequence, rendered ataxic or incoördinate. In some cases there is no spontaneous spasm, but simply incoördination of voluntary movement, varying in degree from mere awkwardness or unsteadiness to wild disorder, and there may be jerking incoördination resembling that seen in disseminated sclerosis.

Regarding the nature and position of the disease which causes these disorders of movement we have as yet but little pathological evidence. The symptom is observed after recovery from the paralysis, and hence in patients who, for the most part, live on and pass out of observation. But two etiological facts are of great significance. The first is that these disorders of movement are far more frequent after cerebral softening from vascular occlusion than after cerebral hemorrhage.* The second is that they follow hemiplegia far more frequently when this comes on in infancy or childhood than when it comes on in adult life. The probable significance of the first fact is that, in softening, slight damage to the cerebral tissue is more extensive than the actual destruction, and the spontaneous spasm must be referred to the over action of gray matter, which is in a state of altered nutrition and function. Hence we can understand the occurrence of this symptom from a lesion which involves extensive slighter damage. The significance of the second fact—the frequency with which the condition follows infantile hemiplegia—is probably the greater facility with which the growing and developing nerve cells recover, and their greater susceptibility to disorder of function when their development is perverted.

Regarding the seat of the disease which gives rise to these symptoms, the facts at present recorded are too few to permit any accurate generalization. It is, however, probable that a distinction must be drawn between the cases in which the lesion occurs in childhood and in adult life. In the latter the condition is rare, and in most of the recorded cases the disease has been situated either in or outside the optic thalamus; in several it involved the posterior part of the internal capsule, a fact which explains the occasional association of hemianæsthesia. The disease of the internal capsule has usually been incomplete, not involving the whole thickness of the capsule, and it has always involved the gray matter either of the lenticular nucleus or the caudate nucleus. Since the optic thalamus is not in the motor path, disease limited to this must produce the symptom indirectly by disturbing the function of the motor cortex. It is certain, moreover, that the symptom may result from disease which is limited to the cortex.* Regarding the cases that date from childhood, we have very few facts. In those that have been examined, the disease has varied much in position and extent. This, and the frequency of its occurrence, suggest that, in these cases, the symptom is due to the quality of the lesion (impairment of the nutrition of growing motor nerve cells) rather than to its site.

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* See "Athetosis," etc., *Med.-Chir. Trans.*, *1876*.

The relation between these various forms of post-hemiplegic diseases of movement is shown in the following table.

POST-HEMIPLEGIC DISORDERS OF MOVEMENT.†

Quick, clonic spasm, of intermitting type	Regular (continuous, or on movement) .	Tremor	Fine. Coarse.	
		Certain regular movements, due to interossei, pronators, etc.		
	Irregular (continuous or on movement) .	Choreoid Jerking	Continuous spasm, or incoordination of movement.	
Slow, mobile spasm, of remitting type.	Continuous = "Athetosis."			
	On movement = Slow, cramp-like incoördination.	"Spastic contracture" of hemiplegic children.		
Tonic spasm, varying . . . . . . Fixed rigidity, unvarying . .	Of interossei conspicuous			
	Of flexor longus digitorum conspicuous = late rigidity.			

CONVULSIONS.—Convulsions are frequent in organic brain disease. They are produced in two ways: (1) By active irritation, as in meningitis, growing tumors, encephalitis, and in acute cerebral lesions, hemorrhage or softening. (2) By altered nutrition of gray matter around a stationary lesion; in consequence of the alteration the gray matter possesses deficient stability and "discharges." By each mechanism convulsions are produced most readily and most frequently when the disease is at the cortex, and the second mechanism is practically confined to cortical lesions. Stationary lesions scarcely ever cause convulsions, unless they are situated in or near the motor convolutions. On the other hand, irritating disease often causes convulsions when it is at a distance from the motor region, both when it is in the cortex itself or in the deeper parts of the brain, even in the pons. Convulsions also result from general increase of intracranial pressure.

The convulsions that result from organic brain disease may be general, and similar to those of idiopathic epilepsy, consisting first of tonic and then of clonic spasm, with sudden loss of consciousness. A full description of their character is unnecessary here, since they are described in the chapter on Epilepsy. The fits are usually of this type when they are due to a diffuse morbid process, as meningitis, or to disease away from the motor region, and also whenever the discharge is intense. But in most cases in which the convulsions are due to focal disease, especially when this is in or near the motor region of the cortex, the convulsion is of a different type, distinct in at least some of the attacks. The discharge commences at the seat of irritation, and spreads thence in the motor region. Hence the onset of the convulsion is deliberate and local; often consciousness is not lost until the cerebral discharge has progressed beyond its point of commencement. The local commencement may be seen by an observer, and often the patient is aware of the commencing fit, and of the manner in which it commences. The local commencement of the convulsion is usually in the side of the face, in the arm, or in the leg. If slight, the convulsion may be limited to the part in which it commences (partial convulsion), and consciousness may not be lost at all. If more severe, the convulsion extends to all parts of the side on which it commences, and consciousness may or may not be lost. If still more severe, it may extend to the opposite side, and then consciousness is almost always lost. But there is a further distinction to be made in the mode of affection of the muscles of the two sides. These are involved in different degree and order, according to their unilateral or bilateral use (Broadbent, Hughlings Jackson) and corresponding innervation from one or both hemispheres (see p. 511). We have seen that, in proportion as movements are bilateral, those on the paralyzed side escape in hemiplegia. In the same proportion those on the sound side are involved in convulsion. The bilateral representation which permits their escape in one case insures their involvement in the other. Hence we may have three degrees of extension: (1) In a slight unilateral fit the unilateral muscles of one side are alone involved; the fit affects only the arm and leg. (2) In a more severe fit, in addition to the unilateral muscles, the bilateral muscles of both sides are involved, *e. g.*, both sides of the thorax, and sometimes the opposite leg, the arm remaining free. (3) In a still more severe fit the opposite arm is also involved.

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* A conclusive case has been published by Demange, *Revue de Médecine*, May, 1883, Case ii, p. 375.

† From "Athetosis and Post-hemiplegic Disorders of Movement," *Med.-Chir. Trans.*, *1876*, p. 291.

Yet another distinction is necessary. There are muscles on the two sides which, together, have a unilateral action. These muscles, as we have seen (p. 512) are innervated from the cerebral hemisphere according to their action, *e. g.*, the muscles which move the head and eyes to the right are innervated from the left hemisphere. We have also seen that in unilateral paralysis the movement is lost by an impairment of the muscles on both sides. In a unilateral convulsion the spasm has a corresponding distribution; the head and eyes are usually turned toward the side convulsed, by these contra-lateral muscles, as they may be termed. In a fit which is from the first general, it is common to have the convulsion greater on one side than on the other, with a corresponding deviation of the head and eyes. In a fit in which one side only is at first affected, the other side is often affected later, while the convulsion on the first side is lessening. The passage to the second arm (probably due to the extension of the discharge to the opposite hemisphere) is attended by a corresponding movement of the head and eyes, which, turned at the onset toward the side first affected, are afterward directed toward the second side, when this becomes involved in spasm.

Convulsions begin locally when the disease irritates a part of the brain in which the centres for the different parts are separate, and chiefly when the disease is in or near the cerebral cortex, in the central (motor) convolutions or paracentral lobule. If the centre is destroyed, convulsions rarely begin in the part, which is then permanently paralyzed. Hence convulsions occur chiefly from disease which partially damages the centres, or which is in their neighborhood, and irritates them. When the irritation is in the highest part of these convolutions, the convulsion usually begins at the foot; when in the middle, at the hand; when in the lower part, at the face, and is then commonly associated with temporary loss of speech. It is probable that a similar differentiation of spasm may occur in limited disease of the white substance or internal capsule, but it is very uncommon for convulsions to result from disease in this situation. When the convulsion begins in one limb, it may commence by a motion or a sensation, according as the motor or sensory elements lead in the discharge. We do not yet know whether this difference has a localizing value. Probably it has not. Disease of the central convolutions may cause convulsions commencing by a sensory aura. We have seen that these convolutions have sensory as well as motor functions. We must at present regard the commencement of the fit by a sensation in the arm, leg or face, as of the same diagnostic value as the commencement by motor spasm.

In idiopathic epilepsy special sense auræ are not infrequent, but in organic brain disease they are rare. They signify that the disease is adjacent to the special sense centres in the cortex. Thus I have mentioned elsewhere* a case in which a tumor of the occipital lobe caused a visual aura, and another case in which an auditory aura preceded convulsions due to a tumor which commenced beneath the first temporal convolution.† In a third case lately under my care, a tumor in the middle of the posterior limb of the fissure of Sylvius, invading the first temporal convolution, caused convulsions on the opposite side, which also began with an auditory aura (see the chapter on Intracranial Tumors).

The convulsions that attend the onset of a vascular lesion are usually general, but commence on the side which is subsequently paralyzed, and they may be confined to this side, especially in the case of surface lesions, *e. g.*, thrombosis in a superficial vein. In cortical disease they may continue after hemiplegia has become established; in deeper lesions they usually cease after the onset of the paralysis, and if they continue they usually affect only the unparalyzed side. The late post-hemiplegic convulsions usually affect only, or chiefly, the paralyzed side.

Unilateral or local convulsion often leaves behind it transient weakness in the part convulsed, lasting for a few hours and then passing away (post-convulsive paralysis). A severe fit probably produces this weakness by exhausting the nerve elements (Robertson, Hughlings Jackson). But similar weakness often follows or accompanies very slight fits, especially those in which the first (and sometimes the sole) discharge is sensory, and it is then probably of inhibitory nature.‡ If fits succeed each other with great frequency for many days, this paralysis may persist during the brief intervals, and be very considerable in degree, but pass away rapidly after the attacks have ceased.

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* "Epilepsy," 1881, p. 68.

† Ib., p. 70.

‡ Ib., p. 98.

The great characteristic, therefore, of the convulsions of organic brain disease is their local commencement. A local fit, *e. g.*, limited to one limb, and local commencement of a unilateral or general fit, have the same significance. The difference is of degree only. Each shows that the discharge in the brain begins locally, and indicates a local change causing a persistent instability at the spot. But local commencement, while it proves local changes of nutrition, does not prove that the disease is what is called "organic," *i. e.*, such as can be detected by the naked eye, or even by the microscope. Such local commencement is sometimes met with in idiopathic epilepsy, but it is very rare in this form, in which the common onset is by a general or a visceral aura, or by initial loss of consciousness. The local commencement, therefore, *suggests* organic disease, just as the visceral aura (as a sensation at the epigastrium), or general convulsion without any warning, *suggests* idiopathic epilepsy. If the local fits are very slight and frequent, the probability that there is organic disease is very great, because there must be an excessive degree of local instability, unlikely to result in idiopathic epilepsy, in which the morbid state is generally widely distributed through the brain, and the instability is seldom confined to one part. In all cases, therefore, in which fits begin locally, a careful search must be made for any other indication of organic disease.

Tetanic attacks have occasionally, although rarely, been produced by disease of the cerebellum, or by disease that exerts pressure under the tentorium. They may last for hours, and, in the rigidity of the extensors of the spine, bending backward of the head, and closure of the jaws, may closely resemble paroxysms of traumatic tetanus. It is not at present known whether they originate from the cerebellum or from the pons. Forced movements of the trunk, as a tendency to rotation, are extremely rare, and their significance is mentioned in the section on Localization.

Hysteroid convulsions may occur in various organic diseases of the brain, the result of the general disturbance of the cerebral function. Thus I have known them to occur in many cases of tumor of the brain, in meningitis, and even at the onset of embolic hemiplegia. Their chief importance is due to the readiness with which they may mislead in diagnosis. The symptoms of hysteria should never prevent a careful search for any indications of organic disease, and do not in any degree lessen the significance of the latter, if such indications can be found.

SENSORY SYMPTOMS.

LOSS OF SENSATION—HEMIANÆSTHESIA.—Disease of the brain may impair or destroy sensation, both in the skin, muscles, etc., and also in the organs of special sense. The affection, like that of motion, is commonly unilateral, the side affected being that opposite to the cerebral lesion. It usually depends on damage to the fibres which conduct sensation, and which probably pass through the tegmentum of the pons, and certainly in that of the crus, and in the posterior third of the hinder limb of the internal capsule, between the extremities of the optic thalamus and lenticular nucleus, and thence radiate to the central and parietal cortex. Loss of sensation is sometimes due to disease of the cortex itself, but complete hemianæsthesia is rare from such disease, because, to produce it, a lesion must be very extensive. Some of the sensory fibres are probably connected with the optic thalamus, perhaps, also, with the lenticular nucleus, but the nature and functional significance of this connection are not yet understood; and it is not probable that the activity of these ganglia influences consciousness, or that their disease causes any loss of sensation. Moreover, outside the posterior portion of the optic thalamus, the coronal fibres pass from the optic nerves by the posterior extremity of this sensory tract, and then leave it to radiate to the occipital lobes. By this sensory portion of the capsule also pass fibres from the other nerves of special sense. These special sense tracts have likewise undergone decussation, although that of the optic nerve is incomplete. Hence disease here may cause loss of the special senses, as well as of common sensibility on the opposite side, the affection of vision being hemianopia. To this region, as we have seen (p. 478), the name "sensory crossway" has been given by Charcot.

Thus in the internal capsule, a common seat of disease, the paths for motion and sensation are separate, but contiguous. It is common for a lesion to damage one much and the other but little or not at all. Hence it is common, on the one hand, to have hemiplegia without, or with only slight, impairment of sensation; and, on the other hand, to have hemianæsthesia with but little motor weakness. But an extensive lesion may damage both. Sensory fibres seem to go to the motor cortex, and it is common for disease there to cause some impairment of sensation, always most marked in the extremity of the limbs, and in monoplegia confined to the extremity of the limb that is weakened.*

In ascertaining the condition of sensation, it is necessary to examine separately the various forms of sensibility. The method of making this examination and the precautions to be taken have been already described (p. 30).

Besides the ordinary forms of sensory loss there is one to which much attention has been directed. The patient may be unconscious of the position of a limb, on active or passive movement. In brain disease that causes this defect, the power of active movement is usually lost, but if an extremity, *e. g.*, the hand, is placed in a given posture, and the patient is told to imitate this posture with the other hand, he may be so far wrong as to show that he has a very imperfect perception of the posture, and this even when there is no demonstrable impairment of cutaneous sensibility. The hand and fingers should be grasped firmly by the observer, so that the direction of pressure may not *suggest* the posture. The loss is regarded by Munk as a loss of the processes on which "conception of posture" depend, processes that are, in effect, the result of sensory (cutaneous and muscular) impressions. The loss is commonly supposed to be due to cortical disease, but it may, as I have seen, be very distinct when the disease is in the central ganglia. Its actual significance has yet to be established by clinical and pathological observation. This may be said also of the loss of other sensory "conceptions" as distinguished from mere sensations. Thus, when tactile sensibility, tested in the ordinary way, seems to be normal, the patient may be unable to recognize the nature of objects in contact with the skin, although they are at once recognized when placed on the unaffected side.

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* See the references on p. 461; also Petrina, *Prager Zeitschr. f. Heilk.*, *1881*, ii, No. 5.

It will be seen, from what has been already said, that loss of cutaneous sensibility may be chiefly on the limbs, and especially on the extremities of the limbs, or it may involve the whole of one side, including the trunk and the head. It is to the latter that the term "hemianæsthesia" is generally applied. The loss often extends up to the middle line, and exists on the mucous membranes as well as on the skin. But it is not always thus complete; it may be more considerable in some parts than in others, and may even be unequally distributed over different regions of the trunk. Nor does it involve equally all forms of sensibility; either touch or pain may be chiefly affected. It is often associated with impairment of the special senses, because, as we have seen, the paths of special and cutaneous sensibility are contiguous, and their cortical centres probably occupy adjacent regions in the outer surface of the cerebral hemisphere. In these cases vision may be impaired either as "hemianopia," or "crossed amblyopia," the significance of which has been mentioned in the account of the structure and functions of the brain; their characters will be described in the account of affections of sight.

Crossed anæsthesia of limbs and face occurs only in disease of the upper part of the pons, affecting the fibres of the fifth nerve on one side and the path from the limbs on the other. Bilateral anæsthesia, affecting the limbs on both sides, may occur from disease of the pons, but is seldom complete.

Sensory irritation, pain in the limbs, is sometimes considerable when the disease involves the sensory tract and imperfectly destroys it. In one case of partial hemiplegia, in which pains in the limbs were very severe during several years, there was complete hemianopia and little loss of cutaneous sensibility, so that the disease had probably destroyed the optic path at the posterior extremity of the internal capsule, and had damaged the latter sufficiently to irritate the fibres, but not to interrupt conduction. Numbness, tingling, formication, etc., are met with in similar cases, and are common at the onset of acute lesions in this situation. Similar symptoms occur also in irritating disease of the motor cortex. Tingling may attend the onset of local convulsions from disease in or near the motor convolutions, and in cases of tumor of this region the paralysis is sometimes accompanied by much pain in the limbs, increased by passive movements, and these may excite convulsive attacks. Pain in the region of the fifth nerve is frequent when there is disease of its fibres or nucleus, and is often the first symptom of such a lesion. An instance is afforded by the case illustrated in Fig. 234. The spot of softening near the nucleus of the fifth caused severe neuralgic pain in the region of the distribution of the nerve. Similar symptoms of irritation of the special senses are met with in disease of their paths or centres, especially in association with convulsive seizures (p. 525), and are further considered in the special account of the nerves concerned.

HEADACHE.—Pain in the head is a frequent symptom of organic brain disease. At the same time, it is far more common apart from such disease, as a result of what we call "functional" disturbance, of morbid blood states, or of gastric derangement, etc. Moreover, the pain of neuralgia in the wall of the cranium often resembles the pain of organic disease. Hence the mere occurrence of headache is of little significance. Its character is somewhat suggestive, and still more frequently its severity; but the chief significance of pain in the head is derived from its associations.

The pain of organic disease varies much in degree. It is sometimes intensely severe, and is also constant. Paroxysmal exacerbations occur, but the pain does not cease during the intervals, and it often prevents sleep. The latter is an important characteristic, because other forms of headache rarely keep the patient awake. The precise character of the pain varies much; it may be dull or acute, but it is almost always an actual pain. Cephalic sensations other than pain, such sensations as those of "pressure," "weight," "creeping," etc., seldom result from organic disease, while they are common in functional disorders. The pain of organic disease is almost always increased by whatever augments the blood pressure in the skull, such as effort, cough, stooping. In situation it may be general, or in the frontal or occipital regions, or in any part of one side. The disease causing the pain is sometimes in the part of the head to which the pain is referred, but there is a close correspondence between the two only in some cases of disease at or near the surface of the brain. The pain from disease beneath the tentorium is generally felt in the occiput and back of the neck. But frontal pain may be due to disease anywhere in the cerebral hemispheres, and has been known to result even from cerebellar disease.

The intracranial maladies that cause headache are for the most part active, irritating diseases, such as inflammation, tumor, abscess and the like. If active growth and irritation cease, the pain usually lessens, and may even be no longer felt, in spite of the persistence of the disease in a stationary condition. Diseases that increase intracranial pressure, without causing tissue irritation, such as internal hydrocephalus, may also cause pain, but often run an almost painless course. These facts, together with the readiness with which headache occurs apart from organic disease, make it difficult to give a satisfactory explanation of the mechanism by which the pain is caused. We do not even know in what structure the pain is really felt. The dura mater receives sensory fibres, and, when inflamed, may unquestionably be the seat of pain. Only sympathetic fibres have been traced into the pia mater, but other organs which receive only sympathetic fibres may be painful when inflamed, and acute pain attends meningitis when the dura mater is but little affected. The cerebral substance seems, under normal conditions, to be destitute of sensibility; but from this fact the assumption has perhaps been too hastily made, that it cannot be the seat of pain when diseased. It should be remembered that the normal sensibility of the peritoneum would not prepare us for the intense pain of peritonitis. In the brain, intense pain may be caused by very small lesions which do not come near the surface.

Of the associations of headache in organic disease (besides local derangement of function) vomiting and optic neuritis are especially important. The vomiting often occurs during the severer paroxysms of pain, and patients sometimes say that the pain "seems to make them vomit." The association with optic neuritis is also of great importance, but is not pathognomonic, since the two occur together in some cases of anæmia and of kidney disease.

VERTIGO.—The word means, literally, a turning, and is used to designate either an actual rotation of the patient, or a sensation of rotation, or a sensation of movement in some direction (which may not actually amount to a sense of turning), or a correlated impression that other objects are moving. This involves a lessened perception of the relation of the individual to external objects, *i. e.*, a slight obscuration of consciousness. Hence the corresponding English word "giddiness," and still more the looser term "dizziness," are applied to a slight dulling of consciousness, not amounting to actual loss, although there is no sense of movement objective or subjective. In pure vertigo there is no actual loss of consciousness, although this may be obscured at the height of an intense attack.

Vertigo is a frequent symptom, apart from organic disease. It results from many causes, and is therefore described more fully in a later part of this volume. Like headache, it does not in itself suggest intracranial disease, and derives its significance chiefly from its associations. Of these, vomiting has not the significance which it has when associated with headache. Intense giddiness, whatever its origin, usually causes vomiting, and this does not, therefore, do more than emphasize the fact that the vertigo was severe. This symptom may be caused by disease in almost any part of the brain, and by disease of various nature. It is most common in lesions of the cerebellum and of the pons, especially at the side of the pons, involving the middle peduncle of the cerebellum. A lesion here sometimes causes not only a sensation of turning, but an actual rotation. When due to disease elsewhere, it occurs chiefly as a symptom of irritation, as part of slight "discharges." This association is seen in epilepsy, in which vertigo is frequently the earliest subjective intimation of an attack, severe or slight.

MENTAL SYMPTOMS.

The mental functions of the brain are frequently disturbed in organic disease, and their derangement chiefly depends on disturbance of the cortex. Such disturbance may, however, be produced by disease at a distance, as well as by organic changes in the convolutions themselves. With the much-disputed question of the relation of mind to brain the physician has nothing to do. It is enough for him to recognize that mental manifestations and cerebral activity invariably coincide, and that the character of cerebral processes in some way determines the character of mental processes—in some way determines mental states. The tendency of the most advanced psychology of the present day is to keep carefully distinct the two series of phenomena. In the study of diseases of the brain we are concerned only with cerebral processes. Unfortunately, however, the chief terms available are those of psychology, and we are obliged, therefore, to speak of mental processes when all that we need to speak of, and are indeed justified in speaking of, are cerebral processes. However undesirable such a confusion may be, it is practically unavoidable.

The changes which occur in mental processes as a result of organic brain disease consist for the most part in exaltation, perversion, or defect, and these are often combined. These changes may be manifested by loss of consciousness, by delirium, or by chronic mental failure, and are also seen, in a more restricted form, in cerebral affections of speech.

LOSS OF CONSCIOUSNESS.—The highest general function of the convolutions subserves consciousness, and loss of consciousness is one of the most important and most frequent symptoms of cerebral disease. The terms "conscious" and "consciousness" are, however, used in two senses: first, to signify the subjective knowledge of the occurrence of mental processes; secondly, to designate the outward manifestations of such processes. In medical language the term is chiefly employed in the latter sense. A patient is said to be "unconscious," or to have "lost consciousness," when there is no spontaneous evidence of mental action, and none can be elicited by sensory stimulation. Hence the term "insensible" is often used in the same manner. Another confusion is introduced by the frequent relative use of the words "conscious of" in the sense of cognition or knowing. Thus a delirious patient may be said to be unconscious of what is occurring around him, although he is not said to be unconscious.

Loss of consciousness may occur suddenly or gradually, and may vary in degree, as is, indeed, implied in the statement that its onset may be gradual. The variation may be in the degree of subjective consciousness, or in the external manifestations of consciousness. It is to the latter that the term "partial loss of consciousness" is commonly applied, as, for instance, in the case in which a patient seems to be asleep, but opens his eyes for a moment when spoken to, and then relapses into his former state. Such state of partial loss is sometimes termed "*stupor.*" Complete loss of consciousness, in which a patient cannot be roused, is termed "*coma,*" if it is prolonged. In stupor, the reflex action in the limbs is preserved, and may be increased, the lower centres being in an over-active state from the deficiency of cerebral control, and the patient swallows, automatically, liquids placed in the mouth. In coma, the reflex action in the limbs may be preserved, but it is often lessened or lost in the more severe degrees, the depressed condition of the highest centres being apparently propagated downward to the lower. Swallowing is possible only in the less intense degrees of coma. In severe cases, muscular tone throughout the body may give place to flaccidity, and myotatic irritability may be lost. The pupils may be widely dilated or small; in stupor they act to light; but in deep coma they are motionless, and the conjunctivæ may be touched without the occurrence of any reflex contraction of the eyelids. When the reflex action of swallowing is lost, the palate generally shares the muscular relaxation, and, moved by the respiratory current of air, causes a peculiar "stertor," which is a familiar indication of the depth of coma. Even the respiratory movements become lessened, apparently in consequence of lowered activity of the respiratory centre; they become shallow, infrequent, and sometimes present other variations, such as the "Cheyne-Stokes rhythm;" these are described elsewhere. The lessened respiratory movements do not clear the air passages of the secretion, which accumulates in the tubes (often erroneously regarded as evidence of bronchitis), and finally even in the trachea, causing the familiar "rattle" which is popularly and rightly recognized as a frequent harbinger of death.

Loss of consciousness is the result of the interference with the highest functions of the brain, those that are the most readily deranged, and it may be produced by almost any one of the morbid processes to which the brain is liable. Chronic diseases cause it chiefly when they affect a considerable area of the cortex, but it may result from sudden lesions in any part of the brain; it is then termed "apoplexy."

Loss of consciousness may be due, however, to other causes than organic disease. It is one of the most common elements of epileptic seizures, and also occurs when the action of the nerve cells is interfered with by an imperfect supply of blood (as in acute anæmia and syncope), when the renewal of the blood is hindered by mechanical congestion, and also when the blood conveys to the brain toxic material that interferes with the action of the nerve cells—material either engendered within the body (as in uræmia) or entering from without (as in various forms of poisoning).

APOPLEXY.—When coma comes on suddenly it is termed apoplexy. The word means, by its etymology, a *striking from*, and was used by the Greeks, and is still used, to signify sudden abolition of consciousness and power of motion; and thus, in popular English also, is often called *a stroke*. Cerebral hemorrhage being the most frequent cause of this condition, "hemorrhage into the brain" and "apoplexy" came to be used as synonymous expressions. Subsequently, the hemorrhage was itself spoken of as the "apoplexy," the word being thus used to designate the pathological condition causing the symptoms which it at first epitomized. Ultimately it was applied to a similar pathological state elsewhere, and thus hemorrhages into the substance of the lung, the spleen, or the retina were, and still are, termed pulmonary, splenic, or retinal "apoplexies." Such a use of the word is alike needless and inaccurate.

The chief cause of apoplexy is a sudden organic cerebral lesion, and the most effective is intracranial hemorrhage. It may also result from laceration of the brain, from simple concussion, and also from the sudden arrest of the blood supply to a part of the brain, whether by a clot brought from a distance (embolism) or formed at the spot (thrombosis). It probably results, in rare cases, from congestion of the brain, although far less frequently than is commonly supposed.* A similar condition may come on in the old without any visible lesion by which it can be caused. This has been termed "simple apoplexy." In the old the brain is shrunken, the convolutions are small, and the spaces between them are occupied by serum. Before this fact was recognized, undue importance was attached to this serum in the cases of "simple apoplexy;" it was thought to be the cause of the symptoms, and the condition was termed "serous apoplexy," a disease that has no real existence, although the word is still sometimes to be heard at inquests and to be seen on certificates of death.

The characteristic feature of apoplexy is sudden and prolonged loss of consciousness, not due to failure of the heart's action. The onset may be so sudden that the patient, without warning, falls insensible, as if "struck down" by some unseen hand. Sometimes it is attended with a convulsion. Occasionally it is slow and gradual, occupying many hours in development ("ingravescent apoplexy"). The face may be flushed or pale, but it is rarely very pale. The heart and arteries beat, often with greater force, and sometimes less frequently than normal. The condition is that of coma, already described. In a case of moderate severity, the reflex action soon returns, and the patient, after a few hours, presents some indication of returning consciousness, may make some movements, and may open his eyes when spoken to. On the other hand, in severe cases, the coma may continue and deepen in intensity, and the patient dies, usually from the interference with breathing described in the account of coma, less commonly from arrest of the action of the heart. Occasionally, death occurs at the end of an hour or two, or even less (see Cerebral Hemorrhage).

It is not often, however, that there is only this general loss of cerebral function, uniformly distributed, and gradually deepening or passing away. Much more commonly the symptoms of a local cerebral lesion are added to those of apoplexy. Such symptoms—unilateral weakness or convulsion—may precede the loss of consciousness, or they may be recognized during the attack by the indications mentioned on p. 512. As the patient recovers, these symptoms become more and more distinct, and the patient may be found to have lost the use of language.

In *ingravescent apoplexy* the commencement of the cerebral mischief may be marked by symptoms of general shock. There is commonly pain in the head, and there may be other local symptoms. In the course of some hours, or rarely a day or two, consciousness gradually becomes impaired, and coma comes on and deepens. This form of apoplexy, first described by Abercrombie, is usually due to a slowly increasing cerebral hemorrhage.

The temperature in cerebral apoplexy is at first lowered, but usually the fall is small, and is succeeded, after twelve to twenty-four hours, by a rise. Its exact course varies considerably according to the cause of the apoplexy, and will be described in the account of the several lesions. An important exception to the initial fall is presented by some cases of a sudden lesion of the pons or medulla, in which the temperature at once begins to rise, and may attain a hyperpyrexial elevation in the course of two or three hours.

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* The possibility of congestive apoplexy has been denied on theoretical grounds, but the clinical evidence of its occurrence is strong. See the chapters on "Hyperæmia."

The mechanism by which apoplexy is immediately produced has been a matter of dispute. The condition was formerly ascribed to the pressure exerted by the clot on the rest of the brain, either influencing directly the cerebral tissue, or pressing on and emptying its capillaries (Niemeyer). That such pressure is exerted by a large hemorrhage is unquestionable. That an increase of the intracranial pressure will cause loss of consciousness is also certain. A dog becomes unconscious when there is a pressure on the surface of the brain equal to a column of mercury 130 mm. high. It is highly probable that the intensity of apoplexy is due in part to this cause. But this will not explain the occurrence of the symptom in small hemorrhages, by which no general pressure is exerted, or not more than is at once relieved by the displacement of the mobile fluid which surrounds the vessels. It will not explain the occurrence of apoplexy in laceration of the brain, or the early loss of consciousness in severe hemorrhage, in which, as Jaccoud insists, it should, if merely due to pressure, be a late rather than an early symptom. Moreover, it will not explain the apoplexy which results from the sudden closure of a large vessel, a lesion that involves no increase in the intracranial pressure. There can be little doubt, from these considerations, and from the cases in which there is no recognizable brain lesion, that the increase of intracranial pressure is not the only, and perhaps not the chief, element in the production of apoplexy. The one element common to all cases of apoplexy from organic disease is the suddenness of the lesion. Hence it has been generally recognized that one mechanism of sudden apoplexy must be an arrest of function in the cortex ("inhibition" in current phraseology) by the irritation of the sudden lesion. The more suddenly the lesion is produced the more energetic will be this influence; the more gradual the lesion the slighter the inhibition. It may thus be absent at the onset of a slow hemorrhage. The increase in the intracranial pressure is effective chiefly in hemorrhage; it develops gradually, and doubtless intensifies and maintains the coma in severe cases, and is the reason why this is deeper and more prolonged in hemorrhage than in vascular occlusion. Moreover, pressure is most effective when rapidly developed. Duret has shown that compression slowly applied has to be ten times as great to produce the same effect as when it is suddenly applied. In slow hemorrhage pressure may be the chief mechanism, but the amount of hemorrhage has to be greater, and hence, when consciousness is lost in such cases, the patient rapidly passes into a state of great danger.

Although apoplexy is merely a symptom, and not an independent disease of the brain, it is convenient to consider the more important elements of the differential diagnosis of the condition. From the unconsciousness due to cardiac *syncope*, apoplexy is easily distinguished. In the former the heart's action fails, the pulse is weak and imperceptible, the face is very pale, the respiration may be sighing and irregular, reflex action is rarely abolished, and the sphincters are not relaxed.

From the several forms of *toxæmia* the diagnosis is often easy, sometimes extremely difficult. It is easy when, on the one hand, the symptoms of apoplexy are preceded or accompanied by those of a local cerebral lesion, or when, on the other hand, the direct or circumstantial evidence of poisoning is clear, or the symptoms of toxæmia unmistakable. Where there are no local symptoms, and where no guiding history is to be obtained, the diagnosis is difficult, but a correct opinion may commonly be formed by an attentive comparison of the symptoms present. There may be, as just observed, indirect evidence of toxæmia; the breath may smell of opium or alcohol; the urine may contain albumen. But albuminuria or a smell of spirits may mislead. Cerebral hemorrhage often occurs after drinking; spirit is frequently given to a person in a fit. A smell of spirit must, therefore, only be allowed weight in the absence of any evidence of cerebral mischief. So, too, albumen is always present in the urine in uræmia, but it is also frequently present in cases of cerebral hemorrhage. Alone, this evidence of Bright's disease is of little value, except there be general œdema and the patient be young; then uræmia is more probable than vascular degeneration and cerebral hemorrhage. But with other symptoms that indicate uræmic poisoning, albuminuria is conclusive. Hence the age of the patient is an important element in the diagnosis. Late life is in favor of brain disease. The history of a fall or blow on the head adds weight to other symptoms of cerebral mischief.

The character of the coma will sometimes guide. In uræmia, and commonly in alcoholism, it is less profound than in cerebral disease. The patient can be aroused, at least partially. In apoplexy, in opium poisoning, and in the most intense alcoholic poisoning, the coma may be profound. On the other hand, the patient may sometimes be roused to answer questions in cerebral hemorrhage, and still more frequently in cerebral softening. Violent struggling is strongly in favor of drink. The mode of onset is important. In apoplexy it is usually sudden; in uræmia it is usually slow. The uræmic patient becomes first drowsy, then comatose. But with convulsions uræmic coma may come on suddenly. The onset of the coma of opium and alcohol poisoning is also slow. Ingravescent apoplexy is of deliberate onset, but a profound degree of coma is quickly reached.

General convulsions at the onset exclude drunkenness, and also opium poisoning, while they favor uræmia. Apoplexy sometimes commences with a convulsion, and if the convulsion is unilateral it is strong evidence of brain disease. Rigidity of limbs or local muscular twitching during the coma is, if constant in seat, in favor of cerebral mischief; if variable in position it is in favor of uræmia. Post-epileptic coma is of course preceded by a convulsion, and should be borne in mind.

The state of the pupils is alone of little importance. Great contraction occurs in and suggests opium poisoning, but it is also present in hemorrhage into the pons Varolii. The pupils may be normal or dilated in uræmia, in alcoholic or opium poisoning, and in apoplexy. Inequality of pupils, a unilateral symptom, points to brain mischief. The retina should be examined, since the presence of albuminuric retinitis, in the absence of the signs of a localized cerebral lesion, points strongly to uræmia.

Lastly, the temperature should be noted. In uræmia there is persistent uniform depression; in cerebral lesions the initial depression is succeeded by a rise to a point above the normal. But it should be remembered that the temperature may be raised in uræmia by some local inflammation, such as pneumonia. I have known this to cause an error in diagnosis. Hence it is most important that the examination should be thorough.

The differential diagnosis of the *cause* of cerebral apoplexy will be described more fully under the heads of cerebral congestion, hemorrhage, and softening.* "Simple apoplexy" cannot be diagnosed during life, since freedom from the symptoms of a local lesion does not afford ground for inferring that there is no such lesion. The prognosis and treatment of the condition are those of the lesions causing it, and are described in the account of those diseases.

DELIRIUM.—Instead of arrest of the manifestation of mental processes, these may be perverted, and the perversion may be manifested in speech or action. This condition is termed delirium.

The characteristic of healthy mental processes is, that they are in harmony with the actual sensory impressions of the present or with the memory of those of the past. In delirium this concord is lost. Mental processes cease to correspond to reality. There are false ideas, "delusions;" or sensory images without sensory impressions, "hallucinations;" or actual sensory impressions excite erroneous sensory images, "illusions," which may or may not seem to the patient to be true.† The condition of delirium is essentially the same as that which constitutes insanity, but the term "delirium" is usually confined to the acute mental derangement that occurs as a consequence of organic brain disease, of pyrexia, of toxæmic conditions, or of inanition. In these conditions the disease of which it is a symptom is otherwise recognizable. The similar mental state which occurs apart from these conditions, and which constitutes the sole evidence of disease, is regarded as "insanity." Delirium is due to brain disease much less frequently than to toxæmic states.

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* A reader who desires a description of the general diagnosis of the cause of apoplexy will find it in my lectures on "Diagnosis of Diseases of the Brain."

† These three terms have been variously used by different writers on mental disease, and the resulting confusion has been increased by still greater variety of use in non-medical writings, and of definition in dictionaries. The term "illusion" is widely used, and is certainly needed, to express erroneous sensory conceptions in which the error depends on the nature of the sense impression itself, and is or may be recognized. Thus, the impression of relief in the stereoscopic image is an illusion. It is mainly in this sense that the word has been made by Sully the title of a book ("Illusions," *Internat. Scientific Series*). In this sense the word was used by Bacon and by Pope, and the use corresponds with the original meaning of the word, a "deception." If used in connection with morbid mental states it should be confined to false ideas and images, the falseness of which the patient recognizes; for these a word is needed, and some writers have thus limited it. "Hallucination," by its derivation, is associated with mental disease, and may conveniently be restricted, as was suggested by Esquirol, to "sensations perceived at a time when there are no appropriate external objects to excite them." This, also, is not inconsistent with old usage. "If vision be abolished it is called *cæcitas* or blindness, if depraved and receives its objects erroneously, *hallucination*" (Sir J. Brown, "Vulg. Errors," 1646). The use of "delusion" to designate erroneous ideas, and not merely erroneous sensory perceptions, also agrees with the common use of the word, but it is a term of more general application than the others.

Delirium may be "quiet" or "active." In quiet delirium there are delusions and hallucinations (especially of sight) which dominate the patient's ideas. He does not recognize his friends, mistakes inanimate objects for persons, animals, etc., and often talks almost continuously, but usually in a low, monotonous voice, with words imperfectly finished, so that considerable attention may be needed to ascertain what is said—a condition which is amptly termed "low, muttering delirium." On the other hand, in "active delirium" the patient tries to act according to his erroneous ideas; he may insist on getting out of bed, going down stairs or out of doors, and may even, under the guidance of some delusion, jump out of a window, or commit suicide in some other way.

Although the general elements of delirium are identical with those of the mental derangement termed insanity, yet certain common features of insanity are rarely seen in delirium. Such are the extreme and persistent emotional depression of melancholia, the exaggeration of idea which characterizes some cases of general paralysis of the insane, the outrageous delusions of personal identity met with in the latter and in some cases of chronic insanity; and, lastly, the rhetorical loquacity of acute mania, is, to say the least, extremely rare in symptomatic delirium.

Among organic brain diseases that cause this symptom, the most frequent are meningitis, cerebritis, tumors, multiple or extensive softening, and multiple degenerations (*e. g.*, scleroses).

There is little in the mere character of the delirium to indicate which, of its many causes, is at work. In toxic delirium, especially that due to acute alcoholism, there is often conspicuous tremor of the hands and face, and there are usually the visual hallucinations that have furnished a familiar metaphor by which it is popularly known. Chronic alcoholic delirium may present none of these characteristics. The chief difficulty is the distinction between the delirium due to pyrexia, and that symptomatic of organic brain disease. If delirium is the sole nervous symptom, it can only be accepted as evidence of brain disease when the temperature is normal, or, at least, is not raised more than one or two degrees. Those who have indulged excessively in alcohol may indeed be rendered delirious by a slight degree of pyrexia, and the delirium is then of complex origin; but in the sober, pyrexial delirium scarcely ever occurs unless the temperature exceeds 101°. In the presence, therefore, of sufficient fever to account for delirium we are not justified in assigning it to a primary disease of the brain, unless other symptoms of such disease are present. These may be extremely varied in character, but one of the most important is headache. As Sir William Jenner insisted, forty years ago, the headache of fever ceases, as a rule, when the delirium comes on, and if the two coexist there is probably encephalic disease. Other significant symptoms are persistent vomiting, spasm or rigidity in limbs or neck, inequality of pupil, strabismus or diplopia, and optic neuritis. Optic neuritis now and then succeeds an acute specific fever (especially typhoid and scarlet fevers), but it does not come on during the height of the disease. In other cases, the early symptoms of the disease afford important help in estimating the significance of delirium. If delirium distinctly precedes fever, it has evidently the same significance as if it exists alone. If it succeeds a period of initial unconsciousness, or a convulsion, we are justified in assigning to it a primary intracranial cause. The only exception is in the case of acute specific diseases in children, in which a convulsion may occur at the onset; but the nature of such cases is usually clear.

Obvious as these rules may seem, it is difficult to exaggerate their practical importance. The neglect of them has been, and still is, a frequent source of error. In many instances, typhoid fever, or catarrhal febricula in children, has been thought to be tubercular meningitis, because undue weight had been assigned to the delirium. Grosser errors, indeed, are not unknown. On the other hand, care should be taken to avoid the opposite mistake. The combination of delirium and pyrexia must not be accepted as evidence of the general nature of the malady, until the observer is satisfied that there are no symptoms of nerve disturbance for which a blood state will not account. The records of every fever hospital will show how often acute brain disease has been mistaken for a specific fever, because the pyrexia has been allowed to obscure the significance of other indications of nerve disturbance.

SIMPLE MENTAL FAILURE is indicated first and chiefly by *defect of memory*, "amnesia," in the widest sense of the word. There is a physical side to memory as to other mental processes. All functional action of nerve elements is attended by molecular changes in them, and never leaves their nutrition in exactly the same condition as before. A state is left, for a time, in which the same functional action occurs more readily; there is a diminution of resistance in the combination of nerve elements concerned, and this residual disposition is increased by repetition. This constitutes the basis of motor training, in which facility in performing complex actions is acquired by practice. Such actions are learned by a sort of motor memory, which enters but little into the region of consciousness. A similar condition appears to constitute the physical basis of memory, properly so called. During various mental processes, combinations of nerve cells are in action, which differ according to the diversity of these processes, and the residual disposition to act in the same way, in the same combination, renders possible a revival of the original activity, and on the side of mind a memory of a past image.

Thus there is probably no special faculty of memory, physical or psychical, apart from the general cerebral and intellectual processes. There is, however, or seems to us to be, a peculiar power of voluntary revival of these processes, which is popularly termed "recollection."

The diseases of the brain that affect memory are extremely numerous, and comprehend general impairment of cerebral nutrition, as by an acute fever, severe mental shock, and especially peculiar functional conditions, such as the trance state of hysteria.* It results also from organic diseases that damage the nutrition of the cortex, as meningitis, extensive local lesions, hemorrhage, softening, tumor; these often indirectly impair the function of a more extensive area than is structurally damaged. Loss of memory results also from various degenerative processes, which are, for the most part, classed as forms of insanity, *e. g.*, senile dementia and general paralysis of the insane.

The defect may be seen only in the inability to retain new impressions, although those acquired up to the time of the commencement of the symptoms may be preserved. More frequently the loss is retrospective as well as present, and it is usually the later processes that cannot be revived; the recent not the remote past that is forgotten. The loss may extend back for a few days or for months or years. This is often seen in degenerative diseases of the brain; in senile amnesia, for instance, the events of early life may be vividly remembered, and those of later years be lost, and thus we have the anomaly that "the new perishes, the old endures" (Ribot). A clergyman once came under my notice who had lost all memory of the last twenty years of his life, and maintained, with consistent perversity, that his age, length of parish work, etc., were just twenty years less than they really were. In rare cases, in which lost memory has been regained, recovery has occurred in the reverse order, from the past toward the present. Thus, a man, after an accident, had forgotten entirely the events of the three preceding days, and each day was successfully remembered until, at last, the accident was recalled.† It is said that in the loss of memory the more special acquisitions are first lost, the most general are preserved longest. This is best seen in the case of words, among which proper names are first lost, and then nouns, and then names of qualities. In more general loss of memory the order is difficult to trace. Loss of memory is sometimes partial, *i. e.*, there is considerable or complete loss of some class of ideas, as, for instance, of a foreign language. These cases, although curious, are of little interest from a medical point of view.

Another symptom of mental failure, with which, indeed, the loss of memory is closely connected, is deficient power of attention—that is, the deficient power of excluding from the domain of consciousness all but one subject. Closely connected with this, also, is incoherence of idea. Instead of the definite sequence of mental processes that we recognize as normal, one mental image excites another by some accidental association (as word resemblance), which would be, in health, subordinated to logical sequence. Often the connections that determine the morbid sequence are so subtle as to evade detection. This incoherence of idea is portrayed in language. It is conspicuous in delirium, and is also frequent in simple mental failure.

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* Several remarkable cases of complete loss of memory after such attacks have been recorded (see Carpenter, *Brain*, vol. i).

† Kömpfen, quoted by Ribot, "Diseases of Memory," p. 123.

Defects in the moral sense are also common symptoms of mental failure, although not always present. The defect shows itself in a want of sense of propriety. The slighter defects of this character are relative, rather than absolute, and must be measured by their deviation from the normal conduct of the individual. Many actions, such, for instance, as spitting about a room, would be more distinctly pathological in a man of refinement than in an ill-mannered man of the lower classes. Some actions are unequivocal in their significance, as when a patient deliberately micturates on the floor, or exposes himself indecently without perceiving the impropriety of the action. Urine and stools are often passed into the bed, in cases of brain disease, in consequence of mental failure, when there is no incompetence of the sphincters. This always indicates a considerable depth of mental change, often much greater than appears on the surface. Hence the symptom is often of grave significance, and always of great practical importance.

Another class of actions which result from mental defect are offences against morality and propriety, and these often seem to depend less on want of knowledge of the nature of the action, than on the absence of motive to avoid it.

It is important to note that in children with slight mental defect the absence of natural shyness, and the difficulty of producing any deterrent sense of wrong or shame, often afford more conspicuous indications of the mental deficiency than actual lack of intellectual processes. The children are unabashed by the presence of strangers, and are disobedient, mischievous, meddlesome, wantonly injuring their playthings, and even their playmates. The absence of shyness often leads to a pseudo-precocity which fond parents take to be the dawn of genius.

AFFECTIONS OF SPEECH.*

Speech is deranged in various ways by disease of the brain. The process of articulation is immediately effected by a mechanism of nerve nuclei situated in the pons and medulla, but these are excited to action by centres in the cerebral cortex. Thus there are higher and lower mechanisms; the former is cerebral, the latter bulbar. When there is disease of the lower mechanism, the elements of speech are correct in number and order, but their form is imperfect; there is defective articulation. In disease of the higher mechanism the form of the elements of speech may be correct, but they are defective in number and arrangement. There is, however, one condition in which disease of the cerebrum interferes with articulation—

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* The most important writings on the subject of defects of speech are those of Trousseau in his "Clinique Medicale" (Lect. viii, p. 218, of Bazire's translation); Bastian, *Med.-Chir. Rev.*, January and April, 1869; Broadbent, *Med.-Chir. Trans.*, *1872*, vol. lv; Bateman, "On Aphasia," London, 1870; Hughlings Jackson, *Brain*, vols. i and ii; Wernicke, "Der Aphasische Symptomen-Complex," Breslau, 1874, and Kussmaul's article in Ziemssen's *Cyclopædia* (vol. xiv of the American translation).

when the disease involves the structures concerned in both hemispheres. One-sided disease, either of the cortex of the brain or the path from it, may interfere with articulation for a time, but the loss soon passes away, because it is soon compensated by the other hemisphere. The most important movements are bilateral, and, like other bilateral movements, can be excited from either hemisphere. But symmetrical disease of both hemispheres, if it involves the centres or paths of the muscles concerned in speech, may permanently derange or abolish articulation. The condition is termed "pseudo-bulbar paralysis," from the close resemblance to the effect of disease of the medulla.

DEFECTS OF ARTICULATION.—The changes in articulation that result from disease of the medulla and lower part of the pons, vary from a slight imperfection to a loss so complete as to leave only laryngeal tones to represent articulate speech. This defect is termed *anarthria* or *dysarthria*. Considerable loss is usually associated with obvious palsy of the parts concerned in articulation. The most important effects of such definite paralysis are these: in paralysis of the lips the labial consonants, which depend on precise closure of the lips, are defective; *p*, *b*, and *m* are transformed into *f* and *v*. In palsy of the tongue the linguals *l* and *t* are imperfectly pronounced, and tend to become *y* and *th*. Palsy of the palate not only gives an undue nasal resonance to the voice, but impairs the pronunciation of the explosive labials *p* and *b*, because the patency of the nares prevents the needful compression of the air in the mouth; and these explosives are therefore transformed into the continuous labial *m*.

In slighter and less defined disease of the bulbar mechanism, terminal or subordinate consonants and syllables are imperfectly pronounced, irrespective of their character, and are often run together or elided in what is termed "confluent" or "elisive" articulation. Sometimes consonants that require delicately adjusted muscular action are omitted, or easier sounds are substituted, and occasionally consonants are unintentionally repeated. The *r* is especially a source of stumbling to these patients. In other cases, again, syllables are unduly separated, in what has been termed "staccato," or "scanning utterance."

CEREBRAL DEFECTS.—The defects of speech that result from disease of the cerebral hemisphere—defects not of articulation, but of the elements of speech—are now known by the name *aphasia*. The word has become current as a general designation for various forms of defect, manifested in the various modes of expression, just as the analogous word "anæmia" is applied to various forms of deficiency of blood.* The word owes its currency to Trousseau, who summoned it from a slumber of two thousand years,* as a substitute for the term *aphemia*, with which Broca had in turn displaced the *alalia* of Lordat. "Aphemia" is sometimes still employed in a restricted application to articulate speech.†

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* The inaccuracy of applying an absolute negation to a partial defect has led to the suggestion of "dysphasia" as a frequent substitute. The term does not, however, seem likely to come into use, a matter for little regret, since the word has not the merit of unimpeachable exactness, and it has an unfortunate resemblance in sound to "dysphagia."

The subject abounds in difficulty, arising partly from its complexity and partly from the uncertainty due to a deficiency of facts and the obscurity produced by a redundance of theory. Precise knowledge of the effects of local disease on the various relations of language is scanty, and the possible interpretations of the conditions observed during life are numerous. The clinical facts may be and have been variously explained; and it is almost startling to find, when we come to weigh the evidence, how small is the ground on which some theories are based, and how much deficiency of fact a clear diagram may hide. Hence an attempt to state the definite knowledge we possess may seem to be at once meagre in scope and hesitating in assertion. But if the certain knowledge is insufficient to enable us to explain all the complex phenomena, it furnishes a definite starting-place from which to investigate them, and if the obscurity is recognized we may be saved at least from misplaced confidence. Hypotheses unfortunately cannot be altogether avoided, but it is desirable that they should be simple in nature and restricted in use until more facts have been ascertained.

Speech processes in the cortex of the brain are sensory and motor. By the former language is received, and by the latter it is uttered. These processes go on chiefly in the left hemisphere in right-handed persons, but in those who are left-handed the processes go on in the right hemisphere, and this even when the patient uses for writing not the left hand but the right. There are, however, similar structures in each hemisphere, and if those on the left side are destroyed, the corresponding parts of the right hemisphere may take on the lost function, and the symptoms of the loss may slowly pass away. The proof of this is that, in several cases of this character, a fresh lesion in the right hemisphere has destroyed the reacquired power, and there has then been no recovery. It is probable, moreover, as we shall presently see, that the right hemisphere normally takes some part in speech processes, and that the part it takes varies in different persons and at different periods of life. Loss of speech from disease of the left hemisphere is, as a rule, quickly recovered from in children, by the compensatory use of the right hemisphere; a similar recovery takes place in some adults more readily than in others, and in some it does not take place at all. If such compensation occurs—and the occurrence is unquestionable—it is evident that, very soon after the left hemisphere has lost some of its function, the right hemisphere must begin to coöperate with it in effecting the speech that quickly returns. Such coöperation, admitted when the damage is great, probably occurs also when the damage is partial. It is evident that we have here an element of great uncertainty. If the actual lesion is not known, we cannot be sure to what extent the speech processes that return, and therefore the disorder they present, are to be ascribed to the left hemisphere or to both hemispheres; and we must incur some error, and it may be great error, if we fix our attention exclusively on the left hemisphere. Examples of this will be manifest as we proceed.

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* Trousseau seems to have been unaware of the antiquity of the word. It was apparently "invented" for him, in 1861, by a Greek, M. Chrysaphis (see Trousseau's "Clinique Med.," lect. lxi, and the footnote at p. 218 of Bazire's translation).

† "Aphemia" was rejected by Trousseau, and by Broca himself, at the instance of Littré, because its Greek significance is not "speechlessness" but "infamy."

The sensory relations of speech are with hearing and sight, and also, in blind persons, with touch. The relation to the sense of hearing is the most important; it is the first speech relation to be developed in the child, who can understand many words before he can utter one. The part of the brain concerned in the auditory perception of words is the posterior half of the first temporal convolution on the left side. It is apparently the auditory centre itself, at least structures at present indistinguishable from this centre. If this region is diseased there is often transient deafness in the opposite ear (see p. 464), and there is persistent inability to understand the meaning of words, although they are still heard as sounds. This condition causes considerable derangement of speech. It was first described by Dr. Bastian, and accurately referred by him to destruction of the "auditory perceptive centre." Wernicke, to whom belongs the credit of discovering the precise seat of the disease that causes the symptom,* proposed for it the term *sensory aphasia*, and Kussmaul afterward called the loss *word deafness*. The sensory condition is now generally known by the latter term, and the former is applied to the general disorder of speech that results. The dependence of this system on disease of the first temporal convolution is well established. This part was diseased in every one of seventeen cases collected by Sepelli.†

The part of the brain concerned in the perception of visual word symbols seems to be in the lower and hinder part of the parietal lobe, also on the left side of the brain. Disease of this part has caused inability to read even the simplest word, a condition that has been termed "word blindness." Of the cases recorded by Sepelli, in each one of which word blindness coexisted with word deafness, the disease extended into this part of the parietal lobe. This region, as we have seen (pp. 462, 463), is probably the seat of the "higher visual centre," and transient "mind blindness" has been caused by its disease, *i. e.*, an inability to recognize not only words, but objects also. Such mind blindness has also occurred in a few cases as a persistent symptom, but we do not know whether it then depends on a unilateral or bilateral lesion (see Affections of Vision). Moreover, the crossed amblyopia, which results from extensive disease in this part, has not often been recognized in cases of impairment of visual word processes. The speech defect interferes with the detection of the visual loss, and it may be that word processes are related to only a part of the region that has to do with vision. It must, however, be noted—and the fact very much increases the difficulty of the subject—that inability to read does not prove disease of this part in the same way as inability to understand heard words proves disease of the first temporal. The mechanism of reading is complex, and, as we shall see, it may be deranged indirectly.

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* "Die Aphasische Symptomen-Complex," 1874, a work of remarkable ability.

† Sepelli, *Revista Sper. de Fren.*, *1884*, p. 94 (see also Amidon, *New York Medical Journal*, January, 1885).

Motor word processes take place in the posterior part of the third frontal convolution, and perhaps in the adjacent part of the ascending frontal. The latter contains the cortical centre for the movement of the tongue and lips, to which the motor speech centre is thus adjacent, and perhaps in part corresponds. The path from it passes through the white substance of the hemisphere to the internal capsule at the junction of its two parts, in front of the fibres of the pyramidal tract, and it lies to the inner side of these fibres in the crus cerebri. It is commonly thought that the path from the speech centre is distinct from that for the tongue and lips, but the evidence of this is insufficient. It is more probable that the speech path is that for the simple movements of the parts concerned, although the cortical centre is more extensive. Whether the island of Reil forms part of the speech centre is still uncertain; speech defect, from disease limited to it, has not yet been observed, but its position between the motor and auditory centres, and the way in which fibres between the two pass by it, have led Wernicke and others to regard it as part of the speech region. All the cases adduced as proof of the dependence of aphasia on disease of the island of Reil are inconclusive, either because the lesion was not limited to it, or because the duration of the case was too short to permit indirect symptoms to pass away.*

Destruction of the motor speech region on the left side causes total, or almost total, loss of voluntary speech. Such loss has resulted from a lesion in the third frontal no larger than a hazel nut.† If the loss is old or partial, words are often wrongly formed. Hence the loss has been termed *ataxic aphasia*,‡ but this, as a distinctive term, is inexact, since a very similar disorder of words may arise from pure word deafness. Hence it is far better, with Wernicke, to term the effect of disease of the motor speech region "*motor aphasia.*" Its precise characters will be presently described.

Motor aphasia may be produced by disease of the conducting path from the cortex, as well as by the disease of the cortex itself.§ The loss from disease of the path is only permanent when the lesion is just beneath the cortex. If the lesion is near or in the internal capsule, the loss passes away in the course of a few weeks. The explanation of the difference is probably this: if the fibres from the left speech centre to the corpus callosum are intact, the speech processes, arranged on the left side, can find expression through the corresponding centre on the right side; if the lesion is just beneath the cortex, these callosal fibres are interrupted, as well as those to the internal capsule, and the loss is as permanent as if the centre were destroyed. In the former case, a second lesion in the motor path of the right hemisphere, even in the internal capsule, may cause permanent aphasia.*

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* Thus, in a case published by Déjerine, as evidence that motor aphasia may be due to an insular lesion (*Revue de Méd.*, *1885*, p. 174), the patient lived for a few days only, and the lower part of the central convolutions was likewise destroyed. (Compare also the case mentioned in the note on p. 546.)

† Rosenstein, *Berlin klin. Wochensch.*, *1868*, p. 182.

‡ First by Sanders, *Edin. Med. Journ.*, March, 1866; soon afterward by Ogle.

§ Raymond and Artaud have collected twenty-three cases of aphasia from disease of the white substance, *Gaz. méd. de Paris*, *1883*, No. 47.

FIG. 238.

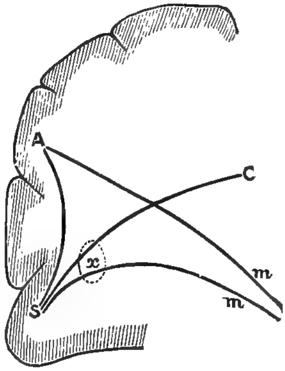

Diagram of probable course of fibres from motor speech centre. A, hand centre in the middle of the ascending frontal convolution; A *m*, fibres from this to internal capsule; S, motor speech centre; S C, fibres from this to the corpus callosum; and S *m* to the internal capsule. S A, fibres from speech centre to hand centre. A lesion at *m m* will cause only transient aphasia, the speech process being able to pass by S C to the corpus callosum and opposite speech region; a small lesion at *x* would cause permanent aphasia, since it involves both the fibres to the corpus callosum and internal capsule, but would not abolish expression by writing, the fibres S A (connecting the speech and hand centres) escaping.

In the act of writing, the speech processes, arranged in the motor centre, pass to the centre for the hand in the middle of the ascending convolutions, and thence the nerve impulses pass by the pyramidal tracts to the cord. The evidence of this is that disease of the motor speech centre abolishes the power of writing (termed *agraphia*), even when the hand centre is intact, and the movements of the arm are unimpaired.† The same result follows an isolating lesion just beneath the cortical speech centre, and hence the path to the arm centre must be by the "associating fibres" of the subjacent white substance, and not through the gray matter of the cortex. But it is conceivable that a sub-cortical lesion may be so placed as to interrupt the paths to the internal capsule and to the opposite hemisphere, and not that to the arm centre (see Fig. 238). In such a case there would be permanent loss of uttered speech without loss of the power of writing. Such a condition has actually been observed.‡

Disease limited to the motor speech region sometimes abolishes the power of reading (*alexia*); § sometimes it does not. The difference depends in part on the degree to which motor speech processes are concerned in the comprehension of visual speech symbols. In persons accustomed to reading, the intellectual images may perhaps be aroused directly by the visual processes, without the intervention of the motor processes. In those who are imperfectly educated, the motor processes are essential; the lips may even be seen to move during the process of reading. It must be remembered that children learn to read aloud words they do not understand: indeed, the early effort of a child is merely to pronounce the seen words.*

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* This appears to have been the case in an instance recorded by Luys, *L'Encephale, 1885*, No. 3.

† In Rosenstein's case, mentioned on p. 544, in which aphasia resulted from a hemorrhage the size of a hazel nut in the third frontal, the power of writing was lost as well as speech.

‡ Wernicke, *Neurol. Cent., 1886*, p. 124.

§ Alexia, be it remembered, is the loss of the power of understanding written or printed words, not merely a loss of the power of reading aloud.

In addition to these two leading varieties of aphasia, the motor and sensory, there is another in which the patient, without loss of the motor processes, and without word deafness, has a difficulty in recalling words, and uses wrong words. This has been termed "amnesic aphasia," or "loss of memory for words." It has been referred by Wernicke to an interruption of the path between the motor and sensory centres, by disease of the island of Reil, and he has termed it "conduction aphasia" ("Leitungsaphasia").† He assumes that the interruption prevents the direct action of the sensory on the motor centre which is necessary for its guidance.‡ As a fourth variety, "total aphasia" has been described: the form in which both auditory and motor centres are destroyed, and all use of words is lost.

Motor Aphasia.—In a case of motor aphasia from destruction of the motor speech region, the patient is at first speechless, and even expressions by signs may be impaired. He may shake his head when he means "yes." Before long, expression by gestures becomes accurate, and the patient regains the power of uttering a few simple words, as "yes" or "no," § and often other words are at times uttered under the influence of some emotion, or some word may be repeated on every attempt to speak. The patient is able to understand whatever is said to him, but cannot repeat it. He cannot, of course, read aloud, and he is generally unable to understand seen words, such, for instance, as a written or printed direction to perform some simple action. He cannot write, even if his hand is unaffected. He may be able to copy, and even to translate printed letters into writing characters. If the right hand is paralyzed, the patient may try to write with the left hand, and may try to form words with separate letters. The latter is a rather severe test, because it is an unfamiliar process. Processes of thought are little interfered with, and the patient is perfectly aware of his errors in utterance. The use of figures is usually interfered with, as well as the use of words, but to this rule curious exceptions are met with, the significance of which we are not yet able to understand. Thus, in one case of considerable motor aphasia recorded by Volland, a high degree of arithmetical ability was preserved.*

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* At the same time it is doubtful whether the observed differences in the influence of motor aphasia on the power of reading can be altogether explained by the degree of education and of practice in reading. We need more facts regarding the pathological lesions in these cases.

† That defects in speech may be due to interruption of the "set of efferent fibres emerging from the auditory perceptive centre having to do with the incitation to the muscular acts of speech" was suggested by Dr. Bastian in 1869 (*loc. cit.*). Apart from the question of accuracy, the term "conduction aphasia" does not very aptly express the assumption that conduction is arrested.

‡ There is at present no actual proof of the truth of this assumption. Lichtheim has published a case (*Deut. Arch. f. klin. Med.*, Bd. 36, Case 1) which he describes as an "undoubted example of this form of aphasia," but the lesion involved, besides the island of Reil, 1½ cm. of the third frontal and part of the middle of the first temporal. (See also note on p. 544.)

§ I have only seen one case in which a patient, nine months after the onset, was absolutely wordless. There was considerable word deafness as well as absolute motor aphasia. Expression by signs was extensive.

In some cases this condition persists, unchanged, for years. More often there is, after some months, a slow recovery; first one word and then another is uttered; and considerable power of speech may be ultimately regained by the "reëducation" of the right hemisphere.

In other cases, however, in which the disease is partial, the loss of speech is found to be incomplete when the immediate effect of the lesion has passed away, but many mistakes are made in the forms of words and in their use.

In the conditions thus sketched, there are two elements: (1) loss of speech, (2) retention of speech, or at least of utterance of words, with more or less error. The loss is due to the lesion; the retention and error are due to the imperfect action of the remaining motor speech centres, probably on both sides of the brain, if the disease is partial, or on the right only if it is complete. It is obvious that, when there is complete destruction of the left motor speech centre, the utterance that remains must be by the right hemisphere. This remaining power of expression may be of several kinds. (1) Expression by gesture, which may be at first impaired, but is soon regained. (2) Some general propositional utterance; as "yes" and "no," which may be at first used wrongly. (3) Some special utterance, which recurs on all attempts to speak, and may be a word or words, or an unmeaning combination of syllables. (4) Combinations of words which express some strong emotion, and not the proposition to which their form corresponds, such as an oath—"dead propositions" Hughlings Jackson has aptly called them. (5) Words as the vehicles for tone in singing. A patient who can only say "yes" and "no" may sing a song, and in doing so may utter every word of the song, although he is quite unable to speak it.† Often words can be uttered in a half-automatic manner, *e. g.*, in combination with other words, or under emotion, and not by a deliberate voluntary effort. Thus, a patient with slight aphasia was told to say "no," and after one or two ineffectual attempts said, "I can't say no, sir." Thus, likewise, I have known a patient to be unable to put out his tongue when told, although he automatically put it out and licked his lips to facilitate the process, and afterward was still unable to protrude it by a deliberate effort. An oath uttered in anger cannot be repeated without the emotion.

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* *Munch. Med. Wochensch.*, *1886*, and *Neurol. Centralbl.*, p. 274. The patient was a boy, aged fifteen. After a fall on the head, there was complete motor aphasia, only the recurring utterance "Anna" persisting. Recovery was very slow, and the defect had not entirely disappeared six years later. From the first the boy could add, subtract, and multiply, and at school he progressed steadily in arithmetic.

† For an instance, see *Lancet*, *1875*, vol. ii, p. 794.

The "recurring utterances" are very curious. They seem to be either the words the patient was about to utter when taken ill (Hughlings Jackson), or, more probably, the last words actually uttered. A woman who told a cabman to drive her to "Mrs. Waters," and became speechless a few minutes afterward, had the recurring utterance, "Missis." The recurring utterance of a girl, seized when riding on a donkey, was "gee, gee" (Jackson); that of a signalman, taken ill at his post, was "Come on to me;" that of a librarian was, "List complete" (Russell). The phenomena of recurring utterances, which must be effected by the right hemisphere, seem to show that this hemisphere takes some part in normal speech. Fresh processes for words cannot be energized through the will, but the residual disposition of those last energized voluntarily, leads to their excitation by an attempt to speak. As Hughlings Jackson has insisted, the loss of speech from disease of the left motor region is essentially a loss of *voluntary* speech. When speech is slowly regained by the right hemisphere, many of the errors in utterance may be traced to the defective voluntary influence, and to a tendency to the reënergizing of nerve processes recently in activity. Consonants are repeated, instead of the proper consonant being uttered, and those of a recurring utterance may crop up in wrong places. When the patient whose recurring utterance was "Missis" acquired more speech, the "s" was constantly cropping up in other words. Ultimately, almost perfect recovery may occur, and there may remain only slight and occasional errors in the form of words, with a difficulty in finding the word desired, and a tendency to use wrong words.

In motor aphasia the inability to write is usually even greater than the inability to speak. In writing, speech symbols are again symbolized, and in the double symbolism there is more room for error. The patient may be unable to form a single letter, or letters may be formed, but combined wrongly, or there may be merely mistakes of letters in words that are otherwise correct. The patient can often sign his own name—which is an almost automatic performance—when he cannot express a proposition in writing. As an instance of this, and also of the fact that errors in writing are greater than in speech, may be given the case of a man who, after signing his name, *James Slim*, correctly, was told to write down "how he came to the hospital," and wrote *cgng kgig kiyan*, but in answer to the same question said, "Rail, clab," and when asked "How else?" replied, "No way." The tendency to *repetition*, to the rëenergizing of processes recently in activity, causes many of the errors in writing as well as in speech, and is seen in the above sentence. *Glag* for "glad" is another instance. Other mistakes in written letters are due to a similarity of the muscular (and therefore nervous) action in pronouncing them, as the substitution of *l* for *p* and *f* for *v* (observed errors), a proof, if proof were needed, that the written words are first energized in the speech centre.

Loss of the power of understanding seen words, that is, loss of the power of reading—"alexia"—often, but not always, accompanies motor aphasia. This has been already mentioned, and it has been pointed out that the difference probably depends on the extent to which the patient is dependent on the motor processes for the comprehension of the visual symbols. In some persons the latter arouse the nerve processes for "ideas" directly; in others only by the energizing of the motor structures. In the former case, even disease of both temporal and frontal speech centres may not destroy the power of reading, although, of course, the patient cannot read aloud.*

Sensory Aphasia.—In word deafness, due always to disease of the first temporal convolution, heard words are not understood. This is best tested by letting the patient perform some simple action which is not anticipated. Word deafness is seldom absolute, and an expected sentence, such as "Put your tongue out," from a doctor, is understood when an unexpected sentence is not. Expectation involves lowering of resistance in a certain nerve combination, and then perhaps the right hemisphere may suffice. Word deafness always causes considerable derangement of speech. This depends on the fact that the subjective revival of word processes takes place chiefly in the auditory centre. All the residual dispositions—motor, auditory, visual—constitute physical memories for word processes, but as auditory word processes are the first to be developed in the child, so they continue the most important through life. If they are lost, the patient is thrown on his motor memory, and this often misleads.† Hence there is often error in the words used and in their form. But it is especially the voluntary revival of words that is lost, and words may still be revived automatically, just as it is voluntary utterance that is lost in motor aphasia, and words may still be uttered automatically, as in singing a song. Hence these patients have often an extensive use of words, especially general words, which they utter promptly, and in an automatic manner, but they have difficulty in the deliberate revival of special terms. "If I have to say a thing I must say it at once, or I cannot say it at all," one patient observed. It is probable that the automatic revival of words in disease of the left hemisphere is subserved by the centres on the right side. It is certain that slow recovery of the power of comprehension of spoken words is by the "reëducation" of the

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*As in a case reported by Kahler, *Cent. f. Nerv.*, *1885*, p. 393. The statement in the text is only a probable explanation. We need more facts as to the influence of definite lesions on reading.

†In educated persons the visual memory may aid in the revival, and probably develops with the demand upon it. An accomplished man recovered slowly from complete word deafness (with persistent hemiplegia). He said that if he had a difficulty in realizing the meaning of a word, he would repeat it over and over, and at last seemed to see the letters of the word, and its meaning flashed upon him. This is an interesting instance of the way in which one centre may reinforce another. A different example of similar help was presented by a patient who could write, but could only understand a word seen if he traced the letters with his fingers in the air (Charcot, *Leçons*, T. iii. p. 161, 1887). The same case has been recorded by Herczel. So, too, a patient of Wernicke's was able to read writing only by tracing over the letters ("Gehirnkr.," i, p. 338).

right hemisphere, since such regained power has been again lost in right-sided disease. The readiness with which such recovery takes place seems to vary much in different persons, as is the case also in motor aphasia. In some there is no recovery.* As a rule, however, recovery occurs more readily than in motor loss.

The disorder of speech in word deafness varies much in different cases. As a rule, considerable use of words remains, but chiefly in half-automatic spontaneous speech. Parts of speech in more general use, prepositions, adjectives, verbs, etc., are readily uttered, but there is great difficulty in more special words, such as nouns. The patient often employs a circumlocution of more general words instead of the special word that he cannot revive. Instead of the word "knife" he may say, "something to cut with." In extreme cases, there may be much error in the forms of words, and these errors are often distinctly due to the inadequacy of the motor memory on which the patient has to rely. Sometimes, indeed, there is as much formal disorder and "ataxy" of speech as in motor aphasia—one of the considerations that militate against the use of this term as a designation for the motor variety. In very rare cases, extensive disease of the sensory region has caused an almost absolute loss of expression by words, greater, indeed, than can be altogether explained, and such as to resemble the loss in disease of the motor region.† The effect of disease of both temporal lobes is, of course, to preclude recovery, and great derangement of speech results. In one case of complete deafness from cerebral disease, in which such a double lesion probably existed, speech was almost unintelligible.‡

Word deafness renders the patient unaware of his errors; a striking difference from the motor form, in which the patient at once recognizes his mistakes, and is annoyed at them. In absolute word deafness the patient is unable to repeat words uttered to him, but in partial loss he is able to repeat, often with some formal errors.

The patient is also unable to read in many cases of word deafness, but it is probable that this is the result of the auditory loss, or is due to damage to the visual centre, which is supplied by the same artery as the auditory centre, and often suffers in the same lesion. Inability to read, as an isolated symptom, is probably always due to a lesion of the supra-marginal and angular convolutions.* It is not often that there is actual mind blindness, an inability to recognize not only words, but any object, until some other sense than that of sight is employed (see Affections of Vision). In some cases the patient has been able to read aloud correctly, but did not understand what he read. The effect on writing is similar to the effect on speech, but greater, owing to the double symbolism and the extension of error thus produced.

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* As in a case recorded by Rosenthal, in which word deafness remained absolute until the death of the patient, two and a half years after the onset (*Gazeta lekarska, 1883*, and Virchow's *Jahresb., 1883*, ii, p. 85).

† Thus, in a case recorded by D'Heilly (*Prog. Méd. 1883*, No. 2), arterial thrombosis in the course of phthisis caused softening of the left first and second temporal convolutions, and of part of the angular gyrus, without implicating the motor speech region; the patient, during the nine weeks she lived, only uttered the words "parceque," "le plan," and "oui, monsieur." She understood no words, but comprehended signs readily. She had lost the power of reading and writing. In a case of Charcot's, described by Trousseau, in which the lesion involved only the first and second temporal convolutions and the posterior half of the island of Reil, the patient uttered only "ta-ta-ta" during eight months.

‡ Wernicke and Friedlander, *Fortschritte d. Med., 1883*.

We have seen that in partial word deafness, partial from the beginning or partial by recovery, a condition exists in which there is great difficulty in the voluntary revival of words, especially of the more special words, such as nouns. This is the "verbal amnesia," or "amnesic aphasia."† As already stated, it sometimes constitutes the sole defect from the beginning, and is then commonly regarded as a distinct variety of aphasia, and has been ascribed to interruption of the conducting path between the auditory and motor centres (see above, p. 546). Whether the condition is actually thus produced, and whether a simple interruption of this path can produce this effect, remains to be proved. The condition is often conjoined with slight word deafness, and then is probably due to a partial lesion of the auditory word centre. It may not only be a residual condition left by partial recovery from a considerable degree of word deafness, but, as we have seen (p. 548), it may be left by recovery from complete and pure motor aphasia. The fact that a typical "amnesic aphasia" may be merely a residual condition left by a pure motor aphasia is very important. It is proved by several recorded cases in which an autopsy was obtained.‡ Another important fact is that the loss follows a regular law from the general to the special. These facts suggest that the essential condition for the production of this defect is, that the speech processes go on in structures relatively incompetent. In some cases the structures concerned may be those of the right hemisphere, imperfectly reëducated. This form can only, therefore, be admitted as a pathological variety of aphasia with some reservation, until more evidence is obtained regarding the lesion that causes it as a primary and independent defect.

Inability to read, alexia, as we have seen, may have more than one mode of origin. It may be part of a pure motor aphasia in those in whom visual word processes can only arouse mental images through the agency of the motor speech centre. It seems strange that the motor process should directly arouse the nerve processes for the mental conception,§ but there is no evidence that the auditory processes intervene. On the contrary, the loss of these processes do not necessarily abolish the power of reading, as they certainly would do if the motor centre acted only through the auditory centre.* In other cases of motor aphasia, the power of reading is unimpaired, presumably because the visual processes arouse directly those for the ideas. Without motor aphasia, loss of the power of reading seems to be due only to disease of the posterior-inferior region of the parietal lobe, the region of the higher visual centre. We have seen that in all the fatal cases of word deafness in which the power of reading was lost, the disease was found to extend into this region. Hence we are justified in assuming that when alexia exists as an isolated condition, the disease is in this part. This isolated loss of the power of comprehending visual word symbols has been termed by Kussmaul "word blindness." It may or may not be accompanied by "mind blindness," which necessarily involves alexia. Word blindness is indeed partial mind blindness. In this condition there may be ability to speak and to write, but the patient writes, as one of Charcot's patients expressed it, "as if with the eyes closed," and this patient did actually write as well when his eyes were closed as when they were open.† Moreover, he could understand written words if his hand were made to trace the letters in passive movement. Such loss of visual word symbols has been met with in a few cases of hemianopia; it is not a necessary consequence of hemianopia, even when this is of cortical origin, and probably indicates that the lesion extends beyond the limits of the occipital lobe into the parietal cortex.‡ The lesion causing alexia is always on the left side of the brain in right-handed persons; on the right side in left-handed persons.§ The various character of the symptoms due to disease of this part of the parietal lobe may be due to the fact that its blood supply is often derived from two different arterial systems, and the area supplied by each seems to be subject to considerable variations. Hence partial disease is the rule, complete destruction the rare exception.

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* As in a case recorded by Henschen, *Neurol. Cent.*, *1886*, p. 464.

† The use of the word amnesia in this restricted sense is open to the objection pointed out by Hughlings Jackson and Kussmaul, that there is more than one memory for words (see p. 549).

‡ One is recorded by Battey Tuke and Fraser (*Journ. of Mental Science*, April, 1872); another by Sanders (*Edin. Med. Journ.*, *1866*, p. 811).

§ *i. e.*, it may seem strange that a centrifugal process should have an influence which seems to be centripetal; but a similar influence attends other motor processes, *e. g.*, our estimate of position by the movements of the ocular muscles (see diseases of the third nerves).

Inability to write, agraphia, as we have seen, results from destruction of the motor speech centre, and is then usually absolute, even when the right hand is not paralyzed. Spontaneous writing, and writing from dictation, are alike abolished, and hence it would seem that there is no direct path from the auditory word centre to the arm centre, but that the word processes must first be energized, as for articulate speech. But the patient is able to copy, usually only in the characters that are before him; sometimes, however, by a translation of printed into written characters. It is certain that, very often, the words copied are not understood. In word deafness, as we have just seen, the ability to write persists, but not that of writing from dictation. In word blindness, the patient can write, spontaneously or from dictation, but cannot copy. The difficulty in writing, as before stated, is often greater than in other forms of expression, but it is very seldom that it so far preponderates as to resemble isolated agraphia. The possibility of isolated agraphia has been doubted, and is, indeed, difficult to understand. But a curious case of partial agraphia has been recorded by Pitres.* In a syphilitic man, right hemiplegia without anæsthesia came on suddenly; it disappeared under treatment, leaving some rigidity, and there remained also right hemianopia, with inability to write. Speech was never affected. The patient could copy in fac simile, but could not write a single word, spontaneously or from dictation, with his right hand. Nevertheless, he could write easily with his left, and then copy what he had written with his right hand. He could read either printed or written words with perfect facility. This strange case seems to prove the possibility of an isolated partial agraphia. A lesion separating the left arm centre from the motor speech centre, leaving the callosal fibres intact, affords, perhaps, the best explanation of the symptoms, but this, or any explanation, must be purely speculative; and it may be added, the possibility, and even probability, of more than one lesion prevents any conclusion from the combination of symptoms.

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* In this connection it is instructive to note that even the motor processes for writing may assist in arousing the mental images, as in the case mentioned in the note on p. 549. Wernicke and Kussmaul assume that the auditory centre intervenes, and that the uneducated man, who can only read by the help of his motor centre, "has to read aloud to himself" in order to understand;—an assumption opposed to facts.

† Charcot, *Leçons*, Tome iii, p. 160.

‡ A lesion of the white substance involving the optic radiation may also involve the parietal cortex.

§ In the case recorded by Bernheim (see p. 588) of left hemianopia and mind blindness in a left-handed man, alexia was necessarily present.

Aphasia, motor or sensory, may be produced by any kind of lesion, but it is due to softening more frequently than to hemorrhage, on account of the greater frequency with which the former lesion involves the cortex.

The region of the cortex in which the speech centres are situated is supplied by the middle cerebral artery (see Fig. 217, p. 499), and obstruction of this is the most frequent cause of aphasia. Motor aphasia is produced by the softening due to obstruction of the first branch of the artery; sensory aphasia by that of the fourth branch. Since the second branch supplies the chief part of the central convolutions, hemiplegia is often associated with motor aphasia. Another occasional association is visual loss. The occurrence of hemianopia with word blindness has been already mentioned. Hemianopia is also sometimes associated with motor aphasia, but the affection of sight in such cases usually depends on a lesion near the internal capsule. Softening in this position, *e. g.*, near the thalamus, may extend outward to the first temporal convolution, or forward and outward, through the lenticular nucleus, to the island of Reil and third frontal convolution.

Loss of speech in children may be due to the same causes as are effective in adults.† Some of these are exceedingly rare in childhood, while other

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[*] *Revue de Méd.*, *1884*, p. 825.

[†] Ninety cases of aphasia in children have been collected by Steffen (*Jahrb. für Kinderheilk.*, Bd. xxiii, p. 127).

causes are more common in early than in later life. One of the latter is tubercular disease, which is a comparatively frequent cause of aphasia in children. Thus, speech is lost in some cases of tubercular meningitis, probably in consequence of irritative inhibition, which may also cause motor paralysis. The speech centre seems specially susceptible of inhibition. If recovery occurs from an organic lesion that has destroyed the motor speech centre, the power of speech seldom remains absent for more than a few weeks, compensation by the right hemisphere occurring with great readiness.

Aphasia may be due to functional disturbance of the brain as well as to organic disease. It may thus form part of various functional disorders, combined with other symptoms, and it may also occur alone. When right-sided convulsions occur from any cause, but especially from organic disease, there is often transient inability to speak, and this may continue for a quarter or half an hour, or even longer, after the fit. It is especially common when the convulsion begins in the face or tongue, and may occur when the spasm is very slight, and even when there is no convulsion, but merely a sensory discharge, tingling, etc. Aphasia is also met with as part of attacks of migraine, chiefly in those in which sensory disturbance occurs in the limbs of the right side. A patient who has such sensory disturbance, sometimes on one side, sometimes on the other, will have aphasia when the disturbance is on the right side, and not when it is on the left. As an isolated symptom, functional aphasia occurs chiefly in children. It has been excited by emotion, by some peripheral irritation in the intestinal tract or elsewhere, and in the course of acute specific diseases, especially of typhoid fever. Many examples of this have been recorded. Transient aphasia was due, in one case, to the irritation of a tapeworm.* In cases of peripheral injury it is uncertain whether the chief influence has been reflex or emotional. A child became speechless during the division of a tendon without anæsthesia, and continued speechless for eight days.† Aphasia coming on during the course of typhoid fever has certainly been sometimes due to organic disease, for it has been associated with characteristic symptoms, such as hemiplegia. But in other cases it has occurred alone, and after lasting for a few days, or for one or two weeks, it has passed away suddenly. Such cases suggest that the loss was due to an influence of the blood state on the brain. It has come on as early as the first week, and, in one recorded case, it continued until the seventh week. It occurs chiefly in severe cases, but does not seem to render the prognosis more grave. The aphasia has generally presented the characters of the motor variety; very rarely those of the sensory form.‡

Cases that can be regarded as congenital aphasia are extremely rare. In most instances there have been signs of a bilateral lesion of the brain, involving the motor centres for the tongue and lips, movement of which is usually distinctly defective. The cause is probably meningeal hemorrhage during birth.

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* J. A. y Tusset, *Estud. Clin.*, *1884*, and *Cent. f. Nervenh.*, *1884*, p. 491.

† Demme, *Wien Med. Blatt*, *1884*, No. 51.

‡ The subject of aphasia in typhoid fever has been discussed by Kuhn, *Deut. Arch. f. kl. Med.*, xxxiv, 56, and by Longuet, *L'Union Méd.*, *1884*, No. 60.

A very important question in connection with aphasia is its influence on testamentary capacity. It must depend, in any given case, on the ability to understand perfectly what is put before the testator, and to express at least assent and dissent with certainty. Word deafness is incompatible with will making, because it is impossible to know whether the testator really understands what is said to him. With perfect comprehension of spoken words, a valid will might be made if there was the power to express assent or dissent by signs, even if the testator was speechless.

The treatment of aphasia, so far as it is possible, is described in the chapter on softening of the brain. The history of the more important steps in the discovery of the cerebral relations of speech is briefly this. The faculty of language was first attributed to the frontal lobes by Bouillaud, in 1825, and to the left hemisphere, near the island of Reil, by Dax, in 1836. The title of Dax's paper is worth preserving. It was "Lesions of the Left Half of the Brain coinciding with Loss of Memory of the Signs of Thought." The function was further limited to the third frontal by Broca in 1861. The localization of the auditory centre in the first temporal is due to Meynert and Wernicke, and to the latter belongs also the credit of the localization of word deafness to this region of the left hemisphere.

GENERAL SYMPTOMS.

Besides the symptoms that depend on a derangement of the mental, motor, and sensory functions of the brain, cerebral disease may cause symptoms that are general, or consist in disturbance of other organs of the body. These may be conveniently considered together, although they bear different relations to the cerebral disease, and the relation of the same symptom may vary in different cases.

Temperature.—Elevation of temperature is frequent in cerebral disease, and is an instance of the difference of relation just mentioned. (1) It may be merely associated; the two symptoms, the cerebral disease and the pyrexia, being the result of a common cause. In ulcerative endocarditis we may have elevation of temperature from the alteration in the blood, etc., and cerebral symptoms in consequence of embolism in the arteries of the brain. In many cases we may be in doubt to what extent the pyrexia is due to the general or the cerebral disease, as, for instance, in general tuberculosis with a tubercular tumor of the brain or tubercular meningitis; and in pyæmia with secondary abscess of the brain or secondary meningitis. (2) The elevation of temperature may be due to the pathological process, which would have a similar effect if it were in any other situation. Meningitis is an example; the attendant pyrexia is the result of the inflammation. There are two important facts to be remembered regarding this consecutive elevation of temperature. First, it may be absent; the effect of the disease on the nervous system is to prevent the pyrexia which the morbid process would otherwise cause. Secondly, and connected with the last, is the fact that the pulse may not present the acceleration that usually accompanies pyrexia, and may even be abnormally infrequent. Hence the absence of pyrexia does not exclude intracranial inflammation, while elevation of temperature has additional significance if the pulse is not quickened. (3) The pyrexia may be the direct result of the cerebral lesion; that is, of the disturbed function of the nervous system, not of the pathological process by which that function is disturbed. This effect is produced especially by lesions of the pons and medulla; in these the temperature may rise to 104°, 106°, or 108°, and with the hyperpyrexia there is usually an abundant secretion of sweat. In some severe cases of hemorrhage into other parts of the brain there is also an initial and considerable rise of temperature, to 103° and 104° (Bourneville). Such cases are usually rapidly fatal. In some cases of meningitis the temperature becomes very high just before death, apparently from a similar mechanism. The pyrexia that accompanies the peculiar apoplectiform attacks in general paralysis of the insane is probably of this nature, since it is too brief, in proportion to its degree, to be ascribed to the pathological process causing the attack. Perhaps the brief fever that may attend the migrainous headaches of children is due to a similar mechanism. (4) The temperature may be raised as a secondary effect of the disturbance of cerebral function. When severe convulsions are frequently repeated, whatever be their cause, the temperature rises, apparently in consequence of the muscular exertion.

Depression of temperature is less common, and is met with chiefly at the onset of cerebral hemorrhage. It is then accompanied by other signs of severe shock. When there is a large hemorrhage, or when several hemorrhages occur in quick succession, this fall may progress until death occurs, and the rectal temperature may fall to 90°.

Vasomotor and trophic disturbance occurs most frequently in cases of hemiplegia, in the account of which it has been already described. It is also met with in meningitis and some other diseases.

The *pulse* may be increased in frequency when there is febrile disturbance, whether this is a consequence of the brain disease or merely associated with it; but, as we have seen, the acceleration that usually attends inflammatory pyrexia may be absent, and the pulse may even be less frequent than normal. As a direct effect of the brain disease on the cardiac centre of the vagus, the action of the heart may also be accelerated, retarded, or rendered irregular. This influence is exerted most frequently when the disease is near the medulla oblongata, but sometimes when it is distant. When intracranial disease raises the temperature to an extreme degree, the pulse often becomes very frequent and soft. Irregularity is sometimes an early indication of meningitis, especially in children. Retardation to fifty, forty, or thirty beats per minute may occur in meningitis and apoplexy, and sometimes in cases of tumor, abscess, and of increased intracranial pressure. It is probably due to over action of the centre that normally restrains the heart's action.

Respiration also is occasionally disturbed. When the cerebral functions are lowered, as in stupor or coma, the respirations are lessened in frequency and increased in depth. In meningitis and apoplexy (especially in cerebral hemorrhage) the peculiar "Cheyne-Stokes breathing" sometimes occurs, and is usually the precursor of death. The respirations present a cycle of increasing and decreasing depth; the fall goes on to actual cessation of breathing, and after a pause slight respiratory movements recommence, at first scarcely perceptible, gradually increasing in intensity, until deep dyspnœal breathing marks the acme, and the respirations again lessen. During the pause the pupils often contract, and the heart's action becomes less frequent. The period of arrest lasts from five to forty seconds, and the duration of each cycle may be from fifteen to seventy-five seconds, and may vary from day to day. The number of respirations in each cycle never exceeds thirty. This peculiar phenomenon is met with in other diseases besides those of the brain. It occurs in heart disease (in connection with which it was first studied), and in uræmia, and has also been observed (chiefly in children) in rare cases of typhoid fever, scarlet fever, diphtheria, influenza, pneumonia, whooping cough with inanition, and opium poisoning. In general maladies it is a less grave sign than in disease of the brain, and several cases of recovery are on record. In brain disease the patient is almost always comatose, but in other cases consciousness has been retained, and the symptom has been known to continue for as long as forty days.*

A form of respiration which must be distinguished from that just described consists of periods of deep and energetic breathing, which begin suddenly; the respirations gradually lessen in depth until they cease, and after a period of stillness energetically recommence. The sudden commencement is the distinction of this form. In another variety, seen by Biot† in meningitis, the periods of deep breathing not only commence suddenly, but end suddenly.

Many attempts have been made to explain the Cheyne-Stokes breathing, none of which are quite satisfactory. Walshe‡ first suggested that it is due to lessened excitability of the respiratory centre, a theory afterward stated, in more definite form, by Traube§ and by Rosenbach.‖ It is difficult to understand how the gradual character of both the onset and the decline of the breathing can be due to simple lowered excitability, although, since rhythmical action seems a character of the centre itself,¶ it is just conceivable that the larger rhythm with gradual change may be due solely to the functional characteristics of the centre, working under altered conditions. The chief attempt to give a more complete explanation has been made by Filehne,* who found that the breathing could be produced in animals under the influence of morphia, by making them inhale ether or chloroform. He observed, during the pause, an increase of blood pressure, with some pallor of the mucous membranes, and ascribed these to arterial spasm, due to stimulation of the vasomotor centres by the asphyxial blood. He assumed that a lowered irritability of the respiratory centre renders it less excitable than the vasomotor centre; that the consequent arterial spasm in the arteries of the medulla prolongs the stimulation of the respiratory centre (and also that of the vasomotor centre) by hindering the access of the oxygenated blood. Thus the respiratory movements continue energetic after the blood has become well oxygenated. In support of this vascular theory he adduced two other facts. He was able to produce perfect Cheyne-Stokes breathing (even to the changes in the pupils) by gradually constricting and then releasing the arteries conveying blood to the head; and secondly, he found that, in a patient with this breathing, nitrite of amyl, which dilates the vessels, arrested the phenomena. Filehne's theory has been criticised adversely, and some of his facts have even been called in question, but chiefly by those who have vested interests in other hypotheses. On the whole, it may be said that, unless the simple rhythmical tendency of the depressed centre is adequate to produce the phenomena, they can be best explained by the assumption that this rhythmical tendency is modified by some other periodical influence, of which vasomotor spasm is the only one which, according to our present knowledge, can be conceived as acting and adequate.† The gradual onset of the respirations may be due to the fact that the vasomotor dilatation exceeds the normal (as it often does after contraction), and thus the quantity of blood reaching the respiratory centre lessens the stimulating influence of its quality.

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* Murri, *Rivista Clin. di Bologna, 1883*, Nos. 10 and 11.

† "Sur la Resp. de Cheyne-Stokes," Paris, 1878.

‡ "Diseases of the Heart," 3d Ed., 1862, p. 345.

§ Fraenztel, Report of Clinique *Berl. kl. Wochenschr.*, No. 27, 1869.

‖ *Zeitschrift f. klin. Med.*, Bd. 1, Heft 3. Rosenbach's theory is often spoken of as distinguishable from Traube's, from which it differs, however, only by ascribing the lessened excitability to altered "interchange of material in the cells."

¶ See "Foster's Physiology," 3d Ed., p. 355.

Respiration is arrested at once by a sudden lesion in the respiratory centre of the medulla, and more slowly by disease that gradually impairs the action of the centre, such as the effusion of blood into the fourth ventricle. Indeed, the extensive connection of this centre with the brain, seen, for instance, in the readiness with which its action is altered by emotion, makes it sensitive to any considerable depression of the cerebral functions, and in many brain diseases death is thus brought about. Lesions near the centre may cause other disturbances of breathing than those described, such as simple irregularity, or paroxysmal cough, which may be semi-convulsive in character. On account of the gravity of lesions in this part, such disturbance of breathing is generally a fatal omen.

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* *Berl. kl. Wochenschrift, 1874*, Nos. 13, 14, 32, and 35.

† The superficial analogy of the phenomena to the effects of "interference" in the action of two series of rhythmical events, as the waves of sound, can scarcely fail to occur to an observer, but of course no direct inference can be drawn from it.

In all conditions in which the cerebral functions are profoundly depressed, such as coma, secretion accumulates in the air passages; bronchial and tracheal râles are thus produced, and are often erroneously regarded as the indication of bronchitis. The accumulation is due chiefly to the depression of the respiratory centre, in consequence of which the secretion is not removed, as in health, and its amount is, perhaps, increased by passive congestion. Such congestion of the lungs is very common in these cases, and is a frequent cause of death.

Digestive Organs.—Anorexia is common in febrile states, and also in conditions of lessened consciousness. It is said that bulimia is sometimes met with, but it very rarely occurs as a symptom of organic lesions.

Vomiting, however, is one of the most frequent and most important symptoms of intracranial disease. It is frequent in meningitis, tumor, abscess, at the onset of hemorrhage, and in cases of increased intracranial pressure. It may result from disease in any part of the brain, but is especially common when the cerebellum is affected. This symptom often occurs early, and may be produced by disease of very limited extent. The immediate cause of vomiting is an increased irritability of the gastric part of the vagus centre, and its occurrence in disease in various parts of the brain is another indication of the wide extent of the central relations of the vagus. It cannot be ascribed in all cases either to irritation of the membranes or to increased intracranial pressure, although these conditions are certainly capable of producing it.

In cerebral vomiting, food is generally rejected soon after it has been taken; there are no local gastric symptoms; no discomfort or pain is produced by the presence of food in the stomach, a symptom which almost always attends the quick rejection of food from gastric disease. Cerebral vomiting may be, and often is, unattended by nausea, but this is not an invariable characteristic. In some cases nausea is distressing. Occasionally the patient vomits when there is no food in the stomach, but this is not common. The disease increases the irritability of the centre, but stimulation of the peripheral nerves by food is usually necessary to excite the act. The cerebral conditions that cause vomiting usually also cause headache, and the coexistence of persistent pain in the head, with frequent vomiting, should always raise a suspicion of cerebral disease. Optic neuritis is also a common result of the same cerebral condition, and the eyes should always be examined in every suspicious case. For a long time there may be no other symptoms. It is most important, however, to note that in the early stage of disease, when the excitability of the centre is but little augmented, some exciting cause may produce the first vomiting, and the cause may seem adequate to account for it. Before the increased irritability of the centre reaches such a degree as to cause spontaneous vomiting, it may be sufficient to lead to the rejection of an injudicious meal. Such explicable vomiting is often the first sign of intracranial disease, especially in children. Patients who vomit without other indications of gastro-intestinal disturbance should be carefully watched.

Constipation frequently attends intracranial disease, such as tumor, meningitis, etc., although it rarely exists until other symptoms are apparent.

Urinary Organs.—The urine is seldom altered in composition by organic brain disease, although polyuria rarely, and glycosuria occasionally, have been produced by disease of the pons or medulla oblongata. Albuminuria occasionally occurs from the same cause, and may be a transient effect of lesions elsewhere in the brain, or of meningitis. But albuminuria, it must be remembered, is a frequent concomitant of vascular lesions in the brain, because kidney disease is one of the most potent causes of vascular degeneration. The amount of albumen excreted in these cases is often greatly increased for a few days by a cerebral lesion.

Retention of urine and overflow incontinence are common in conditions of lowered cerebral function, from whatever cause; but there is not the paralytic incontinence that is met with in spinal disease (see p. 162). Urine and fæces are often passed into the bed, etc., in cases of mental impairment, without any actual loss of power over the sphincters, and, as already stated, this symptom is always indicative of a considerable degree of mental change.

OPHTHALMOSCOPIC SYMPTOMS.

Morbid appearances in the fundus oculi are frequent in cases of organic brain disease, and are of great practical importance. Some of these are *associated*, the result of the cause of the cerebral lesion; others are *consecutive*, and are the effect of the brain disease. The two kinds are sometimes conjoined.

1. The *associated* changes are due to the constitutional condition, that is, to the ultimate cause of the brain disease, and are sometimes of the same nature as the cerebral lesion. The following are the most important: (1) Albuminuric retinitis, acute and chronic; it is associated chiefly with arterial degeneration, causing either cerebral hemorrhage or softening from thrombosis. (2) Syphilitic disease, usually choroiditis, or the atrophy that such inflammation leaves. This is often to be seen in cases of brain disease due to either inherited or acquired syphilis. (3) Tubercular disease. Tubercles of the choroid are sometimes met with in cases of tubercular meningitis. Very rarely massive tubercle forms within the eye, in cases of tubercular growths in the brain. Other forms of associated growth are so rare as not to be of practical importance. (4) Vascular lesions in the retina sometimes coincide with similar lesions in the brain. Thus embolism of the central artery may occur in the same patient, and even at the same time,* as embolism of a cerebral artery. Miliary aneurisms on the retinal arteries have been observed to coincide with cerebral hemorrhage, probably due to similar aneurisms in the brain. They are, however, extremely rare.† Hemorrhages in the retina are more common, and are of some significance. They

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* See "Med. Ophthalmoscopy," Case 47 (p. 332, 2d Ed.).

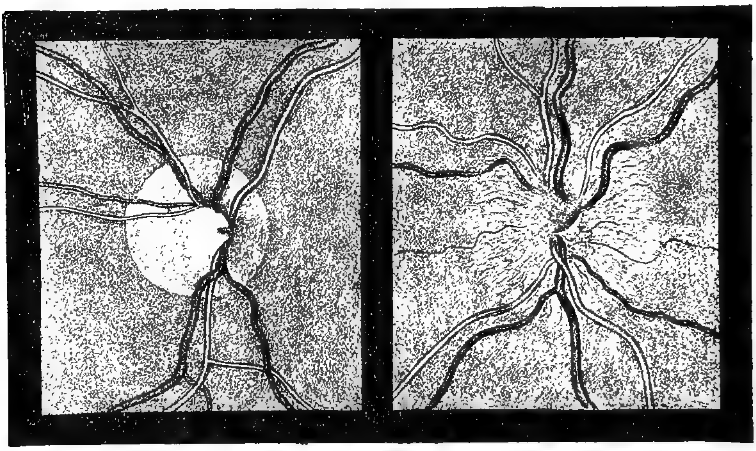
† For an instance, see "Medical Ophthalmoscopy," pl. xii, Fig. 1, and Case 42 (2d Ed., p. 326).

occur especially in albuminuria, in gout that has profoundly affected the system, in leucocythæmia, profound anæmia, purpura, and also in ulcerative endocarditis, and other forms of septicæmia. In the latter they often have white spots in the centre, and are the result of capillary septic embolism. Hemorrhages, identical in aspect, are sometimes found in the same cases in the pia mater. Another associated lesion (to which we shall return) is simply atrophy of the disc, which sometimes coincides with degenerative lesions of the brain.

2. The *consecutive* changes, which are not merely associated with the cerebral disease, but are its consequence, comprehend neuritis and atrophy.

Neuritis or "papillitis," inflammation of the intraocular end of the optic nerve, is manifested by swelling and increased vascularity of the "optic papilla." It will be remembered that the end of the nerve is visible in the fundus as the "optic disc," the boundary of which is the oval opening in the sclerotic, with the corresponding opening in the choroid. The fibres radiate from the nerve on to the retina, but not equally on all sides, being few on the temporal side. The nerve elements are so translucent that they do not obscure the edge of the disc, except sometimes, to a slight extent, above and below, where they are more numerous. The disc is rarely uniform in its surface, because the separation of the nerve fibres leaves a central hollow, in which there are few or no vessels, and which is, therefore, pale, while the periphery of the disc has a rosy tint, from the minute vessels that lie among the nerve fibres (Fig. 239). This central "physiological cup" varies much in size, and may be absent. The vascular portion of the disc also varies in extent (inversely as the cup), and varies so much in tint that no inference can be drawn from its color, unless this is observed to change in the course of time. Mere congestion, therefore, consisting only of increased vascularity of the disc, is very difficult to recognize, and although it probably occurs, it is rare, as an isolated condition, and is of small practical importance. An actual pathological change, however slight in degree, is usually attended by some swelling of the papilla, and especially by lessened transparency of its structures. The effect of this change is first to lessen the sharpness of the edge of the disc, and then to obscure the edge altogether (Fig. 239). It is to this point, therefore, that attention must be chiefly directed. The effect of a morbid change on the distinctness of the edge is greater when the disc is examined by the direct than by the indirect method; on the other hand, if the indistinctness is apparent and not real—is due to the tint of the disc being nearly that of the choroid, and not to lessened transparency of the structures in front of it—the edge appears more distinct in the direct than in the indirect method of examination. This is a very important practical point. In the early stage of neuritis the edge of the disc, seen by the indirect method, may appear a little blurred, and surrounded by a pale halo, while by the direct method the halo is resolved into a striated semi-opaque layer, completely concealing the edge. The early change is greatest on the nasal side, which may be obscured when the temporal edge of the disc, on which there are few nerve fibres, is still sharp. As the change advances, it involves the whole circumference of the disc, and the swelling rapidly encroaches on the physiological cup, which is ultimately obliterated (Fig. 239), although a slight depression usually remains in the middle of the swollen papilla. The prominence of the swelling is readily recognized by the apparent change in relative position of the structures that are on different levels, when the observer moves his head from side to side, or up and down in the direct examination, or moves the lens in a like manner in the indirect examination. The prominence is also shown by the loss of the central reflection from the vessels where they course down the sides of the swelling, and their plane ceases to be at right angles to the line of the observer's vision. The tint of the swollen papilla becomes a full red, or, more commonly, a grayish red, to the indirect examination, but the direct method shows a fine striated vascularity. As the prominence increases, the swelling becomes wider in extent, until it may be two or three times the diameter of the normal disc. White spots may appear on its surface (Fig. 240), due to the accumulation of products of degeneration. At first the retinal vessels present little change in size, but, as the swelling increases, the compression causes the veins to become broader and the arteries narrow, and extravasations of blood may be visible on the surface or margins of the swollen area. The process varies much in the rapidity with which it is developed; it may reach a considerable intensity in a fortnight, or be still moderate in degree at the end of three or four months. As a rule, the more quickly it is developed the more intense does it become. Commencing subsidence is indicated by a diminution in the vascularity, still greater contraction of the arteries, and, later, contraction of the veins also. If the

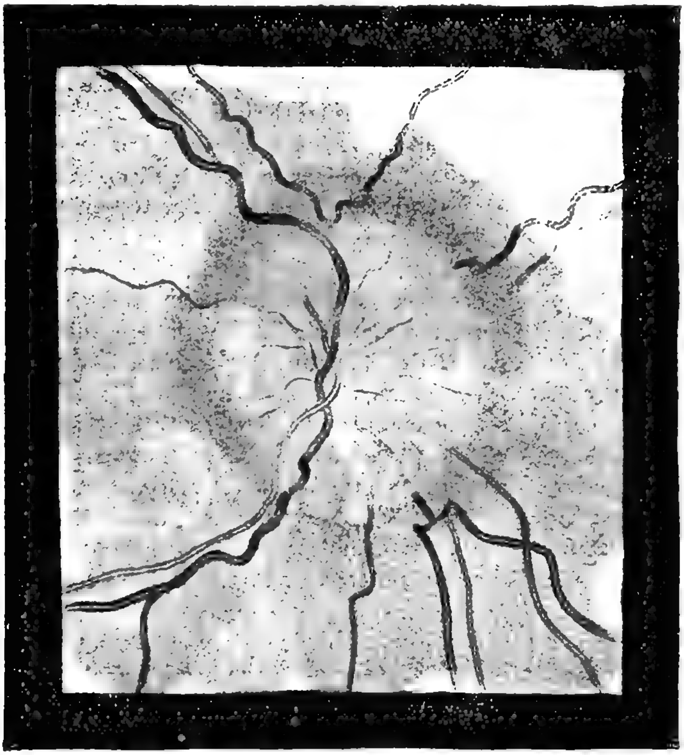
Fig. 239.



Commencing optic neuritis, from a case of caries of the sphenoid bone, with secondary meningitis. The left figure shows the normal right optic disc, with clear outline, and a deep central cup. The right figure, of the left papilla, shows well-marked neuritis; the edge of the disc is concealed by a swelling which extends beyond the normal limits of the disc. The central cup is encroached on, but not yet quite obliterated; before the neuritis commenced the cup was similar to that in the other eye. Some of the vessels are partly concealed at their emergence, and the veins lose their central reflection as they curve down the sides of the swelling.

degree of neuritis has been considerable, the swelling remains, for some time, pale and soft edged, and slowly sinks, until the edges of the disc appear. For a long time the substance of the disc is occupied by new tissue, which, with the narrowed arteries, affords evidence of the preceding inflammation. Other indications of this are often to be seen in damage to the adjacent edge of the choroid. Only when the neuritis has been moderate in degree, does the disc resume a perfectly normal aspect. When much new tissue has been formed, this, by its cicatricial changes, leaves the disc white and atrophied, in the condition of "consecutive atrophy," or "neuritic (papillitic) atrophy," as it is also termed. When the papillitis has been very intense and the swelling wide in area, the adjacent retina may suffer in its nutrition, and products of degeneration may remain as white spots, especially near the macula lutea, simulating the aspect of albuminuric retinitis.

FIG. 240.



Optic neuritis in cerebral tumor. The swelling is great, and occupies an area at least twice the diameter of the normal disc. The arteries are concealed in the substance of the new tissue, and the veins are tortuous, and lose their reflection as they pass down the side of the swelling. The products of degeneration of the nerve fibres have caused white spots near the margin of the inflamed papilla.

The substance of the swollen papilla presents, under the microscope, distended vessels, numerous capillaries and nuclei, with accumulations of leucocytes in the tissue and peri-vascular sheaths. In the early stage, the nerve fibres and connective-tissue elements may be separated by œdematous effusion. Subsequently, the nerve fibres present degenerative changes, moniliform enlargements containing myelin globules, and aggregations of the products of degeneration into "granule corpuscles," while the walls of the arteries may be conspicuously thickened. Similar, although slighter, changes may be traced back in the substance of the optic nerves, or in their sheaths, to the chiasma, and they may be even more intense in front of the chiasma than they are midway between this and the eye. The outer sheath of the optic nerve is often distended by liquid, so as to form a pyriform swelling behind the globe. The space within this sheath is continuous, behind, with the subarachnoid space around the brain, and, in front, with the lymphatic spaces in the optic papilla.

A slight and even considerable degree of optic neuritis may cause no symptoms. Acuity of vision may be unimpaired, color vision normal, the visual fields unrestricted. But, when intense, sight suffers in each of these characters, and may be entirely lost. The damage to vision is often greater during the stage of subsidence than it is during the active period of inflammation, probably because the nerve elements suffer from the cicatricial contraction of the tissue elements formed during the inflammation. But the affection of sight is not necessarily due to the visible papillitis; it may be the result of more intense inflammation behind the eye, or the effect of the intracranial disease. To decide this, the degree of neuritis and the degree of affection of vision must be compared, and the character of the failure of sight must be considered. The latter is described in the chapter on affections of the optic nerve. After the neuritis has quite subsided, some improvement in sight often takes place, from the recovery of some of the damaged fibres. But there may remain considerable amblyopia, and even absolute blindness, and often very irregular changes in the fields of vision, both for white and colored light.

Optic neuritis may result from many diseases of the brain, but its most frequent cause is tumor, and a considerable degree of neuritis is rarely due to any other cause. Next in frequency is meningitis, especially affecting the base of the brain, but the papillitis caused by meningitis is usually less intense than that of tumor, perhaps on account of the briefer duration of the disease. Cerebral abscess is another cause. Optic neuritis scarcely ever results from cerebral hemorrhage or from thrombotic softening, but it has been several times observed in cases of softening from embolism, when the source of the obstructing plug was acute endocarditis, perhaps because the material carried from the valves is of an irritant septic character, and determines a greater degree of secondary inflammation in the softening produced. It has also been met with in a case in which no naked-eye lesion of the brain existed, but in which the microscope revealed general slight inflammatory changes—diffuse cerebritis. In all these diseases the neuritis is, as a rule, double, although it may develop more rapidly in one eye than in the other. Occasionally it is one sided, and then it is generally on the side opposite the lesion. Unilateral optic neuritis is much more frequently due to various diseases at the back of the orbit, or at the optic foramen, than to diseases of the brain itself.

It must be remembered, however, that optic neuritis may result from other causes than intracranial disease. It is very seldom primary; but it occurs in chlorosis, albuminuria, lead poisoning, anæmia, and after certain acute fevers, especially scarlet fever and typhoid; in many of these conditions its occurrence is associated with some cerebral symptoms. When neuritis preponderates over the other changes in albuminuria, for instance, there is usually much pain in the head, and in lead poisoning it is usually associated with the acute cerebral symptoms which have been called "encephalopathia saturnina." This fact sometimes increases the difficulty in diagnosis.

The mechanism by which optic neuritis is produced is a subject on which various opinions have been held * It is sufficient here to say that the early theory that neuritis is due to increased intracranial pressure, acting mechanically, has proved erroneous. I have known slow increase of intracranial pressure to separate the sutures without causing papillitis. In a large number of cases there is distinct evidence of a descending inflammation, either along the trunk of the optic nerve or along its sheath, and in cases of meningitis such descending inflammation is invariable. The distention of the optic sheath is not essential for the production of neuritis; it is sometimes absent, and its occurrence is related especially to the presence of an excess of subarachnoid fluid. The signs of mechanical "strangulation," which are to be observed in cases of intense neuritis, are no evidence that the inflammation was caused by any mechanical process. The cause of the strangulation is the compression of the veins by the inflammatory products within the swollen papilla, and not, as was at first thought, compression within the sclerotic ring, or behind it, by the distention of the sheath. Within the ring the vessels, after death, never exhibit any sign of narrowing, whereas, within the papilla, their compression is usually distinct. The conclusion is, that optic neuritis is probably rarely due to a single factor, that the most potent element is the descent of a process of tissue irritation, which, when it reaches the papilla, sets up a more intense inflammation; that in some cases this factor is alone effective; that in many others its influence is aided by the effect of distention of the sheath, hindering the escape of effete liquids, increasing the œdema, or even conveying irritating material. The distinction between optic neuritis and "choked disc" is one of degree, not of cause. So far as optic neuritis has any single significance it is that of the presence of an irritative process within the skull.

There is an important relation between the chronicity of the neuritis and that of the intracranial process. A chronic cerebral process may cause an acute neuritis, but a chronic neuritis never results from an acute process, and the degree of the chronicity of the neuritis is an indication of the degree of chronicity of the intracranial disease. When the cerebral pro-

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* A full account of these will be found in "Medical Ophthalmoscopy," 2d Ed., p. 65.

cess begins to improve, the neuritis lessens, and the commencing subsidence of the neuritis is often the first indication of the improvement in the brain lesion.

Atrophy.—Of the three forms of atrophy of the optic nerve, *primary*, *secondary* (to disease of the nerve further back), and *consecutive* (to neuritis), only the two latter occur as a result of uncomplicated intracranial disease. Apparent exceptions to this are disseminated sclerosis and general paralysis of the insane: in both of these affections primary atrophy may occur, but in these there is widespread degeneration, usually involving the spinal cord as well as the brain, and the atrophy must be regarded as associated rather than the result of the brain disease. Consecutive (neuritic or papillitic) atrophy, the characters of which have been already described, has the same causes and the same significance as the inflammation of which it is the consequence.

Secondary atrophy is the result of damage to the optic nerve behind the eye, or to the optic chiasma. Its characteristic is that the signs of atrophy follow instead of accompanying the failure of sight. The causes of this form of atrophy will be described in the chapter on diseases of the optic nerve.

DISEASES OF THE CRANIAL NERVES

AND DERANGEMENT OF THEIR FUNCTIONS.

Many symptoms of organic brain disease consist in derangement of the functions of the cranial nerves, and it is convenient to consider these together with the diseases of the nerves themselves. Such an arrangement is not strictly logical, but its practical convenience is great, since much needless repetition is thereby avoided, and those symptoms can be described together that are of the same character, and have to be considered together in practical diagnosis. The paths of the cranial nerve fibres within the brain have been already described, and need not be here repeated, except in so far as they have a bearing on special points.

OLFACTORY NERVE—AFFECTIONS OF SMELL.

The olfactory bulb contains many nerve cells, and has been thought by some to be analogous to the nuclei of the other cranial nerves; by others to be a detached portion of the cerebral cortex. The bulb is connected with the cerebral hemispheres by the olfactory nerve, the central connections of which have been described on p. 492. We have seen that each nerve seems to be related to both hemispheres, since the sense of smell on one side may be deranged, not only by disease of the hemisphere (uncinate gyrus) on the same side, but also by a lesion of the hinder part of the internal capsule on the opposite side, and probably, also, by disease of the convexity.

The sense of smell includes more than is popularly assigned to it. It includes not only the recognition of odors, but also that of flavors. When the olfactory nerves are influenced through the anterior nares, the sensation is termed an "odor;" when through the posterior nares, it is termed a "flavor." In the latter case, the sensations are blended with those of taste proper, bitter, sweet, sour, etc., and the two are confused. If the sense of smell is lost, all perception of flavors is lost with it. Hence, patients who have lost smell usually say that they have also lost taste, and sometimes they only mention the loss of "taste," which is, to most persons, the more serious deprivation.

In testing the sense of smell, care must be taken to employ only substances (such as musk, asafœtida, or oil of cloves) that act on the olfactory nerve alone, and it is best to use such odors as are readily identified and named. If volatile pungent substances are used, such as ammonia or acetic acid, which also stimulate the fifth nerve in the nasal mucous membrane, the patient may perceive that which he cannot smell.

Loss of the sense of smell, anosmia, is less frequently due to a lesion of the nerve than to disease of the mucous membrane of the nose, chronic inflammation, nasal polypus, etc. It may also be caused indirectly by paralysis of the fifth nerve, and consequent trophic changes in the membrane, or dryness from lessened secretion. The olfactory nerves may be damaged by falls or blows on the front or back of the head; the delicate filaments are torn from the bulb, or are lacerated in their passage through the ethmoid bone. The olfactory bulbs sometimes suffer, by compression or inflammation, in cases of tumor in the anterior fossa of the skull, caries of adjacent bone, and local meningitis, syphilitic or other. They may also be compressed by internal hydrocephalus.* In these cases the bulbs may be distinctly involved in the disease, or may be flattened and wasted. Tumors have been supposed to cause an olfactory neuritis, analogous to optic neuritis. In locomotor ataxy, anosmia occasionally occurs, although not frequently. Primary atrophy of the bulbs is occasionally met with in old age (Prévost). Lastly, excessive stimulation of the olfactory nerves has caused their paralysis, just as blindness has resulted from a very brilliant light; in several cases, after exposure to an exceedingly strong odor, the sense of smell has been permanently lost. Thus, an officer in Ireland superintended the emptying of a cesspool, at the bottom of which some weapons were said to be concealed; the odor was most offensive. Next day he found that his sense of smell was gone, and it never returned. Other similar cases are on record. It has been conjectured that the odor causes congestion and hemorrhage into the mucous membrane, because, if the musk-deer hunters do not stuff up their nostrils before extracting the musk gland, they suffer from epistaxis.† But it is unlikely that capillary hemorrhage into the mucous membrane would cause complete anosmia, or would occur without epistaxis. More probably the loss of smell is the direct result of the over-stimulation on the nutrition of the nerve elements.

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* Quincke, *Cor. Bl. f. Schweiz. Aerzte, 1881,* No. 9.

† Althaus, *Lancet, 1881,* vol. i, p. 771, where some interesting information on the history of our knowledge of anosmia will be found.

In diseases of the cerebral hemisphere loss of smell is rare. It has occurred with symptoms of embolism of the middle cerebral (Hughlings Jackson), perhaps from damage to the root fibres at the commencement of the fissure of Sylvius, since the loss of smell has been on the same side as the lesion. It has also been observed, on the opposite side, in cases of disease of the "sensory crossway," at the hind part of the internal capsule,* and in at least one case of extensive disease of the convexity.† Functional loss occurs only in hysterical hemianæsthesia. The olfactory nerves are sometimes congenitally absent. Dr. Sharpey, in his lectures on physiology, used to mention the instance of a chief scavenger of the city of Leipzig, who was known to have no sense of smell, and in whose brain no trace of olfactory nerves could be found. A few similar anatomical facts are on record; in most cases a minute gray prominence marked the place from which the nerve normally springs. In other cases congenital absence of the sense has been noted during life, and has been known to descend from father to son (Breschet). It does not seem certain, however, that the absence of the nerves invariably entails absence of the sense. In some cases there has been strong reason to believe that the individuals possessed the sense.‡ The only conceivable explanation is that suggested by Duval,§ that the fifth nerve subserved the olfactory function.

The diagnosis of an affection of the nerve depends, first, on the detection of the loss of smell, and secondly, on the exclusion of disease of the mucous membrane. In the latter case, purulent discharge has generally occurred at some period, and a history of such discharge always affords strong reason for suspecting a local cause. Loss of smell, with ability to recognize flavors, is always due to such disease, partial in extent. The importance of a local examination is obvious. A lad was once sent to me who was said to have lost smell, taste and hearing; I was asked where the brain disease was likely to be to cause these effects. There was scrofulous inflammation of the nasal mucous membrane, caries of both ears, and neither loss of taste proper, nor any evidence of intracranial mischief.

Besides the ordinary tests of smell, the nerves can be stimulated by voltaic electricity, a phosphorus-like odor being perceived. This is proof of their integrity, but the application is very painful unless the nose is filled with water, or, better, a weak saline solution, in which the rheophore is placed. In health the order of response has been thus found to be similar to that of other nerves: (1) KCC, (2) AOC. The induced current has no effect. An electrical examination can very seldom be necessary. The fact that disease of the fifth nerve may cause considerable anosmia must not be forgotten in diagnosis.

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* See note on p. 461.

† The case of Demange, p. 461.

‡ Case of Bernard, quoted by Althaus, *Lancet, 1881*, i, 772, and of Lebec, *Prog. Méd.*, No. 48, 1883.

§ *Bull. de la Soc. de Biol.*, Paris, No. 17, 1883.

The prognosis of disease of the olfactory nerve is generally unfavorable. Considerable improvement has been observed only in cases of syphilitic disease of short duration, and in some traumatic cases.

The treatment is mainly that of the cause of the anosmia. The local application of strychnia to the mucous membrane has been recommended—as a solution in olive oil (Brunniche), or as snuff, $\frac{1}{24}$ grain mixed with sugar (Althaus). In cases in which there is reason to believe that the active disease has ceased, voltaic electricity has been recommended, the positive pole being placed behind the mastoid process, the negative to the nasal bones. Only a few cells can be used, or unpleasant giddiness will be produced. In one recorded case, after a fall on the back of the head, considerable improvement followed two applications (Jacob). Of course, all that electricity can do is to excite the nerve elements that are structurally perfect, but for some reason functionally inactive, and in most cases of anosmia it is useless. Iodide of potassium has also been recommended, but its value, except in cases of syphilitic origin, is doubtful.

Increased sensitiveness of the olfactory nerves—"olfactory hyperæsthesia," "hyperosmia"—is occasionally met with. It occurs chiefly in hysteria, in which remarkable, almost animal, acuteness of the sense is sometimes present, so that not only objects but persons have been discriminated by this means. In insanity the same condition is sometimes met with. It is usually associated with, and has to be distinguished from, an altered appreciation of odors, shown in the abnormal enjoyment of, or disgust at, the odors which are recognized with natural or preternatural acuteness.

The condition rarely calls for special treatment. Morphia in snuff or hypodermic injection has been suggested, but the local application of cocaine will probably be found more effective.

Subjective Sensations of Smell.—Subjective sensations of smell occur from central disease or from irritation of the nerve. In the insane, olfactory hallucinations occur, though less commonly than those of the optic or auditory nerve. Schlager met with them in five cases out of six hundred. In epilepsy, subjective sensations of smell occur as occasional warnings of fits, and disease in such a case has been found to involve the olfactory region in the anterior part of the temporo-sphenoidal lobe. It was so in a case of tumor, recorded by Sander, and also in one of softening of this region.* A subjective sensation (usually resembling that of phosphorus, and such as is produced by electrical stimulation of the nerve) sometimes precedes loss of smell from organic disease, as by a tumor. It was so in a case of loss of smell in tabes recorded by Althaus. Doubtless the sensation is due to the irritation of the nerve elements by the morbid process which ultimately destroys them.

Perversion of the Sense of Smell.—This is a rare condition, which occasionally results from irritation of the nerve or central organ. In a case recorded by Legg, some time after an injury to the head, all substances "tasted" of gas or paraffine, and there was marked diminution in the acuteness of the sense of smell.

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* Hamilton, *New York Med. Jour.*, June, 1882.

OPTIC NERVE AND VISUAL SYMPTOMS.

The central relations of the optic nerve have been described at p. 490, and the cortical centres for vision at p. 462. The fibres from the region of the macula lutea are at first in the outer part of the nerve, and reach its axis near the optic foramen. Each optic tract contains the fibres from the same named half of each retina, *i. e.*, from the temporal half of the retina on the same side, and the nasal half of the retina on the other side, and hence disease of the tract causes loss of vision in the opposite half of each field, the temporal half of the one, the nasal half of the other. This is termed *homonymous hemianopia*,* or *lateral hemianopia.*

Why do more fibres cross to the opposite tract than pass to the tract on the same side, if the two sets come from equal retinal areas? Because the prominence of the nose shuts off the rays from the peripheral part of the temporal half of the retina, and the power of sight extends but little further than the area habitually stimulated. Hence, while the retinal halves are of equal size, the functional area is smaller on the temporal side, and fewer nerve fibres proceed from it.† The fibres from the temporal side are those that do not cross, and hence the crossing fibres are more numerous than those that pass in the tract without decussating. The fibres which cross occupy the middle of the chiasma, the direct fibres lie on each side. Fibres from the macula lutea and region around it pass, in most individuals, at least, to both optic tracts. The division between the two halves of the retina (from which fibres pass to the respective tracts) is generally in the vertical line of the macula lutea, but it probably varies in its precise direction in different individuals, and seems sometimes to be slightly oblique. These two facts will be considered more fully in the description of the symptom. The fibres which pass from one tract to the other by the posterior part of the commissure (and are probably connected, by the internal geniculate bodies, with the corpora quadrigemina) have not, at present, any medical importance.

We have seen that the visual path passes, on each side, by the optic tract to the optic thalamus, and thence by the optic radiation to the cortex of the occipital lobe. This constitutes a half-vision centre which seems to be double, for light and for colors. The two half-vision centres are, probably, blended in a higher visual centre in front of the occipital lobe. The arrangement is, however, very complex; in each centre both fields are represented, but chiefly that of the opposite eye. Each higher visual centre can supplement its fellow, to some extent, and compensate its loss, but in the half-vision centres there is no power of supplemental action, and no compensation for loss is possible.

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* It was formerly called "hemiopia," but the anomaly of the use of the term "half sight" in the sense of "half blindness" has led to a general endeavor to employ the more exact "hemianopia" or "hemianopsia." Although it is seldom desirable to change a familiar word, the alteration, in this case, is trifling.

† Semi-decussation of the optic is not the invariable rule in the animal kingdom. In some creatures there is a total decussation. The amount of decussation seems to be roughly proportioned to the amount of separation of the fields of vision. In man the fields half overlap when the eyes are directed forward, and there is a half decussation. In those animals in which the fields are totally distinct, and cannot be made to overlap, as fishes, there is a total decussation.

ETIOLOGY.—The numerous affections of the optic nerve within the eye do not come within the province of this book, nor does the primary atrophy, which so often accompanies certain degenerative diseases of the spinal cord, and occasionally complicates similar diseases of the brain. Optic neuritis, or "papillitis," such as results from intracranial tumors, is rather a general symptom of cerebral disease than a special affection of the nerve, and has been considered with other symptoms of the same class (p. 561). Moreover, interstitial diseases of the nerve itself, behind the eye, such as interstitial neuritis, or hemorrhage into the substance of the nerve, also fall into the province of ophthalmology.

The diseases that are of medical significance may be divided according as they affect the nerve, the chiasma, or the path from the chiasma to the cortex. This classification corresponds to an important difference in the symptoms produced.

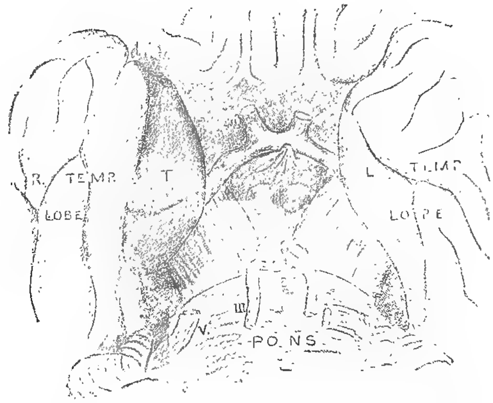
(1) The *nerve* itself may be damaged by tumors within the orbit or within the skull. The intracranial course of the nerve is, indeed, so short that it is seldom thus affected, but it is occasionally compressed by a tumor of the pituitary body, or by one springing from the bone. The nerve may be damaged by an aneurism of the ophthalmic artery within the orbit, or of the internal carotid within the skull. It is occasionally the seat of inflammation, arising in the sheath or spreading to it from an adjacent focus, such as caries of the sphenoid bone or an orbital abscess. It often becomes inflamed in meningitis, but symptoms result chiefly through the agency of papillitis. A rare but important form is rheumatic inflammation. Of this I have seen three instances; all were in women, and two had previously suffered from a neuritis of the facial nerve and from other manifestations of rheumatism. The symptoms followed exposure to cold. In two cases the nerves to the eyeball muscles were also involved, and from this it is probable that the seat of this inflammation is at the back of the orbit. The optic nerve is occasionally injured in falls on the head, either by laceration at the optic foramen or by hemorrhage.

(2) The *chiasma* is damaged most frequently by tumors arising in its neighborhood, especially by those springing from the pituitary body, sometimes by tubercular or syphilitic growths, in the chiasma or outside it, rarely by chronic inflammation in its vicinity, commencing in the dura mater and leading to thickening of the tissues about the commissure. Another occasional cause of damage is internal hydrocephalus; the distended infundi-

bulum of the third ventricle presses on the middle of the chiasma and flattens it, as Cheselden noted a century and a half ago.* It is probable that it is sometimes the seat of interstitial inflammation. Symptoms of a local lesion at the chiasma occur in rare cases of tabetic atrophy of the optic nerve, and may develop slowly or rapidly. In one case which I have recorded, the characteristic loss of sight came on quite suddenly. A sudden onset has been observed in other more simple cases, and suggests a vascular lesion. Interstitial hemorrhage has been actually observed, apart, however, from visual symptoms.†

(3) The *optic tract* is chiefly damaged by tumors, either of the base or springing from the inner part of the temporo-sphenoidal lobe, which may either compress the tract or invade it. An instance of such compression is shown in Fig. 241; although it is doubtful whether the hemianopia from which the patient suffered was thus produced, since the disease began in the hemisphere, in the neighborhood of the optic radiation, and the hemianopia was an early symptom. (The cerebral growth is figured in the chapter on Intracranial Tumors.) If the growth *invades* the tract, it generally spreads also to the crus, as in a case in which such a tumor caused first hemianopia and then hemiplegia.‡ The tract is seldom damaged by softening or hemorrhage; partial softening has, however, been observed to result from thrombosis of the internal carotid. It is sometimes the seat of an islet of disseminated sclerosis.

FIG. 241.



Tumor of right temporo-sphenoidal lobe, compressing the right optic tract, but previously interrupting the optic radiation. (See chapter on Intracranial Tumors.)

The intra-cerebral path may be damaged by softening, hemorrhage, tumors, or traumatic lesions. These may involve the thalamus, posterior part of the internal capsule, white substance of the occipital lobe, or cortex.

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* *Phil. Trans.*, No. 337, p. 281.

† For details of lesions, see Wilbrand, "Hemianopsie," Berlin, 1881, and Hill Griffith, *Med. Chronicle*, January, 1887.

‡ *Cent. f. Med. Wiss.*, *1878*, No. 31.

INVESTIGATION OF THE FUNCTIONS OF THE OPTIC NERVE.—The manifestations of disease consist in alterations of the fundus oculi, and in impairment of vision, or of the reflex action of the iris to light. All these should be carefully examined in every case; and it is often necessary, also, to ascertain the state of refraction in the eye, lest a defect of sight should be ascribed to disease of the visual path or centre, when it depends solely on an error in refraction. This is of great importance, especially since a considerable deviation from the normal refraction is apt to set up other functional disturbances, pain, etc., which may lead to a suspicion of organic disease. The suspicion may be apparently confirmed by the impairment of sight. On the importance of an ophthalmoscopic examination it is needless to insist; without it, amblyopia may be ascribed to brain disease, when it is of purely ocular origin, and many morbid processes in the brain reveal themselves in the optic papilla. The more important changes in the fundus have been already mentioned (p. 561).* The mode of testing the action of the pupil will be described in the chapter on Diseases of the Third Nerve.

The *examination of vision* includes several points, each of which needs careful investigation. (1) The acuity of vision, for which test types are employed, and the result is expressed in a fraction of which the denominator is the distance at which the type can be read by a normal eye, and the numerator is the farthest distance at which it can be read by the patient. (2) Color vision may be tested in two ways: (*a*) by the method of "confusion," in which the patient selects from a series of colored objects those that are of the same kind of color as a standard, and reveals a defect by picking out tints that are essentially different; (*b*) by asking the patient to name certain colors. This is open to the fallacies that a name may be given accurately when a color is not rightly perceived, and that the patient may not be familiar with the names of colors. The former fallacy is especially dangerous in cases of congenital color blindness, in which colors may appear different, and be associated with certain names in the mind, when they are not rightly perceived. The method of naming colors is, however, more often useful in medical than in ophthalmic work; if a patient is known to have previously had good color vision, or if one eye only is affected, and the vision with it can be compared with that of the other eye, the method of naming colors often gives valuable information, especially as to the character of the sensation excited. (3) The fields of vision must be examined, and if any defect is found they should be mapped out, if possible, by means of a "perimeter." The field may be roughly tested by moving some object midway between the observer and the patient, the eye tested being fixed on that of the observer, who can thus readily detect any movement of the patient's eye from the fixing point toward the object, and, at the same time, can use his own field of vision as a standard. For a very rough examination the hand may be employed, but it is better to use a small piece of white paper, one-third of an inch square, fixed on the end of a dark-colored rod, such as a penholder. With a little care, very exact observations can thus be made, although the result cannot be recorded so precisely as by means of a perimeter. In this instrument an object is made to move along a quadrant of a circle, divided into degrees, and rotating on one of its extremities (at which is the fixing point) so that the quadrant can be placed in each radius of a hemisphere of which the patient's eye is at the centre. The result is recorded on a chart in which are concentric circles divided by the radii, at intervals of ten or more degrees. The field does not extend equally in all directions, being limited above by the projecting eyebrow, and on the inner side by the nose. In the diagrams here given only that part of the chart is shown which is included within the average normal field, and for the sake of simplicity, only the radii and concentric circles at 30° distance are represented, although many more were employed in taking the fields.

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* The reader will find a full account of them in "Medical Ophthalmoscopy," 2d Ed.

It is important, in testing vision, to know that in conditions of functional feebleness of the nervous system, such as hysteria and so-called neurasthenia, the sensibility of the peripheral parts of the retina lessens considerably after attention has been directed to them for a short time, so that even normal fields become smaller when taken several times consecutively, the reduction being confined to the part tested. This effect is still more marked when there is a pathological contraction. It seems to be produced, not on the retina, but on the centre, because, if one part of the field is tested for a short time, and becomes narrower in consequence, the same narrowing is found to have taken place in the corresponding part of the field of the other eye.*

It is necessary, in a complete examination, to test the field for colors as well as for white, since defects may be found in the color fields where none exist for white. Small pieces of colored paper may be employed in the same way as a white object. The normal fields for colors are smaller than those for white, and vary for each color; beyond their limit the object can still be seen, but appears gray instead of colored. The fields for red and green are the smallest, and undergo most narrowing in morbid states; it is, therefore, generally necessary to test only these fields. If the power of distinguishing objects is lost in any part of the field, it is desirable to ascertain further whether light can still be perceived in the amblyopic region.†

SYMPTOMS.—*Optic Nerve.*—Disease of the nerve impairs or abolishes vision in one eye, and lessens the reflex action of the pupil in proportion to the interference with vision. The ophthalmoscopic appearances may be at first normal, but if the damage is considerable the optic disc slowly passes into a condition of atrophy. This is termed "secondary atrophy," to distinguish it from the "consecutive atrophy" which follows papillitis. When the nerve is damaged by inflammation, it is not uncommon for slight neuritis to be visible within the eye, but this is usually too trifling in degree to account for the loss. The impairment of sight involves acuity of vision, and commonly also the field. A common change in the field is concentric contraction, because the peripheral layer of the nerve is most damaged by processes which begin outside it. An irregular defect is not rare. Such a defect is shown in Fig. 242, from one of the cases of rheumatic neuritis mentioned on page 571. In the two others the loss of sight was absolute and lasting. In all, visible atrophy of the nerve slowly supervened without any recognizable papillitis. A central loss is much less common, and is due to what is termed "axial neuritis," inflammation (or degeneration) in the centre of the nerve. The affected fibres only occupy the centre of the nerve at the back of the orbit; further forward they come to (or near) the surface on the temporal side.* This form is met with in "tobacco amblyopia," and it sometimes complicates degenerative diseases of the brain, but does not, strictly speaking, result from them.

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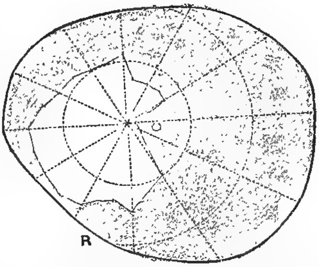
* Schiele, *Archiv f. Augenheilk.*, Bd. xvi.

† It has been recently asserted that the perception of light depends on a different centre from the perception of form (Wilbrand). It is not yet certain that this is so, but observations on this point are desirable.

FIG. 242.



Limitation of R. field, greatest in right half, due to orbital neuritis; onset acute, with palsy of ocular muscles and a normal disc; subsequent recovery of the muscles and slow atrophy of the disc.

Chiasma.—The characteristic symptom of disease of the chiasma is "temporal hemianopia," a loss of the outer half of each field of vision or of part of this half. This temporal hemianopia (shown in Figs. 243 and 244) is the common symptom, because the morbid processes which damage the chiasma act chiefly on its central portion, and affect the decussating fibres from the nasal half of each retina. In many cases, however, the morbid influence does not remain limited to the central portion, but involves one side of the commissure, or one tract just behind the commissure, or, less frequently, one optic nerve in front of it. In either case the result is the same; on the side toward which the morbid process extends, the non-decussating fibres are involved and the corresponding eye becomes totally blind, while in the other eye the loss remains limited to the temporal half field. Often the whole chiasma ultimately suffers, and then the impairment of vision progresses through the stages just mentioned to total loss in both eyes. The rarest of all symptoms of disease of the chiasma is nasal hemianopia. It can only result from a lesion which involves each side of the chiasma, and spares the central portion. Only an external inflammation of peculiar distribution, or a symmetrical interstitial affection of the nerve, can produce this effect, and in practical experience this is, perhaps, the least frequent of all affections of sight. The only case that has come under my own observation was one of tabetic atrophy, and is not, therefore, simple. In one eye there was a complete loss of the lower nasal quadrant, extending, however, beyond the middle line; in the other a well-defined amblyopia of corresponding situation (see Fig. 245).

In the ordinary form of temporal hemianopia the dividing line is seldom exactly vertical. The loss in the two eyes sometimes corresponds closely; more often there is a difference between the two fields. Strict correspondence depends on the strictly central position of the lesion, which seldom obtains in the case of the processes which cause the loss. Sometimes the

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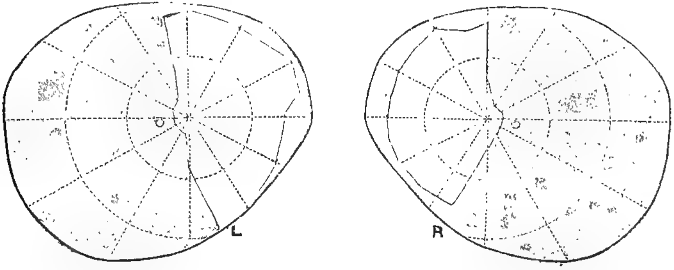
[*] Nettleship and Edmunds, *Trans. Oph. Soc.*, vol. i, p. 124; Samelsohn, *Cent. f. Med. Wiss.*, *1880*, 418, and *Trans. Med. Congress*, *1881*; Vossius, *Archiv f. Oph.*, xxviii.

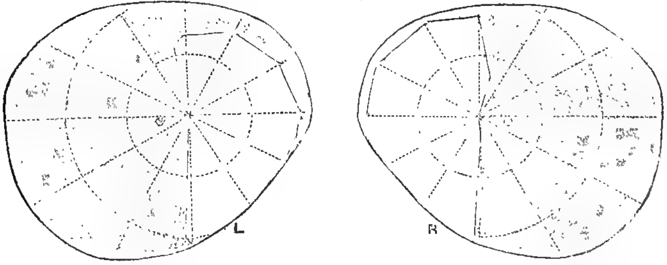
dividing line appears to pass through the fixing point, as in Fig. 244; in other cases it diverges to the temporal side of the fixing point, so as to leave a small area around this within the seeing half. The difference probably depends on individual variations in the decussation; in some persons fibres from the macular region pass in both divisions of the nerve, the crossing and the direct (see "lateral hemianopia," p. 583). In most cases there is no peripheral contraction of the half fields that remain. When such contraction exists, it is probably due to damage by inflammation to the peripheral layer of the optic nerves in front of the chiasma. The loss of sight in temporal hemianopia involves all forms of vision; the color loss has the same limit as that for light.

FIG. 243.



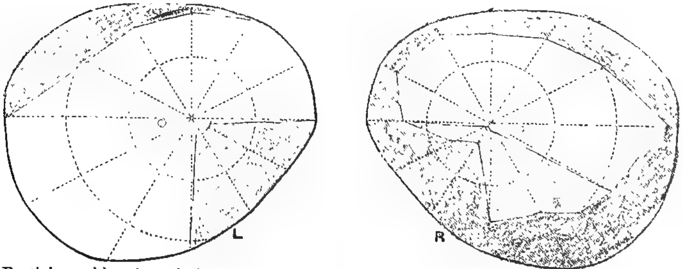
Temporal hemianopia, gradual onset; no other symptoms. Not progressive.

FIG. 244.



Temporal hemianopia without other symptoms than some headache; possibly a tumor of the pituitary body.

FIG. 245.



Partial nasal hemianopia in a case of optic nerve atrophy. The quadrantic loss in the left field was not absolute; in the right the darker shading indicates the absolute loss.

The mode in which temporal hemianopia comes on depends on the nature of the morbid process which causes it. The onset is usually gradual, since the lesion is commonly chronic. Occasionally it develops rather rapidly, and then inflammation, secondary to the primary lesion, is probably its immediate cause. Very rarely the onset is actually sudden.

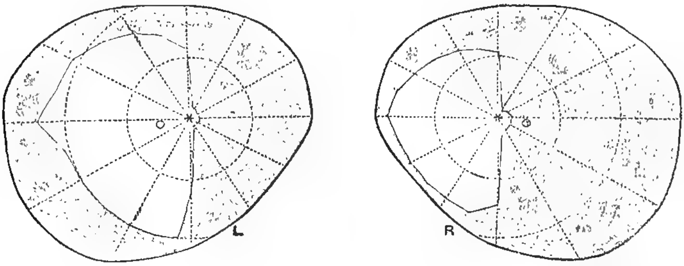
The associations of temporal hemianopia vary according to its cause. The most frequent is paralysis of some of the adjacent motor nerves to the eyeball muscles, but this complication only occurs when the primary disease is outside the chiasma. Other structures may suffer if a tumor attains a large size. Optic neuritis does not often result from the cause of the hemianopia, certainly less frequently than might be expected; but hemianopia sometimes *succeeds* optic neuritis, when the commissure suffers compression from a distended third ventricle, and the cause of the effusion is a subtentorial tumor. Thus, a man with a stationary tumor, probably syphilitic, and symptoms of internal effusion, has considerable amblyopia from optic neuritis which had subsided into atrophy, when he gradually developed temporal hemianopia, and then became completely blind, without any change in the condition of the fundus oculi.

The course of temporal hemianopia necessarily depends on that of its cause. As we have seen, it may go on to complete blindness; failure of the remaining half field of one eye may leave the patient only the nasal half of the other field during the further course of the intracranial lesion to the fatal issue which progressive disease usually entails. But the symptoms are not progressive in a considerable proportion of the cases of temporal hemianopia. In many instances a total loss of the temporal half fields remains without extension. Less commonly considerable improvement occurs. This more favorable course is met with chiefly in cases in which the symptom has developed rapidly or suddenly, and is unaccompanied by indications of pressure on adjacent structures. Now and then such hemianopia of gradual onset does not advance. We are necessarily ignorant of the nature of the lesion in such cases. It is conceivable that this may be a tumor of the pituitary body, the growth of which is arrested, or an interstitial process, inflammatory or vascular. Interstitial inflammation is, perhaps, the more probable.

Tract and Hemisphere.—The loss of sight from disease of the visual path behind the chiasma, "homonymous hemianopia," depends on the anatomical arrangement, already described. There is an arrest of the impressions from the side of each retina corresponding to the side of the lesion, and hence there is a loss of vision in the opposite half of each field.

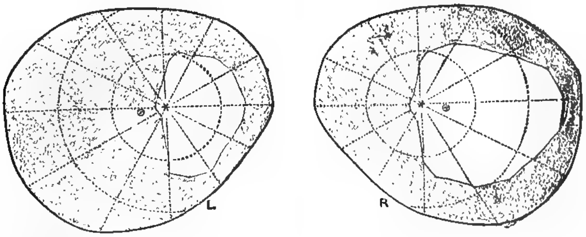
It is equally common for the loss to be on the right side and on the left. Of cases collected by Wilbrand, eighty were on the left side and seventy-four on the right. The half loss varies in its extent. In cases of complete hemianopia it extends up to the vertical middle line (Fig. 246), but the precise character of the dividing line varies considerably in its minute characteristics. The first important difference is, that in some cases the line seems to pass through the fixation point, while more frequently (as in most of the cases figured) the fixation region* is included in the seeing half.† The line of division may occupy the vertical meridian above and below, but about 8° or 10° from the fixation point it diverges, and curves round this point, at about the same distance from it, to regain the middle line below (Fig. 246). Sometimes the divergence occurs some distance above the fixing point (Fig. 247). The central area thus enclosed varies from 3° or 4° to 10° or even 15° radius. In other cases again, the line continues in the meridian, and seems to pass through the fixing point itself. Rarely, it seems to pass through the fixation point in one eye and beyond it in the other. In a third group the line diverges from the meridian, and, when near the level of the fixing point, turns toward it and seems to reach it, so as to make a reëntrant angle in the outline of the seeing half (Fig. 248). This important difference, the division through the fixing point, or the inclusion of the fixing point in the seeing half, does not depend on the position of the lesion; each form occurs in cases of hemianopia due to disease of the tract, thalamus, or hemisphere, and we have already seen that each form occurs also in

FIG. 246.



Right hemianopia; sudden onset with right hemiplegia and hemianæsthesia, which passed away in a few days, leaving the hemianopia the only permanent symptom.

FIG. 247.



Left hemianopia from the case of cerebral tumor shown on p. 572, but probably due, not to the pressure on the tract, but to the interruption of the optic radiation.

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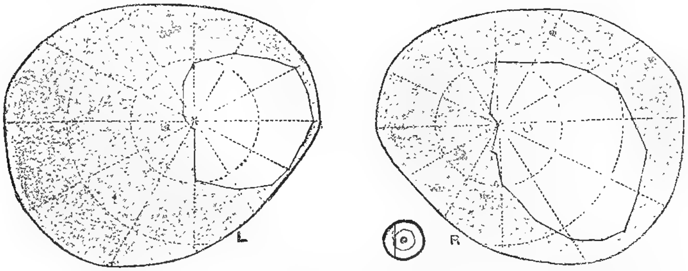
* The region around the fixation point, subserved by the central region of the retina around the macula.

† Wilbrand found that of fifty-six cases in which the point was specified, the line seemed to pass through the fixation point in twenty-three cases, and to one side of it in thirty-three. See, however, the remarks on p. 579.

cases of temporal hemianopia from disease of the chiasma. The difference can only be explained by individual variations in the decussation of the nerves. The inclusion of the central region in the seeing half occurs equally in right and left sided hemianopia. It must be due, therefore, to the passage of fibres from the macular region to both optic tracts and both hemispheres, so that this region is not blinded by disease of either tract.*

Since the distance from the fixing point at which the dividing line passes varies, in different individuals, it appears that there is a corresponding variation in the size of the central area, from which fibres pass to both tracts. This area is very small when the line passes very near the fixing point, and if it passes through the fixing point itself there is no double passage of the central fibres, but a division of these as of those from the rest of the retina. Recent careful observations, however, lead me to doubt whether the dividing line ever passes actually through the fixing point. When it has seemed to do so, on testing the field with a perimeter, I have found, on a more minute

FIG. 248.



Left hemianopia; sudden onset; no other symptoms. In the right eye the loss appears to extend to the fixing point, but the vertical line in the small figure was drawn by the patient as the limit of the blindness when the central spot was fixed.

examination, that the line passed a little to one side of the actual fixing point, and did not go through this. In the right field shown in Fig. 248, for instance, the loss, according to the perimeter, seemed to extend quite up to the fixing object, but when the patient fixed a small dot, the area of vision was found to extend beyond it, as shown in the small diagram. If so, the variations in different individuals will depend merely on differences in the area from which the central fibres pass to both tracts, and the apparent difference in kind resolves itself into one of degree.

Other differences in the dividing line have probably the same origin. In most instances it is somewhat irregular, and in some cases it is oblique above and below. The deviation may be toward one side above and the other side below, so that the general direction of the division is the same, and it may be quite similar in the two eyes.* Another difference in the dividing line is also probably due to the anatomical arrangement. The line is usually sharp; good vision suddenly changes to blindness. Sometimes, however, a narrow band of indistinct vision limits the blind half. This must be due to the passage of some fibres from the medial zone by the tract on the same side; they are not numerous enough to give the distinct vision of the macular region, but they are enough to maintain some vision in the middle band.† There is no relation between the existence of an amblyopic zone and the seat of the lesion.‡ All these variations, and also slight differences sometimes to be found between the two fields, must be due to individual differences in the character of the decussation. When the hemianopia is incomplete, the irregularity of outline must be referred to the extent of the lesion.

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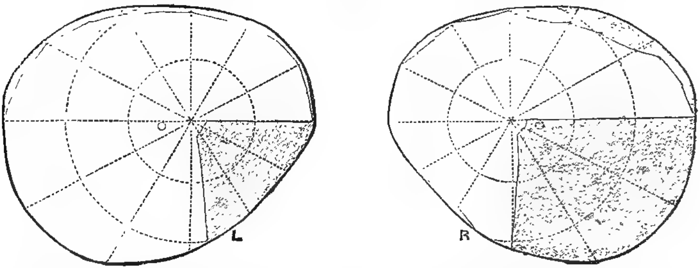
* It has been asserted that such passage has been proved by the fact that, in cases of axial neuritis with a central loss of sight, degenerated fibres have been traced into both optic tracts. But they might be so traced, were there a strict division through the macula; the central loss always extends farther from the macula than the area of preserved vision in hemianopia. The passage of fibres from each side of the macula into each tract could not possibly be followed.

Hemianopia is not always complete. Only part of a half field may be lost, but the same part of the field is defective in each eye. Fig. 249 shows a loss of the lower left quadrant in each eye. The lower quadrant seems to be more frequently lost than the upper.§ Sometimes the loss is thus partial from the first; more often, as in this case, the hemianopia is at first complete, and vision is regained in some part of the affected half, but remains absent in another part. The loss may extend up to the neighborhood of the fixing point, or, in slight cases, may be confined to the peripheral part of the field (Fig. 250). Such partial loss depends on the partial extent of the disease in the path or centre. It is very rare in disease of the tract, in which all the fibres lie very near together, but has been observed in

FIG. 249.



Partial left hemianopia, involving the lower quadrant. The fields were taken ten years after the onset. The hemianopia was at first complete, and was accompanied by left hemianæsthesia (including the special senses), slight hemiplegia, and optic neuritis. The lesion was probably a syphilitic gumma near the hinder part of the right thalamus.

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* A well-marked instance of this obliquity is shown in Fig. 15 of "Diagnosis of Diseases of the Brain."

† Hirschberg, *Nagel's Jahresber.*, *1876*, 79.

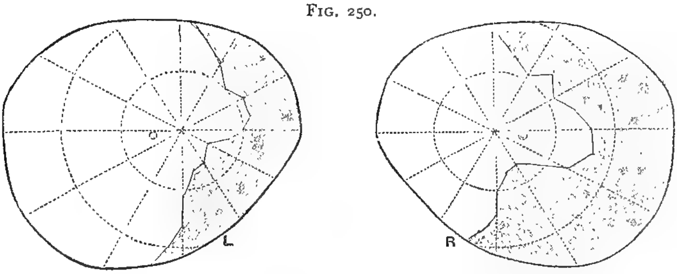
‡ See Wilbrand, "Ueber Hemianopsie," p. 160.

§ In the case here figured, and in another figured in "Diagnosis of Diseases of the Brain" in which a quadrant was defective, this was the lower one, and the hemianopia was left sided. Wilbrand's statistics give—left hemianopia, lower quadrant in twenty-one and upper in five cases; right hemianopia, lower quadrant three and upper five—a curious difference.

a case of partial softening (Marchand). In the case shown in Fig. 249 it probably resulted from disease of the optic thalamus or optic radiation near the internal capsule, which was also implicated. Partial hemianopia is often due to disease of the occipital lobe.

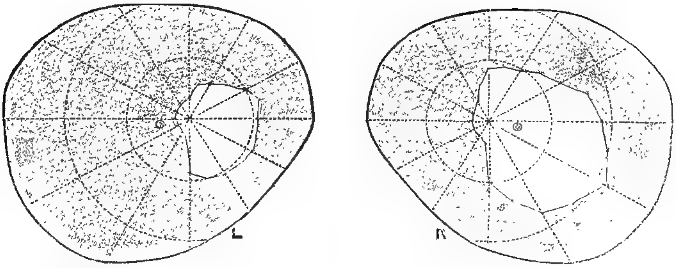
The acuity of central vision is often lowered in hemianopia, even when the fixation region is included in the remaining half field. Indeed, as Wilbrand has suggested, there should always be some central amblyopia; if fibres from the macular region pass by each optic tract, the loss of those which pass by one tract should lessen vision. Of ninety-three cases collected by him, central amblyopia was noted, however, only in fifty-three, and in some of these ophthalmoscopic changes were present. The cases which I have carefully examined show that some diminution of acuity can be detected, if a stringent test is used, even though a cursory examination may reveal no defect.* The number of fibres supplying the macular region is, however, so large that a loss of one half may have but a slight effect.

FIG. 250.



Right hemianopia, partial, involving only the periphery of the half field.

FIG. 251.



Left hemianopia, with a great reduction of remaining half fields, accompanied by left hemiplegia and recurring left-sided convulsions. The symptoms have now persisted for twelve years.

In many cases of hemianopia the unaffected half fields have their normal peripheral extent. In other cases there is some peripheral reduction, slight or considerable, and this when there is no peripheral lesion, such as optic neuritis, to which it can be ascribed. The reduction has hardly received the attention it deserves. The half field is usually smallest in the eye on the side toward which sight is lost, *i. e.*, opposite to the lesion, but as this is always the nasal half field, the peripheral reduction may not seem so great as in the larger temporal half field of the other eye. The reduction has been supposed to be due to the lesion being in the optic radiation near the thalamus (Wilbrand), but it may be absent even in disease of the occipital cortex.* Its probable significance will be considered presently.

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* Of course, the possibility that the acuity of vision may not have been normal before the onset of the hemianopia must be borne in mind.

Color Hemianopia.—In ordinary hemianopia the loss for colors corresponds to that for white. If, however, there is a limitation of the half fields for white, the color fields are also proportionally reduced in size. It is a remarkable fact that there may be hemianopia for colors when there is none for white—"hemiachromatopia." There is no change in the field for objects, but as soon as the vertical line is passed all colors appear gray. Several cases of this remarkable defect are on record,† and it might, perhaps, be found more frequently if the color fields were tested at the bedside in all cases of cerebral disease. The dividing line appears, generally, to pass through the fixation point. The symptom probably depends on disease of one part of the occipital lobe, and is proof of a separate centre for color vision, but the position of this centre is not yet known; Wilbrand suggests that it is in some part of the occipital cortex in front of the apical region, and that impressions pass through the latter to reach the color centre, since disease of the apex causes complete hemianopia.

With the two exceptions just mentioned—the limitation of the remaining half fields, and the isolated loss for colors—there are no known differences in the character of hemianopia that are due to differences in the seat of the disease in the optic path behind the chiasma.

Double lateral hemianopia, if complete, necessarily involves total loss of sight. It has been known to result from successive lesions in the two occipital lobes, the hemianopia left by the first attack changing to complete amaurosis when the second occurred.‡ A remarkable case of complete color blindness, with apoplectiform onset, is on record. The acuity and fields of vision were normal. Some color vision was regained, but there was persistent blindness to red and green.§

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* *E. g.*, case of Curschmann, *Cent. f. Augenh.*, *1879*, p. 181.

† Bjerrum, *Cent. f. Augenheilk.*, *1881*, p. 471; Samelsohn, *Cent. f. Med. Wiss.*, *1881*, Nos. 47 and 50; Eperon, *Arch. d'Ophth.*, *1884*, p. 356; Charpentier, "De la Vision, etc.," *Thèse de Paris*, *1877*; Swanzy, *Trans. Oph. Soc.*, vol. iii, p. 185. Swanzy's case is especially important, because there was a reduction of the upper part of the field for white, on the side on which color vision was lost, which suggests that the centre for colors is adjacent to that part of the half-vision centre in which the upper quadrant is represented. The remaining half fields for colors were much lessened, and it appears from the chart that there was a slight limitation in that for white.

‡ Cases recorded by Nothnagel ("Topische Diag. der Gehirnkr.," p. 389); Calmeil (*Mal. de Cerv.*, vol. ii, p. 411); N. Moore (*St. Barth. Hosp. Rep.*, vol. xv, 1879); Chvostek (*Oest. Zeitschrift* and *Virchow's Jahresb.*, *1872*, vol. ii, p. 49).

§ Steffen, *Arch. f. Ophth.*, xxvii, 1881.

Lateral hemianopia is attended by no resulting ophthalmoscopic changes in the early stage. Alterations in the fundus may coexist, in consequence either of the nature of the cerebral lesion (as papillitis in cases of tumor) or of associated morbid states (as albuminuric retinitis in cerebral hemorrhage). After some months or years, slight atrophic changes may be visible. Some have thought that pallor could be observed in the corresponding half of each disc. I have not, myself, been able to recognize this, but have seen the disc on the side on which the temporal half field was lost become slightly paler than the other. This is explained by the fact that the area of field lost is greater in this eye than in the other, and a correspondingly larger number of fibres have their function arrested.

Lateral hemianopia which has lasted for some weeks in complete degree seldom passes away altogether, and may remain permanent, with little diminution. Its persistence for twenty-three years has been observed by Seguin.* When a complete hemianopia lessens, it may do so from the medial region toward the periphery, so as to leave a symmetrical peripheral loss, as in Fig. 250, or else from above or below, so as to leave a quadrantic defect. Rarely, however, it clears from the periphery, so as to leave symmetrical scotomata, having one extremity in the central region.†

Hemianopia may remain for a long time unnoticed by the patient, or, if he detects a change in vision, he is apt to think that there is a loss of sight of one eye. The loss is especially apt to escape notice when a considerable area around the fixing point is spared, and central vision is thus but little affected. In one case, the loss was only discovered when it was noticed by the nurse that the patient, at dinner, never ate his potatoes, which were always placed on one side of the plate. Hemianopic patients, in circumstances which require a wide range of vision, often carry the head turned a little toward the blind side, so as to bring objects opposite them nearer the centre of the remaining portion of the field. The loss sometimes leads to accidents, especially in those who are unaware of their defect.

Lateral hemianopia is frequently associated with other symptoms of an organic lesion of the brain. Of these, hemiplegia, transient or permanent, is the most common, and is present in at least half the cases.‡ The loss of power is always on the side of the blindness, when the two are the result of a single lesion; the patient is unable to see toward the side that he is unable to move. Hemianæsthesia is also an occasional complication, and speech defects are sometimes met with in right-sided cases. The significance of these associations will be described in the chapter on Cerebral Localization.

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* *Journal of Nervous and Mental Diseases*, Jan., 1886.

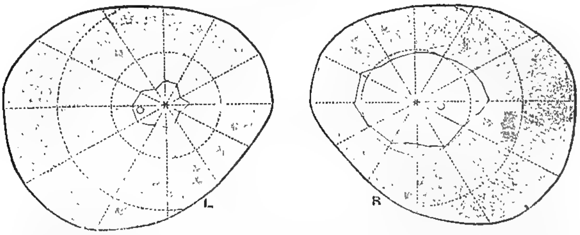
† See a case recorded by Lang and Fitzgerald, *Trans. Oph. Soc.*, vol. ii, p. 231, where references to other cases will be found.

‡ The proportion of published cases is one-third (59 of 154 cases collected by Wilbrand), but this certainly under-represents the proportion. The more thorough examination of cases now made reveals hemianopia in so many cases of hemiplegia that its presence does not lead to the publication of the case unless this is completed by an autopsy, and it is still probably undiscovered in many cases in which it exists.

As a transient symptom, present only during the early stage of the disease, hemianopia often occurs in severe lesions of one cerebral hemisphere, especially in cerebral hemorrhage. If the observer's finger is suddenly brought near the eye of the patient, first from one side and then from the other, it will be found that there is a reflex contraction of the orbicularis when the finger approaches from the sound side, and none when it is on the side of the hemiplegia. This seems to show a hemianopic defect, as part of the initial general interference with the functions of the affected hemisphere, although, when the patient has so far recovered as to permit an examination of the field, no defect can be discovered, and the difference in reflex action no longer exists.

Transient functional hemianopia is common as a part of the phenomena of migraine; and, like other manifestations of that disease, sometimes occurs as an isolated symptom, apart from headache. It is remarkable that hemianopia very seldom occurs as part of the functional disturbance of hysteria, in which another form of sight defect, presently to be mentioned, is so common. Cases of hysterical hemianopia are on record,* but of a large number of cases of hemianopia that have come under my observation, one only *may* have been of hysterical origin.

FIG. 252.



Concentric reduction of fields of vision, greatest in (with considerable amblyopia) the left eye, accompanied by left hemianæsthesia and slight hemiplegia; sudden onset. The patient was a woman sixty years of age.

Oblique or horizontal hemianopia is seldom met with, and needs further study. It has been mentioned that the dividing line in lateral hemianopia may have an inclination to one side above and to the opposite side below, apparently in consequence of an individual peculiarity in the decussation. It is conceivable that a similar deviation may sometimes give rise to a hemianopia which may be fairly termed oblique. Hemianopia in which the dividing line is horizontal, and an upper or lower half is blind, is very rare.† It may be simulated by a symmetrical lesion of the optic nerves, or may be a compound of double partial hemianopia, in which the two lower quadrants are lost. In the same way a loss of the lower quadrant on the one side, and the upper quadrant on the other side, may conceivably be produced (Wilbrand).

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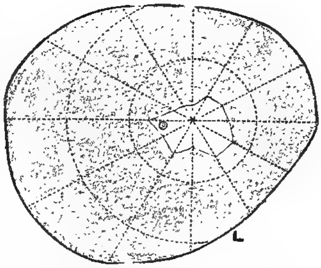
* Bonnefoy ("Troubl. de la Vision de l'hystérie," *Thèse de Paris, 1874*) records some cases observed at the Salpêtrière.

† A case of loss of the lower half of each field is recorded by Anderson, *Med. Times and Gazette, 1885*, No. 1842.

Importance has been attached to the difference between cases of hemianopia according as the blind half fields appear to the sufferer merely vacant or distinctly black, *i. e.*, whether there is or is not a sense of entire absence of light in them. But the significance of the difference has yet to be ascertained. In the case of temporal hemianopia, shown in Fig. 243, the patient, a medical practitioner, describes the blind halves as appearing, not black, but like a fog; nevertheless, he cannot even discern a light in the blind half.

A third form of sight defect from brain disease is what is termed *crossed amblyopia* (see p. 462). There is dimness of sight of the eye opposite to the cerebral lesion, and examination of the field shows this to be concentrically reduced in size, in varying degree in different cases. Sometimes it is reduced to a small area, extending only just beyond the blind spot, about 20° around the fixing point. The color fields are also reduced, and disappear altogether when the diminution for white is considerable. The patient does not complain of the sight of the other eye, but if it is tested there is found to be a reduction in this field also, similar, but much slighter in degree, and never involving entire loss of the color fields, even of that for red.

FIG. 253.



Concentric restriction of the field of the left eye (accompanied by amblyopia) in a woman aged fifty.[*]

This form of sight defect is similar to that which often occurs in hysteria in association with hemianæsthesia. Pathological evidence of the seat of morbid process is meagre, but is sufficient to make it highly probable that the symptom depends on disease of the cortex of the lower and hinder part of the parietal lobe, the supra-marginal and angular convolutions. In the few fatal cases with this loss the disease has involved this region (see p. 463); while the symptom is not caused by disease of the occipital lobe, or of the temporal lobe, or of the superior parietal lobule, or of the ascending parietal. The facts of disease make it probable, moreover, (1) that the eye of the same side must be represented, in slight degree, in this centre as well as the opposite eye in chief degree; (2) that partial disease lowers the function of the centre as a whole, and does not cause a partial loss of one part of the field; and (3) that the centre of the opposite hemisphere can supplement, to a considerable degree, that which is destroyed, since the affection of vision usually lessens after a time. Complete destruction of this centre does not cause complete loss of sight of the opposite eye; some vision and a small field remain, and these must be due to the representation of this eye in the other hemisphere, *i. e.*, in the hemisphere on the same side as the eye concerned. But disease of this region seldom is complete, because the blood supply to it is usually shared by both the middle cerebral and the posterior cerebral. Thus, each higher visual centre must be connected with both occipital lobes; the connection with that of the opposite hemisphere must be by the fibres of the corpus callosum.

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[*] The right eye had been removed many years before; the affection of vision was attended by left hemianæsthesia, dullness of all the other special senses on the left side, and severe headache. There was no symptom suggestive of hysteria. Syphilis was probable. The symptoms developed gradually, and increased until iodide of potassium was given, when they became arrested, and they had distinctly lessened when the patient ceased to attend the hospital.

In the theory here advanced to account for crossed amblyopia, we may find an explanation of the concentric restriction of the remaining half fields, often met with in hemianopia. If disease of the occipital lobe, causing half blindness, extends forward to the higher visual centre, in proportion to the damage to the latter, the remaining half fields will be concentrically reduced, while, if the higher centre is completely destroyed, the half field of the opposite eye will be reduced to very narrow dimensions, vision being subserved only by the opposite hemisphere. It is possible that a lesion of the white substance beneath this region may have a similar effect. We need more facts before any confident opinion can be expressed regarding the cause or causes of this concentric contraction, but the hypothesis here advanced is supported by the fact that the reduction of the half field is often much less in the eye on the side of the lesion than in the other eye, just as is the contraction of the whole field when the higher visual centre is alone diseased. In the cases of hemianopia that I have seen in which the fields were narrowed without peripheral disease of the nerve, and in which there were other localizing symptoms, these pointed to disease of the cortex. Thus, in the case shown in Fig. 251, hemiplegia accompanied the hemianopia, and was accompanied by convulsions in the paralyzed limbs, commencing locally, and recurring during many years, a sure sign of a cortical lesion. There are few recorded pathological observations bearing on the point, but those that exist confirm the view here expressed.*

Mind Blindness.—The last form of visual defect is the singular condition called "mind blindness," which has been already briefly mentioned. The term was given by Munk to a condition which he produced in dogs by extirpating parts of the occipital lobes. The animals, while apparently able to see, failed to recognize the nature of objects, such as food placed before them, or to know persons with whom they had previously been familiar. A similar condition has been observed in a few cases of brain disease. Without other mental defect there has seemed to be an entire loss of visual memory; familiar places and faces seemed strange and unfamiliar, and even the nature of familiar objects was not recognized.* The loss necessarily includes that of visual word symbols.

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* Thus, in a case of right hemianopia recorded by Forster (Graefe u. Saemisch's "Handbuch," Bd. vii, p. 118), in which the peripheral limitation was moderate in degree, the lesion involved the caudate and lenticular nuclei, and also the cortex in a small region in the anterior part of the occipital lobe and posterior part of the angular gyrus. In another case recorded by Hosch (*Klin. Monatsbl. f. Augenkr., 1878*, p. 281), in which the limitation was considerable, a lesion in the occipital lobe extended from the neighborhood of the internal capsule up to the gray substance. In this case, however, there were retinal changes. In a case recorded by Wernicke of right hemianopia, and peripheral limitation of the fields, the lesion of the occipital cortex extended to the posterior part of the angular gyrus ("Gehirnkr.," Bd. ii, p 190). On the other hand, in a case of disease of the white substance recorded by Westphal ("Charité-Annalen," Bd. vi) without contraction of the field the cortex appeared normal. A cortical lesion did not extend beyond the limits of the occipital lobe in cases without concentric limitation recorded by Curschmann (*Cent. f. Augenh., 1879*, p. 181).

The structures that subserve the functions lost in mind blindness are certainly separate from those of the half-vision centres, since hemianopia from disease of the cortex does not necessarily involve this special loss. The centres concerned are probably in front of the half-vision region, either in the anterior part of the occipital lobes or in the posterior part of the parietal lobes. The latter is more probable (see p. 463). The recognition of visual word symbols is subserved by structures in this region in the left hemisphere, and the loss of this function may, as we have seen, be regarded as partial mind blindness. We do not know whether complete mind blindness can be produced by a lesion in one hemisphere, or whether disease of both hemispheres is necessary for the production of the symptom. Its extreme rarity is in favor of a bilateral cause, and experiments on animals suggest the same conclusion. When the condition results from disease of one hemisphere (as in the case figured on p. 463) it is probably transient. If, however, there is a congenital defect or a previous lesion in the higher visual region of one hemisphere, disease limited to the other hemisphere might give rise to permanent mind blindness.

Although hemianopia does not involve mind blindness, the two symptoms have been associated. In one case described by Wilbrand, incomplete left hemianopia was associated with a slight defect in the lower parts of the right half fields suggestive of a lesion in each hemisphere. Another case is recorded by Bernheim, and is remarkable since considerable and persistent mind blindness (including word blindness) accompanied left hemianopia in a left-handed man.†

In connection with the interference with the higher visual functions of the brain, it may be noted that the area of the cortex, which is apparently related to these functions, is very extensive. In no part of the brain is the difference greater between the brain of the monkey and that of man than in the region between the extremity of the fissure of Sylvius and the occipital lobe. This region in man exceeds in size the whole brain of the monkey.

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* Wilbrand ("Die Seelenblindheit," 1887) has analyzed at great length the relations of this symptom to other mental processes.

† Bernheim, *Revue de Méd.*, *1885*, p. 625. This case is consistent with the suggestion given in the text that unilateral disease only causes persistent mind blindness when there is a defect in the opposite hemisphere, for left handedness is often the result of an early lesion or congenital defect of the left hemisphere.

Diagnosis.—Impairment of sight in one eye without any affection of the other, and with loss of action of the pupil, means disease of one optic nerve. A central scotoma generally means disease of the fibres of the nerve which, at the back of the orbit, occupy the middle of the trunk. Peripheral limitation of the field may be due to damage to the peripheral layer of the nerve; it may, probably, also be the result of a slight degree of damage to all the fibres of the nerve, which produces most impairment in the periphery of the retina, where vision is normally dull, and is extinguished first by a general reduction of function. A sectorial defect in one eye only means damage to the nerve, considerable in degree, but partial in extent. Amblyopia with concentric reduction of the field, considerable in one eye and slight in the other, may be due to atrophy or to disease of the higher visual centre in one hemisphere. In the first, the ophthalmoscope reveals the signs of atrophy, and the action of the pupil is lessened; in the second, the aspect of the nerve is normal, the pupil contracts perfectly under the influence of light, and the onset is either sudden or attended with other indications of an organic cerebral lesion. In functional (hysterical) amblyopia the symptoms are the same as in disease of the higher visual centre, and the diagnosis depends on other indications of organic or functional disease respectively. In the rare cases of functional disease in which sight is affected in one eye only, the perfect action of the pupil effectually distinguishes the condition from organic disease of the nerve. Total loss of sight of both eyes may be due to chronic atrophy, to damage to chiasma, to disease of both tracts or both hemispheres. In all cases the mode of development of the symptoms enables a diagnosis to be made; the symptoms are at first partial, and their character indicates clearly the position of their cause. It is unnecessary to describe in detail the significance of the various combinations of symptoms which sometimes occur in the progress from partial loss to total blindness, since the diagnosis involves only an application of the facts already given in the account of the symptoms.

Temporal hemianopia indicates disease of the chiasma. The combination of complete blindness in one eye, and a loss of the temporal half field in the other, generally means disease of the chiasma extending to the outer fibres, and often to the optic tract on the side on which the blindness is complete.

The diagnosis of the seat and nature of the cause of lateral hemianopia depends on the mode of onset and the associations of the symptom; it is a question of cerebral diagnosis, and will be considered in the chapter on Localization. So far as the characters of the hemianopia are concerned, the chief distinction is the concentric narrowing of the field, which points to disease of the cortex. A distinction has been sought between the disease of the tract and of the intra-cerebral path and centres, in the action of the pupil when light is reflected sideways into the eye, so that it falls some distance to one side of the macula. When the light is thrown on to the blind half of the retina the pupil contracts, as much as if the light is thrown on the seeing half, if the disease is in the hemisphere, but contracts much less if the disease is in the tract, because the path to the corpora quadrigemina is then interrupted.* The test is of value, although much care is required in its employment.

Prognosis.—The prognosis depends on the nature of the lesion causing the symptoms. As a general rule, it is not good. Considerable defect of sight, due to a lesion of the path or centres, seldom passes away entirely. Hemianopia is especially persistent, and often presents very slight improvement even when the lesion has been stationary for years.

Treatment.—The treatment depends on the nature of the morbid process causing the symptoms, and this is described in detail in the account of the several diseases.

FUNCTIONAL AMBLYOPIA AND AMAUROSIS.

Toxic Amaurosis.—Bilateral loss of sight, usually complete in degree and extent, but brief in duration, is an occasional result of certain toxic blood states. It is especially common as a consequence of uræmia. The loss of sight comes on suddenly; sometimes the patient wakes up blind. The pupils are commonly dilated; they may or may not act to light. Other uræmic symptoms, such as convulsions, usually accompany the amaurosis. Sight returns when the state of the blood is improved by treatment. There are no ophthalmoscopic changes related to the amaurosis, but albuminuric retinitis often coincides with it. A similar amaurosis has been met with in cases of lead poisoning. In poisoning by quinine, and in the acute anæmia that results from loss of blood, sight is sometimes lost, but changes in the retina are commonly met with, although it is not certain that these changes are the cause of the affection of vision. The variations in the action of the pupil in different cases of uræmic amaurosis seem to show that the nerve elements, on which the blood state acts, are not always the same. Nerve cells are more susceptible to arrest of function than nerve fibres, and, therefore, we must assume that when the action of the pupil is preserved, the poison acts on the cerebral visual centres, and that when the action of the pupil is lost, the retina itself is rendered insensitive, either alone or together with the cerebral centres.† If the amaurosis is sometimes of retinal origin, it must be due to a direct action on the nerve elements, because the absence of any change in the retinal vessels during the loss of sight is well established.

Migrainous Hemianopia has been already mentioned, and will be described more fully in the account of the disease.

Reflex Amblyopia.—Vision is sometimes impaired in one eye, or both, by irritation of other nerves, chiefly by that of the fifth. There is often a concentric contraction of the field of vision, but seldom complete loss of sight. Vision is most affected on the side of the fifth nerve irritated, and the interference is sometimes confined to this eye. The stimulus may be the pain of neuralgia or an irritation of the nerve by organic disease or injury; frequently it is due to a carious tooth. The tooth is generally a molar; although it has been fancied that a recognition of this pathological connection underlies the popular name of "eye teeth" for the canines. The branch irritated has been generally in the second or first division of the fifth nerve; sometimes it has been a branch to the eyeball itself, as when amblyopia accompanies the photophobia of corneal or conjunctival inflammation. The affection of sight generally subsides when the irritation of the nerve is removed. It does not depend on any visible change in the retina, although vascular dilatation has been seen in some cases. Its mechanism is uncertain, but the most probable explanation is that an inhibitory influence is exerted on the nerve cells of the retina or on the visual centres in the brain.* At the same time it must be remembered that a similar irritation has been known to set up an actual inflammation in the eyeball. Cases have been recorded in which amaurosis was supposed to be due to the irritation of intestinal worms, but most of these were observed in pre-ophthalmoscopic times, and more cannot be said than that this is a possible but certainly very rare cause.

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* Wilbrand, "Ueber Hemianopsie," Berlin, 1881, p. 89.

† The state of the pupils in these cases needs more study than it has yet received. In connection with the possible influence of the blood state on both the ocular and cerebral nerve cells, it would be interesting to know whether, when the action to light is lost, this ever returns before vision.

Hysterical Amblyopia.—The characters of the loss of sight that occurs in hysterical hemianæsthesia have been already mentioned, and will be again considered in connection with the other symptoms of this disease. The loss of sight is scarcely ever complete, and when it is considerable in the eye on the anæsthetic side it is usually distinct, in slighter degree, in the other eye. In rare cases, the loss does not conform to this type. Thus, I have met with absolute loss of sight in one eye, without any affection of the other, and a curious case of paroxysmal complete blindness, associated with abductor palsy of the larynx—both certainly "functional"—is mentioned in the chapter on Hysteria.

Amblyopia, such as is met with in hysteria, sometimes occurs as an isolated symptom. Although usually transient, it is sometimes of very long duration, and may, perhaps, be permanent—perpetuated by changes in nutrition, which increase to changes in structure. A middle-aged lady who had suffered from various symptoms of functional derangement of the nervous system, cardiac irregularity, vasomotor swelling of the hands, etc., complained of dimness of sight of the right eye. Acuity of vision was greatly reduced, and the field was contracted to a small area around the fixing point. The fundus and the action of the pupil were perfectly normal. At no time was there any other sensory loss, or any other symptom to suggest an organic cerebral lesion. The patient was seen occasionally during two years, and during that time the condition of vision and of the eye remained unchanged. Sometimes amaurosis, coming on under conditions suggestive of functional disturbance, goes on to optic nerve atrophy. A lady was always peculiarly sensitive to thunder-storms, although not specially alarmed at them; and she could always foretell a coming storm with remarkable accuracy. During one severe storm she became suddenly blind. There was no suspicion of a lightning stroke. Vision never returned. I only saw her years after the onset, and there was then simple but complete atrophy of the optic nerves. Such a case may remind us of Charcot's patient with hysterical paraplegia, who recovered, but relapsed under a mental shock, and the paraplegia became perpetuated as lateral sclerosis of the spinal cord, found after death.

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* Compare the case mentioned on p. 592, in which pain in one eye accompanied visual disturbance as the aura of fits due to a tumor of the opposite cerebral hemisphere.

It is probable that the seat of the morbid process in functional amblyopia and amaurosis is the cortex of the brain. The hypothesis of a higher visual centre in each hemisphere enables us to understand its seat more readily than does the theory that the half-vision region constitutes the highest visual centre. Crossed amblyopia, on the latter theory, involves the assumption that there is a combined partial disturbance of both hemispheres, while all the associated symptoms point to the disturbance of one hemisphere only. The nature of the change is a matter of speculation. Arrest of function in the nerve cells is indicated by the symptoms, and it seems more reasonable to regard this arrest as a primary inhibition, than, by assuming vasomotor spasm, to invoke a derangement of other nerve cells in the vasomotor centres. Such a theory only explains the condition by throwing the difficulty further back, into the obscurity of the sympathetic. The addition to this theory, favored in France, that the vasomotor spasm is the result of uterine irritation, need only be mentioned. The treatment of these functional conditions is that of the general nervous states of which they commonly form part.

Irritation Symptoms in the functions of the optic nerves are very rare in organic disease, although common in certain functional maladies, especially in migraine and epilepsy. In migraine the common form is the zigzag appearance, often colored, and known as the "fortification spectrum." Similar appearances are occasionally seen without other symptoms of migraine, both by those who are subject to headache and by others. One patient under my care was troubled with these appearances for years, as an isolated symptom. Often the zigzag was arranged in definite relation to some actual visual image, as, for instance, around a plate which was before him. At other times the spectrum was unilateral, and appeared to start from one edge of the field of vision. These phenomena are described more fully in the chapter on Migraine.

In epilepsy, visual impressions are very common as the aura of an attack, or as constituting a minor seizure. They are exceedingly varied in character, and are of every degree of elaboration, from a complex visual idea (such as an appearance of an old woman in a red cloak) to a simple flash of light. Stars, or a luminous ball coming nearer or receding, are other forms. Various colors are sometimes seen, especially red or blue; the most common color is a yellowish red, resembling that of fire. Simple colored vision, in which all objects appear colored, is scarcely ever met with. An apparent increase or reduction in the size of objects seen by the patient is a rare aura. For further details, the reader is referred to the chapter on Epilepsy.* I have known simple colored vision, red or blue, lasting for a few minutes, to occur as an isolated symptom in apparently healthy individuals. The cause of the colored vision occasionally met with in jaundice, and from the administration of santonin, is not known.

Visual hallucinations are common in delirium and insanity, and similar illusions occasionally occur as isolated symptoms in various states of nervous weakness. They are especially common in persons of unstable nervous organization, in whose families insanity exists. Such patients are often persistently annoyed by spectral visions of various kinds, grinning faces, horrible sights, and the like, when they close their eyes and try to sleep. Occasionally similar visions disturb such persons in their waking state. Thus, one young lady frequently had a distinct vision of a rat or a dog running across the room in which she was sitting. A curious relation has been noted, in a few cases, between such illusions and functional excitation of the optic nerves. We have just seen that a zigzag spectrum may be determined in its position by an actual sense impression; in the cases now under consideration, a distinct false vision has existed only when the eyes were open, and has disappeared when they were closed. It would seem that the nerve cells are only excited to morbid action by functional stimulation. In a curious case, recorded by Hammond,† a woman, during many months, frequently saw before her the image of a man and a woman whenever her eyes were opened, and could at any time produce the vision by hanging her head down. It disappeared when the eyes were closed, and when the right eye was closed one of the figures always disappeared, and the other when the left eye was closed.‡

As a result of organic brain disease, a visual aura has preceded convulsions in several cases of disease of the occipital lobe. In one case, of a tumor of the occipital cortex extending as far as the angular gyrus, the aura was a flash of light referred to the left eye, and an apparent diminution in the size of objects seen, and sometimes pain in the eyeball.§ In another case, with visual aura, recorded by Traube, the disease was a cyst in the white substance of the occipital lobe.||

Ocular Hyperæsthesia.—A simple increased sensitiveness of the optic

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* In the author's "Epilepsy and other Convulsive Diseases," a full analysis of these symptoms will be found.

† *Journal of Nervous and Mental Disease, 1885*, p. 467.

‡ A relation which we can understand on the theory that in the higher visual centre the opposite eye is represented, but not on the current hypothesis that the half-vision relation obtains in the higher as in the lower visual regions.

§ *Lancet, 1879*, vol. i, p. 363.

|| "Gesammt. Beiträge," Bd. ii, p. 1083. Another case is recorded by Westphal, "Charité Annalen," Bd. vi.

nerve, involving merely an ability to perceive a slighter amount of light or smaller objects than can be perceived by individuals with normal vision, scarcely occurs as an effect of disease. The power of seeing with the naked eye the satellite of Jupiter, may be relegated to the category of physiological curiosities. The term "hyperæsthesia" is commonly used in connection with sight to denote the condition in which distress or pain is caused by an amount of light which causes no inconvenience in health. Such intolerance is common in many ocular inflammations or irritation, especially in iritis and keratitis, and is then termed "photophobia." Apart from ocular disease it occurs in weak states of the nervous system, especially in women, and sometimes in association with symptoms of hysteria. In these cases it is termed "retinal hyperæsthesia," but it is doubtful whether the morbid state is retinal. The symptom was once thought to indicate inflammation of the retina, but in most of these cases the deeper parts of the eye are normal, and actual retinitis, strange to say, may cause no intolerance, nor does this ever result from simple neuritis. In health, an excessive amount of light is distressing, and in albinoes an excessive amount may act on the retina when that which falls on the eye is not more than can be borne by a normal individual. Optic hyperæsthesia is sometimes accompanied by lessened power of sight, or rather by extreme readiness of exhaustion, so that the field of vision quickly contracts under examination, and use of the eyes rapidly dims the sight. Intolerance of light is always associated with a tendency to protect the eye by closing the lids—"blepharospasm."

It is remarkable that the diseases that most readily produce photophobia are those of structures that are supplied by the fifth nerve. The reflex consequence, closure of the eyelids, is related to both the optic and the fifth nerves, guarding the eye from mechanical injury and from an excess of light. It is probable that there is a central relation between the centres for the two centripetal nerves concerned, so that increased action of the centres of the fifth alter the sensibility of those for vision. At the same time the possibility that light may influence the fifth nerve in the eye cannot be altogether excluded. A curious case has been recorded by Hutchinson, in which photophobia was produced by corneal inflammation in a blind eye.

The treatment of intolerance of light is, first, the removal of its cause, whether this is disease in the region of the fifth nerve or defective nutrition of the nervous system, and, secondly, the diminution of the surface sensitiveness of the eye, by the instillation of cocain, and by cold douches.

MOTOR NERVES OF THE EYEBALL.

(THIRD, FOURTH AND SIXTH NERVES.)

The motor nerves of the eyeball are the third, fourth, sixth, and sympathetic. The external muscles are supplied by the three former, the internal muscles by the third nerve and the sympathetic. It will be convenient to reserve for special description the affections of the internal muscles, and to consider first the general symptoms of paralysis of the external muscles, the special symptoms of disease of each nerve, and the causes and treatment of such disease.

When there is no muscular contraction, the eyeball is in the position of rest, and in this position, under normal circumstances, the line of vision is at right angles to the plane of the face. This is termed its "primary position." Any deviation from the primary position is due to muscular action. Many movements are produced by more than one muscle. Each of the straight muscles turns the eyeball in the direction indicated by its special name, but the superior and inferior recti, in consequence of their origin being nearer the middle line of the body than is their attachment to the eyeball, tend to turn the eyeball inward and to rotate it, so as to incline the vertical axis of the globe. The upper end of this vertical axis (which it is convenient to indicate by *S*) is inclined inward by the superior rectus, outward by the inferior rectus. This tendency to rotation is counteracted by the simultaneous contraction of the oblique muscles; the inferior, which rotates *S* outward, acts with the superior rectus; the superior oblique, which rotates *S* inward, acting with the inferior rectus. The oblique muscles also counteract the inward movement of the globe on its vertical axis, caused by the superior and inferior recti, and they also assist the recti in moving the eyeball upward and downward; having a slight elevating and depressing action (see diagrams, Figs. 254 and 255). When the eyeball is moved diagonally, the lateral rectus acquires a rotating influence, but this, in the outward diagonal movements, is counteracted by the opposite tendency of the associated superior or inferior rectus. In the case of slight

FIG. 254.

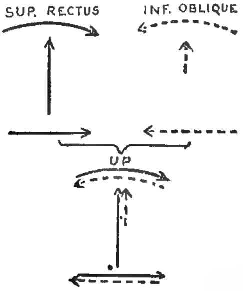

FIG. 255.

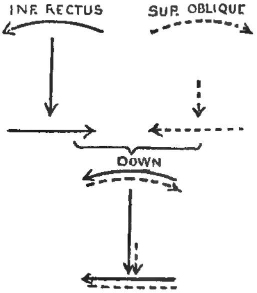

FIG 254.—Action of elevators of right eye: the arrow heads indicate the direction of movement produced by the muscles named. The curved lines represent the rotation on the antero-posterior axis, the vertical lines that on the transverse axis, and the horizontal lines that on the vertical axis of the eyeball.
FIG. 255.—Action of depressors of right eye. See last Fig. and description.

inward diagonal movements the rotating tendency of the superior and inferior recti corresponds with that of the lateral muscle concerned, and must therefore be counteracted by an oblique muscle.

The origin of the eyeball nerves has been already described (pp. 488, 489). The tract of gray matter beneath the aqueduct of Sylvius, which gives origin to the third nerves, is continuous behind with the nucleus of the fourth nerves, and both are connected with the apparently separate nucleus of the sixth by the posterior horizontal fibres. We have, as yet, no direct evidence of the precise distribution of function in the nucleus of the third nerve. The conclusion of Hensen and Voelcker,* from their experiments on dogs, has been stated at p. 489. A case investigated by Kahler and Pick † suggests that the arrangement of centres is not simply from before back, but that there is also a lateral difference of function. They suggest the accompanying modification of the plan of Hensen and Voelcker. The evidence is strong that the anterior part of the nucleus controls accommodation, and the next the action of the iris to light, and that these functions are subserved by the anterior fasciculi of the roots of the nerve.‡

Mid-Line.
Ciliary muscle.
Sphincter iridis.
Lev. palp.
Rect. Int.   Rect. sup.
Rect. infer.   Obliq. inf.
(Obliq. sup., 4th n.)

The relation between the internal rectus and the opposite external rectus, and the anatomical arrangement concerned in the lateral movement of the eyes, will be described in the account of their conjoint palsy.

GENERAL SYMPTOMS OF PARALYSIS.

Loss of power in the ocular muscles is indicated by five kinds of symptoms: limitation of movement, non-correspondence of visual axes (*i. e.*, strabismus), secondary deviation of the sound eye, erroneous projection of the field of vision, and diplopia. Strabismus and diplopia may result from spasm, but persistent spasm is rare, except as the common form of "squint," and in this diplopia is absent, for a reason that will be presently mentioned.

Limitation of Movement.—If a muscle is paralyzed, the eyeball cannot be moved so far as normal in the direction of action of that muscle. In complete palsy the defect is absolute; the eyeball cannot be moved beyond mid-position, unless slight movement by other muscles is possible. After a time the globe is fixed in the opposite position by contraction of the unopposed antagonist of the paralyzed muscle. Thus, in paralysis of the external rectus, the eyeball cannot be moved outward, and after a time is turned inward by the contracture of the internal rectus. If the paralysis is incomplete, movement is deficient in proportion to the amount of palsy, and toward the limit of movement the motion is often jerky ("paralytic nystagmus") from remissions in the contraction, analogous to tremor in a weak limb. When both eyes are open, and acting together, the extent of movement is usually less than when the other eye is closed, and the patient uses the affected eye only, and is compelled to strive to "fix" the object with it.

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* *Arch. f. Ophth.*, Bd. xxiv, 1878, p. 1. See also p. 489.

† *Prag. Zeitsch. f. Heilk.*, *1881*, p. 301, and *Cent. f. Augenh.*, *1883*. The chief evidence is that in two cases of nuclear disease and palsy of some muscles, there was a marked difference in the affection of the medial and lateral fibres of some fasciculi. The observed grouping of palsy in disease is seldom significant, because the process is usually degenerative, and this is often very irregular in its incidence.

‡ It is not probable that the position of these centres in man is as far forward beneath the floor of the third ventricle as it is in dogs, and as it has been assumed to be in man by some writers. The nuclei of the third nerves cannot be traced farther forward than the position of the posterior commissure.

Strabismus and Secondary Deviation.—In consequence of the defect in movement, the axes of the two eyes do not correspond in positions that necessitate the action of the paralyzed muscle. If a lateral rectus is affected, the axes converge or diverge, producing convergent or divergent "strabismus," which increases, the greater the degree of movement of the sound eye in the direction of action of the paralyzed muscle. Paralysis of the external rectus causes convergent strabismus, that of the internal rectus divergent strabismus. The deviation of the axis of the paralyzed eye from parallelism with that of the sound eye, is termed the "primary deviation." If the sound eye is prevented seeing the object, and the patient looks at this (is made to fix it) only with the affected eye, the sound eye is moved still farther in that direction, and hence the deviation of the visual axes is increased. This is called the "secondary deviation." Its existence and amount may be best ascertained by subsequently covering the paralyzed eye, and making the patient fix with the unaffected eye, which, to do so, moves back to its former position. The hand, or a piece of paper, may be so placed as to intercept the vision of the one eye, while leaving it exposed to observation. A piece of ground glass placed over the eye answers the same purpose. The occurrence of secondary deviation depends on the fact that normally two muscles which act together are equally innervated for a given movement. If one is weak, and an effort is made to contract it (as in fixing with that eye), the increased innervation influences also the other muscle, and causes an undue contraction. It is as if a rein acted equally on a hard-mouthed and a tender-mouthed horse, yoked together; the effort to make the former deviate would cause an excessive deviation of the latter. The secondary deviation is a delicate test for weakness of an ocular muscle, and may reveal its existence when the primary deviation is too slight to be observed. Moreover, these deviations afford a distinction from strabismus due to muscular spasm, such as ordinary squint. In the latter, the deviation exists in all movements; in paralytic strabismus only in those movements that call into action the paralyzed muscle. In ordinary strabismus the deviation is the same whether the patient fixes with one eye or the other; in paralytic strabismus fixation with the normal eye evokes the primary, and that with the affected eye the secondary deviation, and these two are similar in direction, but opposite in character, the primary being a defect, and the secondary an excess of movement.

Erroneous Projection.—An interesting and important effect of the increased innervation that causes the secondary deviation, is erroneous projection of the visual field. We judge of the relation of external objects to each other by the relation of their images on the retina; but we judge of their relation to our own body by the position of the eyeball as indicated to us by the innervation we give to the ocular muscles. When there is no muscular effort, and the eyes are at rest in mid-position, we know, to use popular language, that an object at which we are looking is opposite our face; that is, that a line from the centre of the field of vision to the macula lutea is perpendicular to the facial plane. If we turn the eyes toward one side, we know that an object in the middle of the field is to the side of its former position; how far to the side we estimate by the degree of movement of the eyes as indicated by the amount of innervation, and if we want to touch the object, the knowledge thus gained enables us to so contract the muscles of the arm as to effect the desired movement with precision. But the increased innervation needed by a weak ocular muscle gives the impression of a greater movement of the eye than has really taken place; and suggests that the objects seen are further on that side than they really are. If the patient attempts to touch an object, the finger goes beyond it. The erroneous projection exists, of course, only in those movements of the eyes, for which the weak muscle is necessary. It is greatest when the affected eye is used alone; it disappears altogether when this is closed. The erroneous projection is always in the direction of action of the affected muscle. The knowledge of the relation of external objects to the body, gained from the contraction (*i. e.*, innervation) of the eyeball muscles, is one of the most important sources of guidance to the centres that regulate the muscular contractions for maintaining the equilibrium of the body. The erroneous projection of the field destroys the harmony between the visual impressions and the others that are correct; the resulting discord and its consequences may affect consciousness as the sensation of "giddiness." This giddiness, depending on paralysis of an ocular muscle, is termed "ocular vertigo."

Double Vision.—We have seen that the field of vision of the paralyzed eye is erroneously projected. Each object in the field is referred to a position other than that which it actually occupies, and corresponding to the degree of ineffectual innervation. But, if the patient looks with both eyes, the field of the unaffected eye, being normally projected, does not correspond with the field of the affected eye; the images formed in the two eyes are mentally referred to different positions; objects are seen double. The patient is conscious of the doubling only of that object the image of which is formed at the macula lutea of one eye or the other; that is, of the object which is fixed by one eye, for which the eye is accommodated, and to which the attention is directed. The image formed in the unaffected eye, being referred to its correct position, is termed the "true image;" the other is termed the "false image." The distance between the two images depends on the amount of the error of projection, and corresponds to the degree of deviation of the eyeball from the position which it should occupy with that amount of innervation. In consequence of this deviation of the two visual axes, the retinal image is formed at the macula lutea only in one eye (the fixing eye). In the other eye it falls upon a part of the retina away from the macula, at a distance from it which corresponds with the degree of deviation, and, therefore, with the amount of erroneous projection. It was formerly thought that the circumstance that the two images are formed upon non-corresponding parts of the two retinæ affords an adequate explanation of the diplopia. But the diplopia which occurs in secondary contracture of a muscle cannot be explained by the simple theory of non-correspondence of retinal areas. A muscle, the right external rectus, for instance, is paralyzed, and when all the muscles are at rest, and each eye should be directed straight forward, the right eye is turned inward by the permanent shortening of the unopposed internal rectus. The image of an object opposite the eye is formed on the macula lutea of the left eye, but to the inner side of the macula lutea of the right. That this is no reason why there should be diplopia will be evident if we suppose for a moment that the patient has only the right eye, that this is normal and voluntarily turned to the left. It is clear that the object in the position supposed will be seen, not distinctly, because its image falls on an eccentric part of the retina, but still in its actual place. So, too, if we suppose the patient to have only the left eye, and that this is directed toward the object; this will also be seen in its normal place. Thus, there is no reason, in the mere deviation of the eyes, and the non-correspondence in position of the retinal images, why the object should be seen in this eye in an erroneous position—why there should be double vision. The explanation is, that in the case supposed—of a normal eye being turned to the left—the field is mentally projected to the left, in correspondence with the degree of active muscular innervation; the centre of the field lies thus to the left, and the object straight in front is referred to its proper eccentric position in the field. But in the case of the contractured eye, all the muscles are at rest; the field is referred to the position normally corresponding to rest, the centre of the field is projected directly in front, the eccentric image of the object is referred to an eccentric position

FIG. 256.

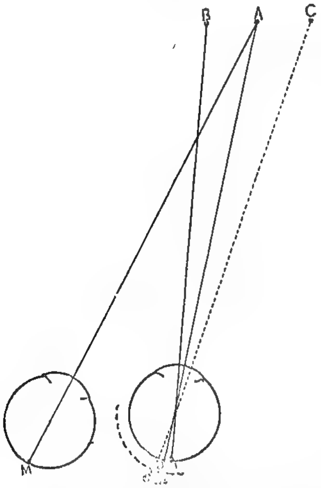

Diagram to illustrate the production of diplopia by the erroneous projection. In consequence of weakness of the right external rectus, the image of A formed at the macula, M, of the left eye, falls within the macula of the right eye at **a**. But the effort corresponds to that necessary to bring the macula to *m* (in the broken arc). Hence B, whose image is at **m**, seems to occupy the position of A, and the image formed at **a** to the left of **m** is projected to the other side of A, and seems to occupy the position C as if it were formed at *c* to the left of *m*.

in the field, and the patient sees the object in a false position with this eye, in the real one with the other. Thus, the non-correspondence of the retinal images does not explain the diplopia, except with the help of the erroneous projection. The latter does explain it without the former. Nevertheless, if there is no contracture, and the paralyzed eye when at rest is in mid-position, as in recent paralysis, the facts of diplopia may be explained, and its laws formulated, on either hypothesis.

Diplopia exists equally whether the patient fixes with the normal or with the paralyzed eye, but in the former case the true, in the latter the false object is the more distinct, that image being always the more distinct which is formed at the macula. The distance between the double images is greater when fixation is by the paralyzed eye, because the greater muscular effort results in a wider error of projection; of this greater effort the secondary deviation of the non-paralyzed eye is evidence. As Graefe has pointed out, if the patient moves his finger from the fixing (paralyzed) eye to the apparent image, in trying to touch it, the line taken by the finger corresponds in direction with the visual line of the other (secondary deviating) eye; each represents the excessive amount of muscular innervation, and the degree of excess is a measure of the erroneous projection of the field of the paralyzed eye.

In recent paralyses, diplopia exists only when the eyeball is in a position that needs the action of the paralyzed muscle. In old cases, with secondary contracture and permanent deviation, diplopia may occur during rest, or even during other movements. The distance between the double images increases the farther the object is moved in the line of action of the affected muscle. As a rule, it is the false image which appears to move from the other, and the direction of its movement is that of the action of the muscle.

The false image may be on the same side of the other as the eye by which it is seen (*homonymous or simple diplopia*), or it may be on the other side (*crossed diplopia*). When the muscle which is paralyzed is an abductor, and the eyes therefore converge, the diplopia is simple; when the muscle is an adductor, and the eyes diverge in paralysis, the diplopia is crossed. This depends on the fact that, in the former case (of convergent strabismus from loss of abduction), the false image is formed on the side of the retina toward the sound eye, and is therefore projected on the other side of the real object. In the case of divergent strabismus from paralysis of an adductor, the false image is formed on the side of the retina farthest from the sound eye, and is therefore projected on the side of the real object toward the sound eye. The student may remember the relation by the rule that when the visual lines (prolonged ocular axes) cross, the diplopia is *not* crossed.

In diagonal positions of the eyeballs the two images are not parallel; the false one appears inclined. On the theory that the diplopia is due to the non-correspondence of the parts of the retinæ on which the images are formed, the obliquity is explained by the fact that an abnormal rotation occurs in these diagonal positions. This rotation is due to the action of the oblique muscle, which should prevent rotation by opposing this tendency of the paralyzed muscle. The obliquity is explicable, also, on the theory that the diplopia is due to erroneous projection; as v. Graefe has shown, the same principles apply to an abnormal rotation as to a defect in lateral movement.

The total field of vision which is brought into view by various movements of the eyes, the head being still, may be represented as a circle, the centre of which is at the fixing point, when the eyes are at rest, while the radii represent the various movements, horizontal, vertical, and diagonal. The circle is termed the "motor field." In paralysis of one muscle, without secondary contracture, there is diplopia only in one part of this field, that corresponding to the movement of the muscle. Thus, in paralysis of the right external rectus there is diplopia in the right half of the field. The line which separates the area of single or double vision varies in direction with each muscle, and corresponds with the position of the axis of the eyeball around which the globe moves when the muscle is in action; but the correspondence is seldom exact, on account of the coöperation of other muscles in certain positions. To lessen the double vision, the patient often holds his head in such a position as to call the paralyzed muscle into action as little as possible. The movement of the head is on an axis which corresponds to the line of separation of single from double vision, and thus to the axis on which the eyeball is moved by the affected muscle.

Patients are not always conscious of double vision. If the muscular weakness is slight, the two images may nearly correspond, and the result may be an indistinct outline to the apparently single image. Again, if the images are far apart the less distinct eccentric image may be neglected, and the patient may be conscious only of that which is seen by the normal eye. This is often the case in old palsies. The detection of diplopia is facilitated by placing a red glass before one eye, preferably the sound eye, because the more distinct image seen with this eye is thus rendered less preponderant. A narrow strip of paper is then held up in various parts of the field; the one image is seen red, the other white. The two are thus more easily distinguished, and their relative positions can be ascertained. If the red glass alone does not at first suffice, the view from each eye may be alternately obstructed, and if there is double vision the position of the object will seem to alter as it changes from red to white.

In employing diplopia in diagnosis we must first prove that it is binocular and not monocular, by ascertaining that it ceases when one eye is closed.* We then learn by the colored glass whether the double vision is simple or crossed, whether the two images are on the same level or not, and the direction in which movement of the object increases the distance between the images. When the images are side by side and the diplopia is simple, the paralyzed muscle is of the eye on the side toward which movement increases the distance between the images. If the diplopia is crossed, it is a muscle of the other eye. If the images are one above the other, the paralyzed eye is that of which the image ascends from the other in looking upward, descends in looking down. If there is secondary contracture of the antagonist of the paralyzed muscle, diplopia exists through the whole range of movement in the plane of action of the muscles, *i. e.*, during the action not only of the paralyzed muscle, but also of its antagonist; but the distance between the images during movement remains the same on the side of the contractured antagonist, while it alters on the side of the paralyzed muscle. It must be remembered that the vertical action of the superior and inferior recti is greatest in abduction of the eye, and that during adduction the oblique muscles also elevate and depress. Vertical movements are, therefore, in the line of action of two sets of muscles, and to discriminate between them we must move the object up and down both during adduction and abduction. Vertical diplopia in abduction is due to the superior or inferior rectus; in adduction only is due to paralysis of an oblique muscle.

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* The cause of monocular diplopia is still unknown. It is sometimes well marked when there is no discoverable abnormality in the refraction or in the fundus of the eye.

Aid in diagnosis is also to be obtained by prisms, on the principle that a prism so placed that its base corresponds with the direction of action of the paralyzed muscle increases the distance between the double images; one in the opposite position blends them. They may also be blended by a prism placed before the unaffected eye in the position in which, before the affected eye, it increases the diplopia. Fusion by a prism before the affected eye removes the erroneous projection; fusion by a prism before the normal eye brings the projection of this field into the same error as the other.

PARALYSIS OF INDIVIDUAL MUSCLES.

We may now consider the symptoms of paralysis of each muscle. It must be remembered that more than one muscle is often affected, and the symptoms are correspondingly combined; and that, in diseases of the central nervous system, palsy and spasm are sometimes so associated as to render an exact diagnosis difficult, and occasionally impossible. This difficulty is often due to the circumstance that a given palsy was preceded by some other derangement of movement, as another slight paralysis and secondary contracture.

The paralyses described and figured are of the muscles of the right eye, except that of the external rectus, which is of the left eye, for comparison with the paralysis of the opposite internal rectus.*

External Rectus (Fig. 257).—The defect of movement is outward, with convergent strabismus; secondary deviation is by the opposite internal rectus. The diplopia is simple, in the left half of the motor field; the two images are on the same level in the horizontal movement, and the distance between them increases as the object is moved to the left. In diagonal positions the false image is inclined, the images diverging from the horizontal plane. Toward the limit of movement there are often slight spasmodic rotatory movements by the unopposed oblique muscle. The head is inclined to the left.

Internal Rectus (Fig. 258).—The defect of movement is inward; the secondary deviation is by the opposite external rectus; the strabismus is divergent. Diplopia is crossed, in the right half of the motor field. In diagonal movements the images diverge toward the horizontal plane. The head is inclined toward the right.

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*The diagrams of double vision are after those of Woinow ("Das Verh. der Doppelbilder," Wien, 1870).

FIG. 257.

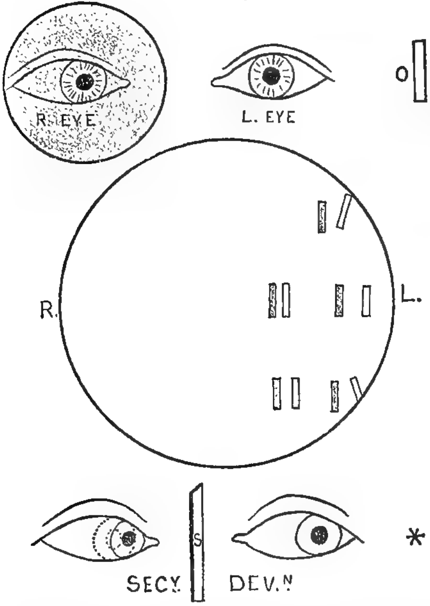


FIG. 258.

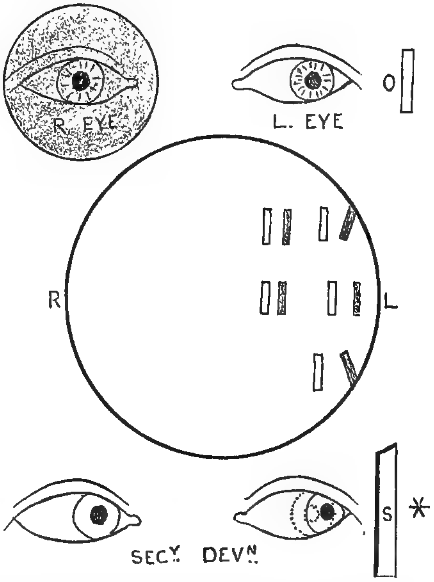


FIG. 257.—Paralysis of the left external rectus; colored glass over right eye; primary deviation on looking toward an object (o) on the left; position of double images; secondary deviation of the right eye when the screen, *s*, obstructing the fixation of * by this eye, compels fixation by the weak muscle. When the screens are removed, the right eye, in fixing, moves back to the position of the dotted outline of the cornea. (In the diagram of diplopia the highest white (false) image should have been represented a little lower than the true image.)

FIG. 258.—Paralysis of right internal rectus; primary deviation; double vision (red glass over right eye); secondary deviation. (Compare Fig. 257 and description.)

FIG. 259.

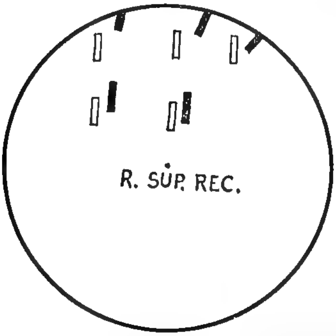


FIG. 260.

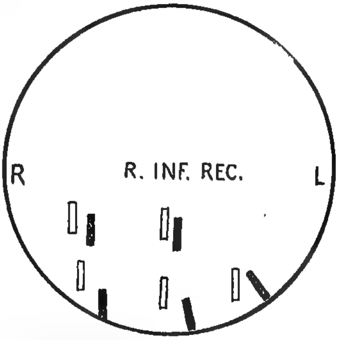


FIG. 259.—Diplopia in paralysis of right superior rectus. (The black image is that of affected eye.)

FIG. 260.—Diplopia in paralysis of the right inferior rectus. (Right image black.)

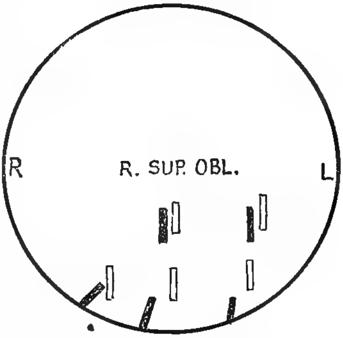
Superior Rectus (Fig. 259).—Movement is defective upward, especially upward and outward, and in attempting the movement the eyeball is rotated, the upper end of the vertical meridian outward, by the inferior oblique.

Secondary deviation is by the opposite superior rectus. The diplopia is crossed and exists in the upper half of the motor field; the images are one above the other, the upper image being the false one and receding from the other as the object is moved upward. The false image is inclined. The difference in height is greatest in abduction, while the inclination is greatest in adduction. These differences are due to the influence of the inferior oblique, which rotates as well as elevates. The head is held backward, and inclined toward the sound side.

Inferior Rectus (Fig. 260).—Movement is defective downward, especially downward and outward, because in the downward and inward movement the superior oblique is able to supplement the paralyzed rectus. The upper end of the vertical meridian is inclined inward by the oblique, from the loss of the rotating action of the inferior rectus. Diplopia exists in the lower half of the field, especially in the outer portion; it is crossed, the images being near together laterally, but the false image is below the other, and recedes with the downward movement. The two images (if the object is held vertically) diverge from the horizontal plane. The difference of height is greatest in abduction; that of obliquity in adduction. Secondary deviation is produced by the opposite inferior rectus. The head is inclined downward and toward the affected side. In an attempt to look down, the upper lid does not descend, but remains raised in correspondence with the position of the eye (see Fig. 262).

Superior Oblique (Fig. 261).—Defect of movement, chiefly downward and inward, since in this position the muscle is a depressor. In moving from the downward to the inward position, or *vice versâ*, the eye moves straight from one point to the other, instead of in a curve. Strabismus exists only below the horizontal plane, and is convergent. Secondary deviation is by the opposite superior oblique, turning its eye downward and inward. Diplopia exists in the inner lower part of the motor field; it is simple; the false image is the lower, and the images diverge toward the horizontal plane. The difference in height is greatest in adduction, the obliquity of the false image is greatest in abduction. The lateral distance is greatest in the middle line, and lessens when the object is moved either inward or outward. The head is inclined forward, and toward the sound side. The diplopia is chiefly noticed when the patient has to look down, as in descending stairs, which appear double. The detection of this palsy is difficult, since the oblique takes so small a share in moving the eye downward, that no absolute defect of movement results from the paralysis. The diplopia is the most important indication, and if the other eye cannot be moved downward, the detection of the palsy may be impossible.

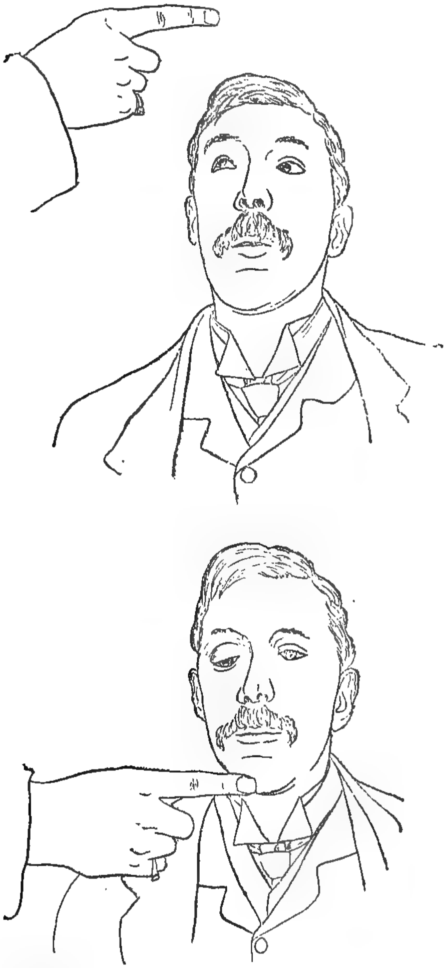
Fig. 261.



Diplopia in paralysis of right superior oblique. (Right image black.)

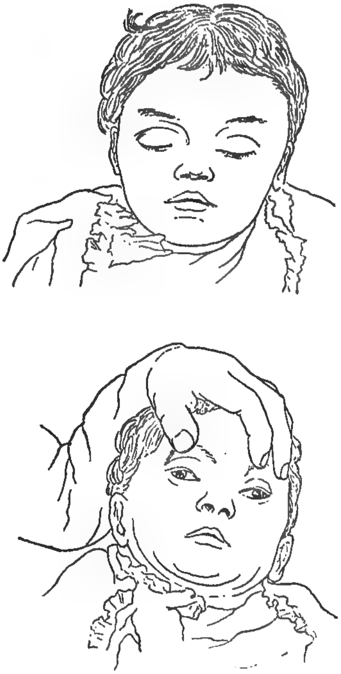
Inferior Oblique.—Defect of movement inward and upward, the moving in a straight line instead of in a curve, from the upward to the inward positions, and *vice versâ.* The secondary deviation is inward and upward by the inferior oblique and internal rectus of the sound eye. Diplopia exists in the upper inner half of the field; it is simple, the false image above the other, especially in adduction, and oblique (the images converging toward the horizontal plane), especially in abduction. The head is inclined backward, and toward the affected side.

Fig. 262.



Partial paralysis of left third nerve. Defective movement upward, from weakness of the superior rectus, and downward, from weakness of inferior rectus.

Fig. 263.



Paralysis of both third nerves in a child two years old. Complete double ptosis, and divergence of both eyes, by the external recti, when the eyelids are raised. The child was of a tubercular family, and presented also double optic neuritis and weakness of the limbs of both sides. No other cranial nerves were affected. The probable cause of the symptoms was a tubercular tumor in the interpeduncular space. Under tonic treatment all the symptoms passed away, and a year later the child appeared quite well.

Of isolated palsies, that of the external rectus is incomparably the most frequent, and the order or frequency of 145 cases was found by A. Graefe to be: external rectus 105, superior oblique 52, inferior rectus 10, superior rectus 9, internal rectus 5, inferior oblique 2. The frequency of isolated palsies of the first two muscles is due to their separate innervation.

AFFECTIONS OF SPECIAL NERVES.

Sixth Nerve, Abducens Oculi.—Only one muscle, the external rectus, being supplied by the sixth nerve, the disease of the nerve causes only paralysis of this muscle. As the figures given above show, it is the most frequent ocular palsy.

*Third Nerve.**—When the whole of the third nerve is affected, all the external muscles of the eye are paralyzed, with the exception of the superior oblique and external rectus; the levator palpebræ, sphincter iridis, and ciliary muscle are also affected. The upper eyelid droops and cannot be raised; the eye can be moved only outward, and a little downward and inward, by the superior oblique. The pupil is of medium size, and does not contract on exposure to light; power of accommodation is lost. In an attempt to raise the eyelid the frontal muscle contracts strongly (see next page).

After a time the deviation outward of the eye increases, in consequence of contracture in the external rectus, and the pupil becomes larger than it was at first, by a similar contracture in the radiating fibres. Paralysis of the third nerve is often partial; the various muscles supplied by it are affected in different degrees, and some may escape altogether. The muscles to the eyeball may be involved, and the levator may escape, but the paralysis of the former is then seldom complete. The levator and superior rectus are often involved together and escape together, but this rule is not invariable. The external muscles may suffer, and those within the globe may be unaffected; the converse relation is only seen in cases of nuclear disease.

Both third nerves may suffer, but it is rare for both to be paralyzed completely, as in the case shown in Fig. 263. There is then complete double ptosis, and if the lids are raised the eyeballs are seen to turn outward, and to be almost immovable. In most cases of bilateral disease the paralysis is partial. In some cases the distribution of the palsy is determined by functional association; thus, each internal rectus may be affected, and no other muscles; this is paralysis of an associated movement, and these cases will be separately considered.

Ptosis.—The drooping of the eyelid may be complete, so that it cannot be raised, or it may be incomplete, and can be raised to a certain point, but not higher. There is usually a conspicuous over-action in the corresponding half of the frontalis, which normally acts with the levator when the eyes are directed much above the horizontal plane. This is an instance of secondary over-action in an associated muscle, analogous to the secondary deviation in an ocular muscle. In some persons the frontalis can produce a slight elevation of the lid, in others it has no real influence. In order to ascertain whether a slight voluntary elevation of the lid is due to the levator or frontalis, it is necessary to fix the eyebrow by pressing it against the bone.

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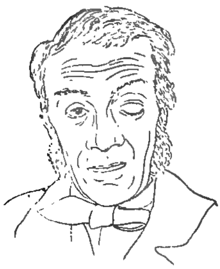
* The third nerve is commonly termed in Germany the "oculo-motorius," a name which has the grave practical inconvenience of preventing the use of the term "oculo-motor" in its exact and much-needed general significance.

FIG. 264.



Paralysis of the left third nerve; ptosis; over-action of frontalis.

Fourth Nerve.—The symptom of paralysis of the fourth nerve is paralysis of the superior oblique, which has been already described.

Sympathetic.—Disease of the sympathetic causes paralysis of the radiating fibres of the iris, with consequent loss of the dilatation of the pupil. There are also slight prominence of the eye and slight ptosis, supposed to be due to atony of the involuntary fibres of Müller in the fascia of the orbit.

INTERNAL OCULAR PALSY.

Ciliary Muscle: Cycloplegia; Loss of Accommodation.—The evidence of this, in normal and hypermetropic eyes, is that near vision fails and distant vision remains good, while near vision can be restored by a convex glass. Usually, though not invariably, the contraction of the iris, which normally occurs on accommodation, is absent also. In myopia and presbyopia the absence of the power of accommodation makes little difference to sight, and the diagnosis of the condition is difficult. Accommodation depends on the third nerve, and is lost in complete, and often in partial, damage to the trunk of the nerve, and in disease of the anterior of its fibres of origin, or of the anterior part of the nucleus beneath the posterior commissure. The path from the third nerve is through the ciliary ganglion and ciliary nerves. Loss of accommodation in one eye only, without the affection of other branches of the third nerve, is possibly due to disease of the ciliary ganglion or of the nervous ganglia within the eye, but we have no evidence at present of the effects of disease of these structures. Bilateral cycloplegia, occurring alone, usually depends on disease of the centres in the nuclei of the third nerves. It occurs in diphtheritic paralysis, of which it is one of the earliest and most constant symptoms. It occurs also as an associated symptom in degenerative disease of the spinal cord, especially in tabes. Its relation to such diseases is similar to that of the loss of the reflex action of the iris, immediately to be described.

Iris: Iridoplegia.—The iris has three actions, two reflex and one associated: (1) Reflex contraction of the sphincter on exposure of the eye to light. (2) Reflex dilatation by the radiating fibres on stimulation of some cutaneous nerve. (3) Contraction on accommodation, usually, but not necessarily, associated with convergence.

(1) *Loss of the Associated Action; Accommodative Iridoplegia.*—The pupil

does not lessen in size when an effort to accommodate is made. To examine this action it is only necessary to make the patient look first at a distant and then at a near object; the two should be in the same line of vision, so as to avoid any change in the amount of light that enters the eye. There is usually paralysis of accommodation, but it is said that the ciliary muscle may be competent, and yet the associated action of the iris may be lost, and *vice versâ.* This loss is less common than is that of reflex action; it is due to the same causes as cycloplegia.

(2) *Loss of the Light Reflex: Reflex Iridoplegia.*—In examining the action of the iris to light, care must be taken to test each eye separately, keeping the other eye covered. Light entering one eye acts on both pupils, and the contraction of each is the result of the total amount of light that enters both eyes. If one eye be covered and the action of the other pupil noted, it will be found to contract still more when the first eye is uncovered. A good light is important; a slight action, distinct on a bright day, is often imperceptible in a dull light. If artificial light is used it is best to make the patient look at an object in a distant dark part of the room, and then bring a light suddenly in front of the eye. If the patient looks at a near light, he will accommodate for it, and the pupil may then contract when there is no action to light. Hence, if the light is looked at, it must be at least four feet from the patient, and should be bright. To avoid the accommodation it is desirable, if a light is brought in front of the eye, that the other eye should be shielded from the light but not closed, so that, by continuing to fix a distant object, accommodation may be kept relaxed. Reflection of light into the eye by an ophthalmoscopic mirror is not a good test. The practical difficulty of saying whether there is or is not a slight action is often very great.

The path through which this reflex action is produced is the optic nerve, both optic tracts, probably the corpora quadrigemina, the anterior part of the nucleus of the third nerve behind the centre for accommodation, probably the second fasciculus of origin of the third nerve, its trunk, the ciliary ganglion and the ciliary nerves.

(3) *Loss of the Skin Reflex.*—The dilatation on cutaneous stimulation occurs in most persons under normal circumstances. The skin of the neck is a convenient place for the stimulation. The faradaic current, applied with a wire brush, so as to produce a sharp, painful sensation, has been usually employed, but in most persons the prick of a needle or pin, or a quill point, and even a pinch, is sufficient.* The motor path for this action lies in the cervical sympathetic, and the fibres connecting this with the cord at the lowest part of the cervical region. The centre on which it depends is said to be beneath the corpora quadrigemina, to the outer side of that for the light reflex. If so, both motor and sensory paths must traverse the cervical region of the spinal cord.

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* In some persons in whom the reflex action is readily produced it occurs on slighter cutaneous impressions. It may occur, for instance, when the palm of the hand is tickled, as schoolboys know who pretend to dilate the pupil by an act of the will.

Each reaction is lost when disease interrupts its path or damages the centre on which it depends. Thus, the light reflex is impaired or lost in disease of either the optic nerve (including the retina) or in disease of the trunk of the third nerve. Disease of one optic tract does not lessen the action, unless the light falls only on the blind half of the retina (see p. 588), no doubt because the fibres from the most sensitive (central) region of each retina pass by both optic tracts, and so disease of one does not abolish the reflex. So, too, the skin reflex is lost in disease of the cervical sympathetic, and in some affections of the cervical spinal cord, especially in those that impair sensibility. In any case we infer the seat of the disease from the other symptoms that are associated with the loss.

When the loss of reflex action occurs without such accompanying symptoms, and is thus isolated, so far as immediate associations are concerned, it is generally due to disease of the centres, degenerative in character.* It occurs in association with disease of the spinal cord, chiefly with locomotor ataxy, in which it is a common and early symptom. The loss is frequent also in general paralysis of the insane, and is occasionally met with in other degenerative diseases of less definite type. It may occur also without other nerve symptoms. In most of the cases I have seen in which it existed alone, the patients had had constitutional syphilis many years before, and the same statement is true of at least one disease (locomotor ataxy) with which the symptom is often associated.

The two paralyses are often, but not always, conjoined. There may be loss of reflex action to light, when the pupil still dilates on stimulation of the skin. The pupils are often small, reduced to two, one and a half, or one millimetre in diameter. The association of these small pupils with spinal disease was noticed before the more significant loss of the light reflex was discovered, and the condition was called "spinal myosis." But the pupils are not always small; they may be three, four, or five millimetres in diameter, although there is no trace of reaction to light. I think that it will generally be found that when the pupils are not small the skin reflex persists, and when they are small it is lost. The small pupils may be the result of the loss of tone in the radiating fibres, accompanying the loss of the skin reflex. In cases of disease of the third nerve, the ciliary muscle is paralyzed as well as the iris. When the reflex loss is due to central degeneration, the ciliary muscle is usually unaffected, and the pupils contract during accommodation. In some cases, however, there is also cycloplegia. All the internal muscles of the eyeball are then paralyzed, a condition to which we shall return.

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* This has not yet been demonstrated, but hardly admits of doubt, because the loss of reflex action occurs under the same conditions as another affection to be presently described—progressive paralysis of the external muscles—the central and degenerative nature of which has been proved.

The frequency of loss of the light reflex without loss of the associated action of the iris was first pointed out by Argyll-Robertson. The fact that loss of reflex dilatation is usually associated with that of reflex contraction was discovered by Erb.

ASSOCIATED OCULAR PALSIES.

Loss of Convergence and of Accommodation.—The power of accommodation may be lost, although the eyes can still be converged, but in many cases the two actions, habitually associated, are lost together. The internal recti contract in a normal manner in lateral movements of the eyes, but the two internal recti cannot be made to contract together. Doubtless there are separate centres for the converging action of the internal recti, as we shall presently see there are for the action of one with the opposite external recti in lateral movements. The former must be closely connected with the accommodation centre, since we only converge when we accommodate. A remarkable example of this loss in a child has been published by Eales.* There was absolute loss of convergence, of accommodation, and of the associated action of the iris, while all other movements of the eyes, and the reflex action of the pupil, were perfect. The loss developed gradually without recognizable cause, and, after lasting for about a year, passed away.

Paralysis of the upward movement of the eyes has been observed in cases of central disease. It may result from disease of the posterior part of the third nerve nuclei, and is then associated with paralysis of the levators (Kahler and Pick). When due to a focal lesion it is usually unilateral. It is possible that there is also a higher centre, disease of which may paralyze the upward movement without the levator, since the isolated symptom may be met with. I have recorded one such case in which the symptom was well marked.†

Paralysis of both upward and downward movements of the eyes, without impairment of the lateral movements, has also been observed, and is probably due to a lesion in the posterior part of the third nerve nuclei. The paralysis has been bilateral and accompanied with ptosis. But it has also been caused by disease of the cerebral hemisphere, situated in the corpus striatum and optic thalamus.‡

Paralysis of Lateral Movement: Conjugate Deviation of the Eyes.—The conjugate deviation of the eyes toward the side of a lesion in the cerebral hemisphere, the result of a loss of the power of moving them to the other side, has been already described (p. 512). The deviation in cerebral disease may also be due to spasm, and is then from the side of the lesion. We infer to which of these two mechanisms it is due, by the associated symptoms in the limbs.

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* *Trans. Ophth. Soc.*, iv, 1884, p. 300, and oral communication.

† "Medical Ophthalmoscopy," 2d ed., 1887, Case 52, p. 340. The patient has died since the account of her case was published. A very small tumor was found in the middle line behind the posterior quadrigeminal bodies, damaging these slightly, the velum, and the adjacent part of the inferior vermiform process of the cerebellum.

It must be remembered that disease of the nerves or their roots may chance to affect only the fibres for the superior recti. This was apparently the case in a patient with interpeduncular syphiloma (Thomsen, *Berlin. Gesellsch. f. Psych.*, June 7th, 1886). One superior rectus was more affected than the other, a character that is probably of diagnostic importance.

‡ Wernicke, *Berlin. kl. Wochenschr.*, *1876*, p. 394, and *1878*, p. 154. A similar loss, associated with hemianopia, was present in a case recorded by Lang and Fitzgerald, *Trans. Ophth. Soc.*, vol. ii, p. 230.

A lesion on one side of the pons causes a loss of the movement toward the side of the lesion, a conjugate palsy of both eyes.* The seat of the disease that has this effect is the tegmental region of the pons, at or above the nucleus of the sixth,† and the facts show that the path for this combined movement descends in the same side of the pons as that toward which the eyes are moved, and that the movement is effected through the nucleus of the sixth. The sixth nucleus must, therefore, act on the opposite third nucleus and internal rectus, and the path by which the influence is exerted is almost certainly the posterior horizontal fibres. It has even been thought that through these fibres, those of the third nerve for the internal rectus actually arise from the sixth nucleus; this assumption is opposed by many facts, and it seems that the horizontal fibres merely connect the two nuclei.‡

Since the movement is excited from the opposite cerebral hemisphere, the path must cross the middle line above the middle of the pons, and it probably does so at the corpora quadrigemina. If so, the arrangement must be that shown in Fig. 265. We do not know, however, whether the pons contains merely the path to the sixth nucleus, or whether there is a special centre for the movement, distinct from this nucleus. The latter is probable on

Fig. 265.

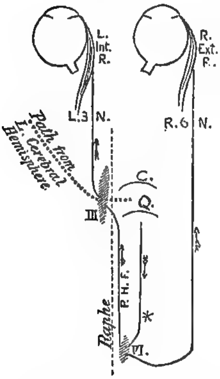

Fig. 266.

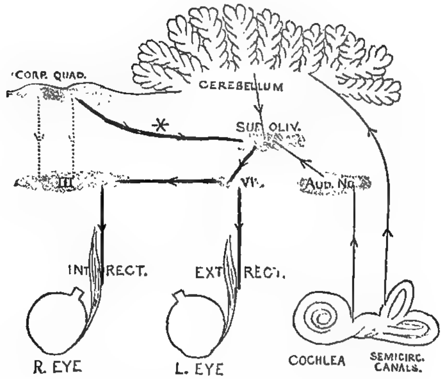

Fig. 265.—Diagram of the mechanism for the associated lateral movement.
Fig. 266—Diagram of probable relation of the superior olivary body to the associated lateral movement. The asterisk in each figure indicates the downward path.

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* Much attention has lately been given to this palsy. The most important writings on the subject are those of Broadbent, *Med. Times and Gaz., 1872*, vol. i; Duval and Laborde, *Journ. de l'Anat. et Phys., 1879*; Bernhardt, "Gehirngeschwülste;" Wernicke, "Gehirnkrankheiten," Bd. i; Mierzejewski and Rosenbach, *Neurolog. Centralbl., 1885*, p. 363, and Bleuler, *Deut. Arch. f. kl. Med.*, 1886, Bds. 37 and 38, in whose paper abstracts of most writings on the subject will be found.

† Very seldom below, and then reaching up to the nucleus. See note on next page.

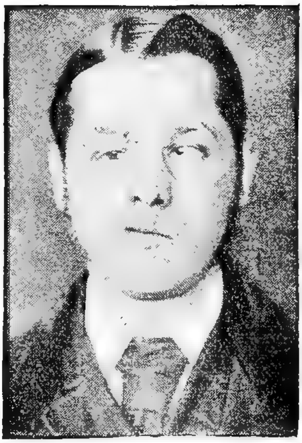
‡ The evidence is that the internal rectus is not always totally paralyzed; it has been known to act in convergence, although not in the lateral movement, and that when the sixth nucleus has been totally destroyed no degenerated fibres could be found in the opposite third nerve.

account of the relation of the movement to sensory impressions, especially to those of the auditory nerve, combined with the fact that a movement of the head is often associated with that of the eyes, so that a connection of the auditory nerve with the sixth nucleus would scarcely suffice. The connections of the superior olivary body, according to Betcherew, are precisely those that such a centre would have. By some fibres of the fillet, which degenerate downward, this body is connected with the corpora quadrigemina, and fibres pass between it and the sixth nucleus, the auditory nuclei, the cerebellum, and the lateral column of the spinal cord; the last may subserve the movement of the head.* If this hypothesis is correct, the mechanism must be such as is shown, in side view, in the diagram of Fig. 266. The arrow heads indicate the direction of conduction, and the thicker lines indicate the path by which the movement is excited by the will.

According as the disease does or does not involve the nucleus of the sixth nerve, the symptoms present some variation. If the disease is above the nucleus, there is a loss of the power of moving both eyes beyond the middle line toward the side of the lesion. If they are moved toward the other side, they can be moved back as far as the middle line, but no further. In most cases the other eye cannot be moved inward in convergence or alone, but in a few instances these movements have been preserved, although the inward movement associated with the outward movement of the other eye has been lost. We do not know the cause of this difference. If, however, the disease involves the nucleus of the sixth nerve, there is total palsy of the external rectus, so that the eye deviates inward and cannot be moved outward, even up to the middle line. The condition of the other eye is the same as in the first case.

In this condition the facial nerve is usually paralyzed as well as the sixth, its fibres being damaged as they course round and through the sixth nucleus. An instance of this palsy is presented by Fig. 267. The onset was sudden, and slight hemiplegia was present at first. The left sixth and facial nerves were completely paralyzed; the left eye deviated inward, and no outward movement was possible; the right eye could not be moved inward; the facial muscles presented the reaction of degeneration. If a lesion in the pons damages the fibres of the sixth nerve away from their nucleus, the paralysis of the external rectus is complete, but exists alone; there is no deficiency of movement

FIG. 267.



Complete paralysis of the left sixth and facial nerves (with inability to move the right eye toward the left) from an acute lesion on the left side of the pons. (*From a photograph.*)

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*In one recorded case the lesion was below the level of the sixth nucleus, the lower end of this nucleus being near the highest part of the lesion. It is probable that the superior olivary body was involved. Senator, *Berl. Psych. Gesellsch.*, July 9th, 1883.

of the opposite internal rectus. The lesion that causes these symptoms may be either a chronic process, such as a small tumor, or an acute lesion, hemorrhage, or, more frequently, softening. Other symptoms of disease on one side of the pons are often present, especially in the region of the fifth nerve on the side of the lesion, sometimes in the limbs on the opposite side.

Total Ophthalmoplegia.—Paralysis of all the muscles of both eyes, internal and external, while theoretically conceivable from disease at the neighborhood of the orbital fissure and optic foramen on each side, either in the orbit or within the skull, is practically only met with in cases of nuclear disease, and will be described in the account of this variety.

CAUSES AND CAUSAL VARIETIES.

Paralysis of the ocular muscles may be due to disease of the nerves in the orbit or at the base of the brain, to lesions of the fibres of origin between the surface of the brain and the nerve nuclei, and to disease of the nuclei themselves. A defect in certain ocular movements may also be produced by disease of the cortex of the brain and of the path from the cortex to the nuclei, but the loss that is usually so produced is that of the conjugate lateral movement of both eyes, already described in the account of hemiplegia, and slight ptosis on the side opposite to the lesion. It is possible, however, that a defect of upward or downward movement is sometimes due to disease of the hemisphere (see p. 609). Of the remaining lesions those of the nuclei (constituting the important group of nuclear palsies) will be separately described.

The nerves in the orbit and at the base of the brain are often damaged by inflammation. This may be syphilitic in either situation. In the orbit the inflammation is sometimes primary and "rheumatic," but is occasionally secondary to cellulitis. Within the skull, primary simple inflammation is almost unknown, and neuritis is generally secondary to meningitis. The nerves in the orbit are damaged by pressure only in rare cases of orbital growths, but within the skull one of the most common causes of their disease is compression by growths or aneurisms. All the nerves to one eye may be compressed in the wall of the cavernous sinus by an aneurism of the internal carotid or by a growth in this region,* and both third nerves may be damaged by an interpeduncular growth or an aneurism of the posterior cerebral artery. The sixth nerves are especially liable to suffer from distant pressure (see p. 481). Tumors of the nerves themselves are rare; neuromata are almost unknown, but nodular syphilitic growths sometimes occur and may be symmetrical, situated, for instance, on the two sixth nerves near their origin (Barlow). The fibres of origin suffer in their deep course from various lesions of the brain, hemorrhage, softening, tumors, and islets of disseminated sclerosis. Such lesions, involving the sixth or third nerve fibres, often also implicate the motor tract, and there results hemiplegia associated with palsy of one of these nerves. In consequence of the decussation of the motor tract below the origin of the nerve, the two symptoms are always on the opposite sides (see p. 515). Very rarely a small focus of softening in the crus may damage only some of the roots of the nerve.* Paralysis of all the ocular muscles has been caused by the fumes of charcoal, probably by an action on their nuclei.†

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* Many instances of such palsy are on record. A typical one is recorded by Nettleship, *Trans. Ophth. Soc.*, vol. i, p. 186.

Diphtheritic Paralysis.—A rare cause of palsy of the external muscles is diphtheria. The external rectus has been occasionally affected, and both internal recti have been weakened in association with the loss of accommodation so common in this disease. In one case all the muscles supplied by one third nerve were affected (Vadelot), and in two others there was paralysis of all the external muscles of both eyes.‡

Tabetic Paralysis.—In locomotor ataxy it is very common for some of the ocular muscles to be weakened. The loss of power is often transient and then may recur; sometimes it is permanent. Each form may occur at any stage of the disease, and is sometimes an early symptom. The external rectus, or the levator and superior rectus are the muscles most frequently affected; occasionally all the muscles supplied by the third nerve suffer. It is generally on one side only, but sometimes both eyes are affected. An example of double tabetic ptosis is shown at p. 295. We have scarcely any evidence at present of the cause of these palsies, but the fact that total nuclear palsy is sometimes met with, makes it probable that these also are sometimes of nuclear origin. The peripheral nerve degeneration of tabes has not been found in the ocular nerves, and its limitation elsewhere to the sensory fibres renders its occurrence in these nerves improbable. It must be remembered that many subjects of tabes have had constitutional syphilis, and that a true syphilitic ocular palsy may coexist with locomotor ataxy.

Rheumatic Paralysis.—Affections of the ocular muscles due to exposure to cold are regarded as due to rheumatic neuritis. They are always one-sided, and commonly involve a single nerve, or a single branch, or two contiguous branches, such as those to the levator palpebræ and superior rectus. Rarely, all the nerves of one orbit are involved, including the optic nerve (see p. 571). The onset of the disease is often attended with pain about the orbit. The occurrence of a rheumatic neuritis does not rest on pathological evidence, but is, nevertheless, highly probable. In two of the cases in which all the nerves of the orbit were affected the patient had previously suffered from facial neuritis due to cold. In one recorded case, in which swelling of the lids seemed to prove that the disease was orbital, the internal muscles escaped, on account, it is supposed, of the more central position of their nerves.§

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* Kahler and Pick, *Arch. f. Psych.*, x, 334.

† Knapp, *Arch. f. Augenh.*, ix, 2, 229.

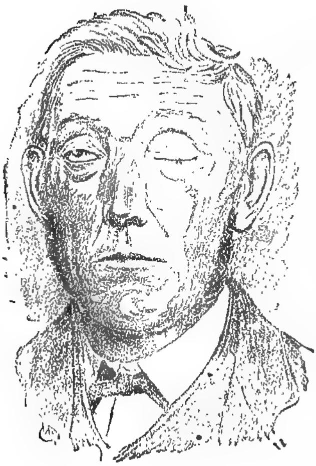
‡ Uhthoff, *Neur. Centralbl.*, *1885*, p. 125, and Mendel, ib., p. 128.

§ Möbius, *Cent. f. Nervenh.*, *1886*, p. 516. The symptoms followed exposure to cold.

Syphilitic Paralysis.—From the list of causes already given it is evident that syphilis may produce palsy of ocular muscles in several ways. The nerves may be the seat of isolated syphilitic inflammation or of a gumma; they may be involved in syphilitic meningitis or compressed by a syphilitic growth outside them. They may also be damaged by an aneurism of syphilitic origin. A young man, a few years after primary syphilis, became affected with palsy of one third nerve, which did not yield to anti-syphilitic treatment. After some months, he was seized with apoplexy and died, probably from the rupture of an aneurism of the posterior cerebral artery. Lastly, syphilis seems to predispose to degeneration of the nuclei of the nerves. Thus, the mere fact that the paralysis is due to syphilis is only the first step in the diagnostic problem of the nature of the disease.

There is one form of paralysis of the third nerves which occurs in the subjects of syphilis and deserves special description on account of its tendency to relapse and persist. One third nerve becomes paralyzed, improves under treatment, and then, perhaps while the treatment is being continued, the other third nerve suffers, and the affection of the first returns. An instance of the palsy is shown in Fig. 268. Syphilis was contracted four years previously, but the sudden onset of the palsy of the left nerve occurred during a severe cold. The patient was treated with iodide and mercury, but the course of the paralysis was that described above, and the permanent condition was that the left eye could only be moved outward and a little upward. There was extreme ptosis of the left eye and slight ptosis of the right. The internal muscles were also paralyzed. In another case, similar in course but less complete, the interval since the syphilis was thirty years. The nature of the lesion in these cases is uncertain. The course and limitation of the symptoms seems inconsistent with nuclear disease.

FIG. 268.



Double ptosis from paralysis of the third nerves, complete on the left side, partial on the right. Over action of frontales, greater on the side of the complete palsy.

Recurring or Periodical Palsy.—The last variety to be described is a rare form of transient palsy, usually on one side only, which comes on at intervals during many years.* It has been miscalled "relapsing palsy," but the attacks are recurrences, not relapses, and, in most instances, have been periodical. Both sexes suffer, but women are more subject to the disease than men. It often dates from early childhood, and has been known to begin at eleven months,† at fifteen months,‡ at five years of age, and later;[*] it continues up to at least middle life. An attack may occur at the same time each year, or at intervals of about six months. A much longer interval usually separates the early attacks. Sometimes the intervals have been long and irregular, amounting to several years,[†] while in other cases they have been short, and attacks have even occurred at each menstrual period.[‡] As a rule, the paroxysm begins with severe pain in the eye, and often with headache and vomiting. These symptoms last two or three days; the palsy may accompany them or may come on as they lessen. In many cases only the third nerve has been affected, but in some the external rectus was also involved. The palsy may be complete or incomplete; the internal muscles are often, but not always, affected; there is usually ptosis. The loss of power lasts for a few days or weeks and then gradually passes away. When the attacks are frequent they are usually brief. In many cases a slight defect of power has persisted during the intervals. In one case there was concentric contraction of both fields of vision, greatest in the paralyzed eye, and varying in degree in proportion to the motor palsy (Thomsen). The attacks usually last for several weeks, from three to eight, and, as a general rule, when the intervals are long, so are the attacks. When the paroxysms recur frequently, as, for instance, every month, they last for a few days only. In several cases the headache was unilateral. In those subject to the disease, an attack has been brought on by mental shock.

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* Cases have been recorded in this country by Saundby, *Lancet, 1882*, ii, 345, and 1885, i, 57, and by Snell, *Trans. Oph. Soc.*, v, 193.

† Möbius, *Neur. Cent., 1884*, p. 307.

‡ Snell, loc. cit.

The nature of this disease is mysterious. It has been compared to migraine, to which it is, perhaps, more closely allied than to any other disease. In one case there was a strong family history of migraine (Snell), but the long duration of the attacks, the motor character of the chief symptoms, their long duration, and the persistence of slight defect of movement, are marked differences from purely migrainous and neuralgic affections.[§] It has been suggested that the cause is organic disease, with periods of activity, but this theory is scarcely tenable. Vasomotor disturbance has, of course, been invoked to explain it, but this is only putting the difficulty further back. The only post-mortem examination yet obtained does not clear up the mystery. A women died of phthisis at the age of thirty, who had suffered, since childhood, from periodical attacks of palsy of the left third nerve.

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[*] Thomsen, *Charité-Annalen*, *1885*, p. 562.

[†] Five to nine years in a case recorded by Camuset, *L'Union Méd.*, *1876*, p. 906.

[‡] Hasner, *Prag. Med. Wochenschr.*, *1883*, No. 10.

[§] A case recorded by Buzzard (" Clin. Lect.," p. 164) is, perhaps, a connecting link between these cases and pure neuralgias. A woman had been subject for many years to fortnightly paroxysms of neuralgia of the first division of the fifth nerve, and for two years before she came under observation each attack was *followed* by partial palsy of the third nerve, lasting for a few days.

In a case recorded by Pflüger there were periodical attacks of paralysis of variable seat; of the left third and facial nerves at eighteen, of the nerves on the right side at twenty, of the left third nerve again at twenty-two, and six months later of the left sixth and facial. Each attack lasted from one to two months, and was preceded by pain, which before the last was about the left mastoid process.

The nerve appeared gray, and its roots were surrounded by small gray granulations which contained tubercular bacilli, which did not extend into the crus. Some fatty degeneration was found in the muscles supplied by it, the other muscles being normal.

NUCLEAR OCULAR PALSY.

Disease of the nuclei, third, fourth and sixth, are frequent causes of paralysis of the eye muscles, internal and external. The symptoms vary much according to the position and character of the disease. The nuclei may be the seat of various lesions, acute and chronic. The isolated affection of the sixth nucleus has already been considered in the account of the conjugate palsy which such disease produces. The nucleus of the fourth nerve may be regarded as part of the third. We have, therefore, only now to consider the lesions of the third nucleus, together with those of the sixth nucleus, in so far as it is associated with the third in primary degeneration. Some of the symptoms of nuclear disease have already been described, and need only be enumerated here to complete the account of the subject.

The nuclei of the third nerves include centres for most of the external and all the internal muscles and movements. Even the reflex dilatation of the iris is said to depend on a centre in this nucleus (although the third nerve fibres are not concerned in the action)—a fact which is not surprising when we consider how closely dilatation and contraction are connected.

The third nuclei are occasionally the seat of hemorrhage, of softening, and of morbid growths. They are said to be sometimes the seat of acute inflammation, which has been termed "acute polio-encephalitis superior."* The most frequent lesion, however, is slow, progressive degeneration. It is this that is the cause of most forms of progressive ophthalmoplegia, which is thus a synonym for nuclear disease. It is, indeed, a more accurate term, for we do not yet know that all cases which seem to be nuclear are really so.†

Acute Ophthalmoplegia; Acute Nuclear Palsy.—Sudden palsy of all the ocular muscles occurs in some cases of hemorrhage into the region of the nuclei, but the ocular symptoms are usually subordinate to those of apoplexy, and most cases are quickly fatal. In a few instances recovery has taken place, and palsy of the third nerves has usually remained partial. It is probable that the lesion in such cases has generally been softening from disease of the arteries supplying the nuclei with blood.‡ It has been conjectured that nuclear extravasations are sometimes caused by injury. The cases of "polio-encephalitis superior," recorded by Wernicke,* occurred, one after poisoning with sulphuric acid, and two in the subjects of chronic alcoholism. There was hemorrhagic softening of the nuclei and focal symptoms consisting of associated palsy of the ocular muscles, progressing to total external paralysis, while the sphincter iridis and the levator escaped. There was some optic neuritis. But the focal symptoms were obscured by those of general cerebral disturbance; somnolence increasing to coma, and ending in death at the end of about ten days. A few other cases have been recorded as acute nuclear palsy, but the patients recovered, and the nature of the lesion is doubtful. In one of these cases, described by Etter,† paralysis of the optic and bulbar nerves was added to that of the eyeball muscles; the symptoms developed in the course of three days, and improvement commenced at the end of a fortnight. In another, recorded by Möbius,‡ paralysis of one side of the face coexisted, and there was difficulty in speaking and swallowing, together with paralysis of the legs and loss of the knee jerk. Whether acute multiple neuritis ever involves the ocular nerves we do not know; the possibility that such peripheral neuritis may simulate central disease must be borne in mind. Diphtheritic palsy may, perhaps, be added to the list of acute nuclear palsies; it is probable that the diphtheritic cycloplegia is of nuclear origin, but whether the rare palsy of the ocular muscles is due to nuclear or peripheral disease is more doubtful (see Diphtheritic Paralysis).

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* By Wernicke, in contradistinction to a similar affection of the bulbar nuclei, which has been termed "polio-encephalitis inferior." It is an inaccurate name, which ought not to come into use, because, if any meaning is attached to it, it should be that of inflammation of the cortex. As a matter of fact, the latter has been termed "polio encephalitis," and yet the qualifying "superior" is attached to a lower lesion.

† An able summary of present knowledge on the subject of nuclear palsy has been published by Mauthner, "Vorträge," Heft 12, 1885.

‡ An instance of such sudden palsy with apoplexy is recorded by Sturge, *Trans. Oph. Soc.*, vol. i, p. 165. According to Heubner the anterior and posterior parts of the nucleus are supplied from different arteries, and hence acute palsy from softening may, conceivably, be partial.

Chronic Ophthalmoplegia; Chronic Nuclear Paralysis.—The chronic forms are of greater frequency and practical importance than the acute varieties. Among them we must include isolated loss of the reflex action of the iris, isolated palsy of the ciliary muscle, palsy of all the internal muscles, of all or most of the external muscles, and, lastly, of both the external and internal muscles. The external palsy was described by v. Graefe under the name "progressive ophthalmoplegia,"§ and was carefully studied many years later by Hutchinson,|| who proposed for it the name "external ophthalmoplegia" in distinction from the "internal ophthalmoplegia" which he had previously described.¶

Later observations have shown that we cannot separate these forms of chronic palsy; all occur under similar conditions; they may be variously combined, and the dependence of one of them on nuclear disease has been conclusively proved. Each form is met with in association with locomotor ataxy; the simple loss of reflex action is indeed present in the majority of

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* "Gehirnkrankheiten," ii, 232.

† *Cor.-Bl. f. Schweizer. Aertze, 1882*, p. 769.

‡ *Cent. f. Nervenh.*, *1882*, 465.

§ *Arch. f. Ophth.*, Bd. ii, 1856, p. 299. The term "ophthalmoplegia" was first used by Brunner in 1850.

|| *Med.-Chir. Trans*, vol. lxii, 1879, p. 307.

¶ Ib., vol. lxi, 1878, p. 225.

cases of this disease. It has been mentioned that, when this loss exists alone, the subjects of it have frequently suffered from constitutional syphilis, and the same fact is true of the other forms of ophthalmoplegia.* They sometimes occur, however, without this antecedent, and occasionally affect young persons. They are met with more frequently in males than in females, resembling in this their congener, tabes. We cannot, however, at present draw any general etiological conclusions from a comparison of recorded cases, because it is probable that some of those that have been regarded as examples of this form are not really such. It is seldom that any immediate cause can be traced; in one instance the symptoms are said to have come on after a wetting.

Progressive nuclear ophthalmoplegia begins with weakness of one or more of the ocular muscles, and often the muscles first affected are those that are associated in action—the superior recti and levators, the two internal recti, or the internal rectus on one side and the external rectus on the other. Sometimes the order of affection is quite irregular. The loss of power, at first sight, slowly increases; often a strong effort shows more power than is habitually exerted, and the weakness may be less in the morning than in the evening. The disease slowly extends, until at last, often after several years, only one or two of the muscles of both eyes retain power, and sometimes every muscle is paralyzed. The levators, however, seldom become completely paralyzed, and may be unaffected. The aspect of the patients is peculiar; when there is partial ptosis, this gives them a sleepy expression, and in total palsy without ptosis, a staring look results from the immobility of the eyes, which are fixed in mid-position. Sometimes, although rarely, they are slightly prominent. Occasionally one eye is much more affected than the other; on one side there may be total palsy, on the other ptosis only. Sometimes the palsy is unilateral. The internal muscles are often unaffected; when this is the case the diagnosis of nuclear palsy can be made with confidence, because the escape of the internal muscles in bilateral disease of the nerve trunks is scarcely posssible. This feature was thought to be a characteristic of progressive ophthalmoplegia by von Graefe, but it is not so; the affection of the external muscles may be combined with loss of reflex action of the iris, with cycloplegia, or with both as "total ophthalmoplegia." In the case presently to be mentioned, in which the nature of the lesion was ascertained, the internal muscles were affected. Double vision may trouble the patient in the early stage of the affection, but it generally passes away as the disease progresses, and is sometimes absent from the first, perhaps when the early loss is of associated movements. The duration of the malady is long. In one of Hutchinson's cases the symptoms continued for seventeen years. In one of Mauthner's the affection was limited to one eye for twenty years. The symptoms may exist alone, but they are more frequently associated with indications of other disease of the nervous system—with optic nerve atrophy, with affection of the bulbar nerves (rarely amounting to typical bulbar palsy), with progressive muscular atrophy, with general paralysis of the insane, and especially with locomotor ataxy. A similar condition is, perhaps, sometimes congenital and even hereditary.*

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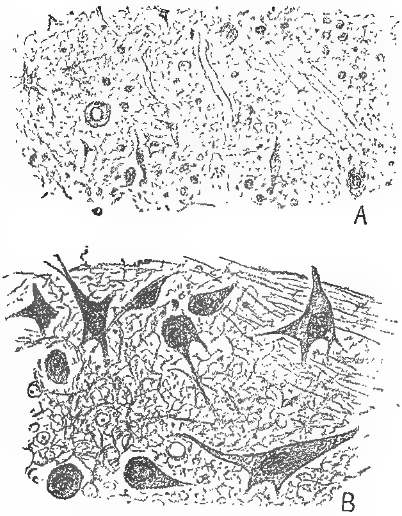
* As in so many of these degenerative diseases, the facts that can be ascertained probably under-represent the relation to syphilis. The case of Mr. Hutchinson, examined by me (see p. 619), is an illustration of this. The man persistently denied any venereal sore and no trace or history of syphilitic disease could be found. But some years later a child of this man was brought to Mr. Hutchinson, with characteristic notched teeth and interstitial keratitis.

In a case of Mr. Hutchinson's, in which I examined the brain, the state of the nuclei of the ocular nerves was nearly the same as that of the gray matter of the spinal cord in progressive muscular atrophy. The patient was a man, aged fifty-five, who had had syphilis. The symptoms began gradually seven years before, with palsy of the internal recti and ciliary muscles. Ultimately, all the ocular muscles became very feeble, the optic nerves atrophied, there was mental excitement, and some palsy developed in the limbs—the condition resembling that of some forms of general paralysis of the insane. The roots of the ocular nerves, outside and inside the brain, were gray, small, and contained scarcely any normal fibres. In their nuclei a few nerve cells of normal size were seen, but these had, for the most part, lost their processes, and a large number of the cells were reduced to small angular bodies or had disappeared (Fig. 269). In the intervening tissue there were many connective-tissue nuclei. The change involved the sixth, fourth, and the whole of the third nuclei. The other nerve roots and nuclei were normal. The spinal cord was not obtained. Thus, the lesion, in a fairly characteristic case, was a degeneration, limited to structures having a common function. The internal palsies occur under similar conditions, and their frequent limitation to a single function in the two eyes point to a like change of more restricted seat. It is only by toxæmic influences or degeneration that structures of common function are selected, as it were, for isolated disease. The same conclusion may be drawn regarding such associated palsy as the loss of convergence and accommodation. But, as we have already seen, the internal loss may pass away, even after it has lasted for many months, and perhaps

FIG. 269.



A, part of sixth nucleus in progressive ophthalmoplegia; B, cells of the normal nucleus, for comparison.

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*Thus Hirschberg (*Berlin Gesellsch. f. Psych.*, June 8th, 1885) has described the case of a man with congenital double ptosis and paralysis of all the ocular muscles, incomplete in the superior oblique and the internal muscles, whose mother presented a similar condition, while his son had congenital ptosis and paralysis of the superior recti.

for more than a year. I have known the light reflex to return, in stationary tabes, after it had been lost for at least two years, and the remarkable case of internal ophthalmoplegia recorded by Eales (see p. 609) is an instance of the way in which such a palsy, limited and complete, may pass away. It is difficult to conceive that paralysis of such duration can be of merely functional origin; it seems more probable that it is due to alterations in the nutrition of the nerve elements, such as may constitute the first stage of degeneration, but do not proceed beyond a molecular, recoverable degree. The analogy of the nutritional changes in diphtheritic paralysis may be borne in mind. It has been thought that chronic ophthalmoplegia may be due to the compression of the nuclei by distention of the aqueduct of Sylvius in internal hydrocephalus, but such a mechanism is extremely doubtful. It is certain that in most cases in which there is extreme distention of the acqueduct there is no ocular palsy.

When there is paralysis of internal muscles in both eyes, or paralysis of the external muscles without affection of those within the eye, disease of the nuclei can be inferred with confidence. The internal muscles never escape in disease of the nerve trunks when this paralyzes many of the external muscles. Central disease is also shown by paralysis of those muscles of both eyes that are associated in their action. When, however, the internal and external muscles of both eyes are involved, the diagnosis of nuclear disease can only be made after careful exclusion of disease at the base of the brain. If other nerves are implicated, it is still probable that the palsy is nuclear if all the symptoms come on gradually, without evidence of a compressing lesion, and under such conditions, or with such associations, as suggest a degenerative lesion. The diagnosis of acute nuclear palsy rests chiefly on the limitation of the symptoms to the range of definite function, on the disturbance of contiguous nuclei, and on the escape of nerves which, at the base of the brain, lie between those that are involved. If two nerves suffer whose nuclei are near together, and a third escapes which lies between the others outside the brain, we may infer that the disease is nuclear, and not basal.

OTHER FORMS OF PTOSIS.

Reflex Ptosis.—In very rare cases irritation of the fifth nerve has been observed to cause transient drooping of the eyelid, which must be referred to inhibition of the centre for the muscle. A physiological connection doubtless underlies this effect; spasmodic closure of the lids frequently results from irritation of the fifth nerve; for closure of the lids there is always relaxation of the levator (as is shown by the slight amount of orbicular contraction which will suffice), and hence irritation of the fifth nerve has a tendency to cause relaxation of the levator. Reflex ptosis has been observed to follow division of the fifth nerve, no doubt from the irritation of the fibres by the section (Longet). It may follow the extraction of a tooth, as the following case shows: An upper right molar, decayed and causing much pain, was extracted from a woman aged fifty-five. The tooth was firm, and the patient had no anæsthetic. A few hours afterward right-sided ptosis existed, varied by occasional attacks of clonic spasm in the levator, each lasting a few seconds. Both symptoms continued, in slighter degree, on the following day, and gradually improved, so that the condition of the eyelid by the fifth day was natural. On the sixth day there was some pain referred to all the branches of the fifth nerve, but this soon passed off, and there was no recurrence of nervous symptoms.*

Ptosis from Paralysis of the Sympathetic.—The unstriated muscular fibres (fibres of Müller), which exist in the fascia of the orbit, and are innervated from the sympathetic, act indirectly on the tarsal cartilages, by the connection of these with the fascia. They probably aid to a slight extent in maintaining the upper lid in its normal position. When the cervical sympathetic is paralyzed, the upper lid on that side is a little lower than on the other. Its movements are unimpaired. Ptosis from this cause is distinguished by the presence of other symptoms of paralysis of the sympathetic, such as contraction of the pupil, and sometimes dilatation of the vessels of the surface or altered secretion of sweat.

Congenital ptosis, not due to malformation of the lid, is usually bilateral and partial. It is generally associated with defective power of elevation of the globes, and is probably due to a congenital central defect. It may be hereditary (see note, p. 619). Slight double ptosis sometimes runs in families, and may (as I have seen) affect chiefly the female members. It may only come on after the time of puberty. The characteristic over action of the frontal muscles causes the anxious aspect already mentioned, and this may actually be more conspicuous than the slight drooping of the lids to which it is secondary. A very similar double ptosis sometimes comes on after middle life in neurasthenic individuals, especially women. In late life it is usually permanent; at an earlier age it may pass away. Allied to this is a form which may be termed *morning ptosis*. During sleep the levator is relaxed, and many sound sleepers find a difficulty in opening the eyes on being first roused. In weakly women this difficulty is sometimes exaggerated; after waking, it may be impossible for them to raise the lids for ten to thirty minutes. During the rest of the day they have no difficulty. This condition is usually recovered from.

Hysterical ptosis is occasionally met with, single or double. It is generally accompanied by a slight spasm in the orbicularis; this can readily be proved by making the patient look upward, when the spasm of the orbicularis becomes much greater, to prevent the lid from moving with the eyeball. When double (Fig. 270) both eyelids droop, and the patient puts her head back when she is told to look up. If the head is held in the attempt to look up, both orbiculares contract, and prevent the lids rising. This contraction of the orbiculares proves that there is no true paralysis of the levators. There is sometimes over action of the frontales associated with the ptosis, as if from a struggle to overcome the contraction of the orbiculares.

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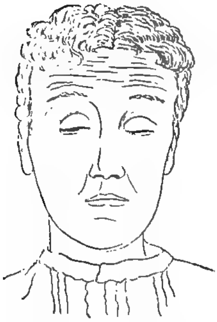
* Communicated to me by Mr. H. R. Gooding, by whom the case was observed.

FIG. 270.



Hysterical ptosis, bilateral. Over-action of frontales.

TREATMENT.—The most important element in the treatment of paralysis of the ocular muscles is that of the morbid processes that causes them, and this is described elsewhere. It is only necessary here to mention those measures that the special effect of the disease renders necessary. Whenever the onset is acute or subacute, or if other symptoms suggest inflammation, counter-irritation should be employed. A blister may be placed behind the ear or at the occiput when the disease is probably at the base of the brain; on the temple, if it is in the orbit. The blister is often followed by a striking increase in the power of the affected muscle. In acute rheumatic cases, hot fomentations should be applied to the orbit, forehead and temple, frequently repeated. Leeches may be applied to the temple, at the onset, if the patient's general condition does not contra-indicate their use. In syphilitic cases, appropriate remedies should be freely employed. In most cases of neuritis, indeed, whether syphilitic or not, mercury is probably the most efficient agent we possess. Defective general health must be corrected by tonics, of which iron and strychnia are the most useful.

For nuclear palsy, in syphilitic subjects, mercury and iodide have seemed useful when the onset was acute or subacute. It is probable that, in some instances, these drugs have cured the patient, but in other cases it is doubtful whether the improvement has been more than the tendency to recovery which usually follows an acute process that has spent its force. In cases that are chronic from the beginning, this treatment is as powerless as it commonly is in locomotor ataxy, and the most effective measures are those suited to primary nerve degenerations elsewhere, arsenic, quinine, strychnine, and small tonic doses of mercury in syphilitic patients. Strychnine may be given, with advantage, by hypodermic injection, as in progressive muscular atrophy (see p. 372). Such treatment as is here suggested appeared distinctly beneficial in one case, the sequel of which renders it peculiarly instructive. A gentleman, aged forty, who had probably not had syphilis, presented entire loss of the reflex action of each iris, with large pupils; accommodation was normal and the iris contracted with it. There was also slight double ptosis, weakness of both internal, of the left superior, and of the right inferior recti, and great diminution in the right knee jerk, all of gradual onset and from six to eighteen months' duration. At the end of a three months' treatment the external ocular muscles had become normal, although the reflex action was still lost, and the right knee jerk had increased to the same degree as the left. He continued without relapse for nearly two years, and then suddenly presented the acute mental derangement of general paralysis of the insane, from which he quickly died.

Recorded cases of the recurrent or periodical palsy do not show that any treatment has a marked influence on the attacks. Early counter-irritation probably affords most prospect of modifying their course, and tonic treatment may be adopted before an expected attack. In cases in which the attacks recur frequently, the treatment adopted should be that for migraine.

Direct Treatment.—Electricity has been recommended and employed in the treatment of these paralyses, but its influence is not great. Direct application to the affected muscle is scarcely practicable. It has, indeed been carried out by a very small electrode, or by a wet camel-hair brush in the centre of which is a conducting wire. The extreme sensitiveness of the conjunctiva renders the application most painful, and although the pain may be lessened by cocain, the application of a voltaic current, sufficiently strong to stimulate the muscle, in such proximity to the retina and on such a delicate structure as the conjunctiva, is scarcely safe. In most ocular palsies the muscles do not respond to faradization. The voltaic current may be applied through the eyelid, in the case of a superior or inferior muscle, the eye being turned in the opposite direction. The considerable diffusive power of this current makes it probable that the electricity would reach the muscle, although even thus a current cannot be safely used of sufficient strength to produce evidence of stimulation. The application of a faradaic current through the eyelid is useless, even if the muscle would respond to it, because a thin layer of contractile muscle prevents any stimulation of the subjacent tissues. For these reasons, most authorities are agreed in deprecating any direct application. A method of indirect electrization has been recommended (by Benedikt and others) which consists in placing one pole (anode) on the forehead, the other (kathode) on the margin of the orbit near the affected muscle. If the faradaic current is employed, the orbital pole is kept still; if the voltaic current is used, this pole is stroked along the skin, or the circuit is alternately made and broken by an interrupter. A slight increase of power in the muscle may be observed after the current has been so applied for a few minutes, but it quickly passes off, and in cases free from sources of fallacy I have never been able to trace any permanent effect. It is true that works on electro-therapeutics contain cases in which improvement or cure was ascribed to this agent, but in most instances the authors have ignored the tendency to spontaneous improvement in recent cases, and the influence of drugs, as iodide of potassium, which were given at the same time in many instances.

The diplopia due to partial paralysis of a muscle may be removed by the use of a prism. One strong enough to completely fuse the images is undesirable, because it tends to prevent the strengthening influence of effort. A weak prism, strong enough so to approximate the images as to permit their fusion by muscular action, sometimes does good. It may be used for an hour daily as a gymnastic exercise. The giddiness due to erroneous projection can only be removed by putting the eye out of action by an opaque glass; this glass may be in a pair of spectacles, the other glass being merely tinted; the opacity is then inconspicuous to an observer. But this has the same disadvantage of withdrawing the stimulus of exertion. An opaque glass over the sound eye is useless for the vertigo, and increases the amount of the secondary deviation.

Operative interference can do little in cases of ocular palsy. The only condition in which it is admissible is that in which antagonistic contracture has been developed, and the paralyzed muscle has regained power, but cannot overcome the opposing contracture. In such a case the tendon of the contractured muscle may be divided, without disturbing its other connections; it forms a fresh attachment a few millimetres further back, and the result on the movement of the eye is often very satisfactory.

The treatment of paralytic *ptosis* is that of disease of the third nerve. The muscle itself is not accessible to electrical stimulation. In the double ptosis of nervous debility, nervine tonics, quinine and strychnia, are useful, and their influence is aided by local treatment, which stimulates the fifth nerve, and so produces a reflex action on the centre of the levator. Electricity may be used, either the voltaic or induced current, applied to the skin about the orbit, but a stimulating liniment which stings the skin (as chloroform liniment on spongiopiline) usually answers as well. The morning ptosis can always be quickly relieved in this manner. The hysterical ptosis is often a very obstinate affection. Blisters to the temple, faradaism, and (in the unilateral form) tying up the other eye, are the most efficient means, and usually in time remove the symptoms.

SPASM OF THE OCULAR MUSCLES.

The varieties of spasm of the ocular muscles are numerous, but only a few are of medical significance. Two classes may be dismissed with a mere mention. (1) Those that are connected with disorder of the ocular visual process, as the convergent strabismus of hypermetropia, the divergence of myopia, the adaptive deviation that occurs when there is partial opacity of the media, and the irregular position that often accompanies absence of sight. (2) The secondary deviation in one eye, consequent on palsy of a muscle of the other eye, and the antagonistic contracture in the same eye. The second class has been already described; those of the first do not come within the scope of this work. Of the remaining forms, one of great importance—nystagmus—will be separately described. The remainder may be grouped into five classes.

(1) *Associated Spasm from Central Disease.*—In a paralyzing lesion of one hemisphere, the eyes deviate toward this side, but the deviation is merely the unopposed influence of the opposite hemisphere. An irritating lesion of one hemisphere causes conjugate deviation toward the opposite side, which is the result of spasm; this occurs at the onset of unilateral convulsions, and in association with muscular rigidity of the corresponding limbs. In one interesting case, deviation to the right was, during four months, a persistent symptom of a depressed fracture below the left parietal eminence, and it ceased when the depressed bone was raised by trephining.*

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* Thompson, *Brain*, April, 1883, p. 99. It may be noted that the fracture was behind the

Since a lesion in one side of the pons causes a loss of the conjugate movement toward the side of the lesion, irritating disease may be expected to cause spasmodic deviation toward that side, but such spasm is seldom observed. An acute lesion, which causes the conjugate paralysis, may, however, produce a distinctly spasmodic deviation toward the opposite side, apparently by an indirect influence on the corresponding centre of the other side of the pons.*

(2) *Irregular Spasm from Brain Disease.*—In irritating diseases of the base of the brain, especially in meningitis, spasm occurs in one or more of the ocular muscles, causing slight irregular deviations of the visual axes. It is comparable to the rigidity that occurs in the limbs, and may vary in seat and degree from time to time. It is probably due to irritation of the motor nerve trunks. Care must be taken not to mistake for spasm the deviation from loss of power which is so common from the same cause. In spasm there is deviation when the eyes are at rest, in the mid-position; in recent paralysis there is not. It must be remembered that paralysis and spasm are often conjoined. Such irregular spasm is, as a rule, tonic. Slight irregular spasm of the ocular muscles sometimes occurs in chorea. It is rarely sufficient to be seen, but causes transient diplopia, which may be thought to indicate organic brain disease, if the dependence on chorea is not known.

(3) *Chronic spasm in individual muscles* is extremely rare, apart from the secondary deviation already described. In the best-marked cases the spasm has not been continuous, but has occurred on certain movements of the eyes, sometimes with pain. Thus, of two cases recorded by Hock,† in one, as soon as an object was moved to the right of the middle line, spasmodic contraction occurred in the right internal rectus (with great pain) bringing the eye into extreme adduction, and as soon as the object was moved to the left beyond the middle line the spasm relaxed, and the right eye moved outward into the fixing position. In the other case there was slight weakness of the right internal rectus, and as soon as the object was moved to the left of the middle line, spasm in the left external rectus moved the eye into extreme abduction. The spasm thus affected the muscle that would be the seat of secondary deviation, but was distinguished from this by its extreme degree. This case is interesting because the spasm occurred with slight left hemiplegia, and, from some accompanying disturbance of taste, may have been due to disease in the upper part of the pons. Sometimes two muscles acting together are involved in the spasm, as, in one case, the superior oblique and inferior rectus (Stilling).

(4) *Hysterical Spasm.*—In hysterical fits the eyes are usually directed

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region in which experiment places the centres for the lateral movement, which is in front of the central convolutions (see p. 456).

* As in a case I have recorded of acute anæmia of the right half of the pons, which caused deviation of the eyes to the left, increased from time to time with violent nystagmus. (*Trans. Ophth. Soc., 1884*, p. 308.)

† *Wiener Klinik*, April, 1876, p. 116.

either upward and to one side, often so as to almost entirely conceal the cornea, or they are directed inward in strong convergence. They never diverge. Sometimes the convergence persists during the intervals, and is usually associated with spasm of accommodation.

(5) *Paroxysmal Spasm.*—In convulsive attacks in which the convulsion is unequal on the two sides, the eyes constantly deviate (with the head) toward the side most convulsed, and if the second side is affected in greater degree after the first, the eyes subsequently deviate toward that side. When the fit is over there may also be a deviation from the side most convulsed. But cases are occasionally met with in which a single muscle is the seat of paroxysmal brief spasm, resembling in miniature an epileptoid seizure, and sometimes attended with transient obscuration of consciousness. During the attack there is diplopia and often giddiness from the erroneous projection. Clonic spasm in the obicularis may occur at the same time. Such attacks, for instance, occurred in a man aged forty-seven; there was very slight permanent defect of power in the left external and both internal recti. Occasionally, without any exciting movement of the eye, tonic spasm in the external rectus would draw the left eye strongly outward for about thirty seconds. During this time there was constant winking with both eyelids, which seemed to the patient to be due to an effort to get the eye right, but could not be prevented. The deviation suddenly ceased, but for a few minutes afterward the left eyelid was about one-twelfth of an inch lower than the other, and then both were alike. There was no history of venereal disease, and syphilis was, for other reasons, very improbable.

Another patient, aged thirty-six, also without history of syphilis, suffered from frequent attacks (lasting only a few seconds), two of which I witnessed. There was a sudden sensation of heat, spreading from the left inner canthus and extending over the eye and temple, accompanied with impairment of sight of both eyes, varying from slight dimness to absolute loss of sight, and with abduction of the left eye from mid-position to about half-way to the outer canthus, the right eye being still. If walking, he deviated to the left during the attack, probably from erroneous projection of the left field. In the intervals, movements of the eyes were perfectly normal. The attacks consisted of tonic and then clonic spasm, and resembled perfectly miniature epileptic convulsions. Some years latter this spasm had ceased, but the patient presented complete internal and partial external ophthalmoplegia, clearly due to nuclear degeneration.

NYSTAGMUS.

Rhythmical movements of the eyes, involuntary, frequent, usually bilateral and similar in each eye, produced by alternating contractions in opposing muscles, are termed "nystagmus." The conditions under which this symptom occurs are many and various. They may be roughly divided into four classes:—

(1) Local affections of the eyes which interfere with sight, but have no other character in common—opacities of cornea or lens, and inflammations

or degenerations of retina and choroid. It scarcely ever results from simple errors of refraction, however considerable. These eye diseases cause nystagmus chiefly when they occur in infancy or early childhood. Nevertheless, it is doubtful whether nystagmus results from blindness which is actually congenital, although slowly-rolling movements of the eyes are observed in such cases. In adult life ocular disease alone rarely causes nystagmus, but it certainly aids the development of nystagmus due to other causes.

(2) In albinism this condition is very common.

(3) It occurs in miners, chiefly in those who work in coal mines and who use the pick in a stooping or lying posture. It is also far more frequent where the dim safety-lamp is used, than in mines which can be worked with brighter naked lights.

(4) It occurs in diseases of the nervous system of the most varied seat and character. It is usually present in disseminated sclerosis and in hereditary ataxy, but not in ordinary locomotor ataxy. In other diseases attended with tremor it is rare. In paralysis agitans it is never met with, a curious fact, considering how closely the alternating movement of nystagmus resembles that of shaking palsy. It occurs in many diseases of the brain, diffuse and focal;* meningitis, meningeal hemorrhage, thrombosis in sinuses, and in cases of tumor, hemorrhage, and softening in various situations. It is especially common in tumors of the cerebellum, and I have also known it to occur in disease of one side of the pons; the quick movement was from the side of the lesion and there was associated palsy of the lateral movement toward the other side. Nystagmus often occurs in cases of degenerative disease of the spinal cord and brain, in which there is no evidence of a focal lesion.

The movement is usually bilateral; very rarely it affects one eye only. Horizontal (lateral) movement is the most common; next a rotary motion; vertical movement is the least frequent, but one-sided nystagmus is said to be generally vertical. The extent of the movement varies from one to ten millimetres; from two to four millimetres is the common range. Occasionally, when it is too slight to be seen with the naked eye, it may be observed with the ophthalmoscope when the fundus is examined. The frequency is usually from 60 to 200 separate movements (*i. e.*, in the same direction) per minute; rarely it is slower than 60; occasionally it is too frequent to be counted. The rhythm is usually regular; in some cases there are slight variations in frequency from time to time. If there is slow nystagmus when the eyes are at rest, the oscillations often become more frequent when the eyes are moved. The alternating movements are not equally quick; there is a sudden rapid movement in one direction and slow return. In describing nystagmus, it is said to be to the side toward which the movement is most rapid. In many cases the quick movement is in the direction in which the nystagmus is most energetic on voluntary movement.

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* I am inclined to think that it is more common in organic brain disease when there is impairment of sight from optic neuritis, etc., than when there is not.

The symptom may be constant, or it may occur only when the eyes are directed in a certain direction, sideways, upward or downward. Very rarely there is a slight movement of the head, corresponding to that of the eyes in time and in direction, or in the opposite direction to that of the eyes.* All forms cease during sleep. In cases that date from infancy the patient is never conscious of the movement, nor is there any apparent movement of objects; in cases which commence in later life there sometimes is an apparent movement of objects; more frequently there is not. The apparent movement of objects is generally in the direction of the quick movement of the nystagmus. Rarely, there is a movement of the upper lid synchronous with that of the eye; this association occurs chiefly when the nystagmus is vertical, but I have seen it once when the movement was horizontal. In vertical nystagmus there is often a slight movement of the lid, communicated to it from the eye; from this, the actual spasm of the lid must be carefully distinguished.

The nystagmus of miners presents considerable variations in different cases. Any form of movement may be met with, and it may occur in all or only in one position of the eyes. It may occur only in the recumbent posture, such as the miner assumes in his work, and may cease when he is upright.†

The physiological pathology of nystagmus is still, to a large extent, obscure. The perfect bilateral symmetry of the movement, conspicuous in the vast majority of cases, indicates its central origin, and is opposed to the simple, but inadequate, explanations which ascribe it to muscular fatigue. Why the steady tonic contraction of health should be broken into clonic contraction, no one has yet been able to say. But the physiological associations of the ocular movements enable us to understand something of the influences that produce it. We may take as the simplest form that in which the movement is lateral. For lateral movement of the eyes, there is a separate centre in the pons on the side toward which the movement takes place. From this the spasm of lateral nystagmus must be directly produced. Each voluntary movement necessitates a relative degree of relaxation of the antagonists and their subsequent contraction to bring the eyes back to the middle line.‡ Hence, there must be a mutual connection between the functional states of the antagonistic centres, and this probably underlies the alternation of movement in nystagmus.

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* In a case lately under my care, with symptoms of cerebellar tumor and lateral nystagmus, the pharynx and larynx were the seat of similar movement; that in the pharynx was horizontal, toward the middle line; in the larynx there was a similar lateral movement of the arytenoid cartilages. The rate of the movement was the same as in the ocular muscles, 180 per minute (Spencer, *Lancet*, *1886*, vol. ii, p. 702).

† See Snell, *Trans. Ophth. Soc.*, vol. iv, p. 315.

‡ That mere elasticity brings the eyes back seems improbable from what we know of the related contraction of antagonists elsewhere. Moreover, any such action depends on muscular tone, and is absent in the atonic palsy of total nerve disease.

The centres are influenced by centripetal influences as well as by the will. They are influenced by visual impressions to a degree which we can only discern indirectly (since the process does enter into the region of consciousness) by considering how accurately, and yet how easily, we can follow with the eye a swiftly moving object.* The newly-born child never "fixes" an object, never follows a light, however brilliant. Not until later is there established the functional control of the motor centres by the visual impression. Hence, it is intelligible that if this visual control is partially, but not perfectly, established (in consequence of early ocular disease) the action of the motor centres should be abnormal. Hence, too, we can understand that imperfect ocular guidance (as the bad light of the safety-lamp) may aid other causes in developing derangement in later life. In albinism the early visual impressions may err on the side of painful and disturbing excess, from the absence of the pigment of the eye.

Again, the ocular motor centres must be acted on by, as they certainly act on, the equilibrial centres. These are guided by the innervation of the eyeball muscles. Thus, these muscles will be brought, indirectly, under the influence of the semicircular canals. Nystagmus can be induced in health by rotation of the body (Donders); quick movements of the eyes occur in the direction in which the body has rotated, and there is a slower return. Thus, the quick movements occur toward the side on which the horizontal semicircular canal has been subjected to increased pressure by the rotation. In chronic otitis, pressure on the ear has been observed to cause a nystagmus precisely similar, ceasing with the pressure.† In the nystagmus of miners, which may occur only in the recumbent posture, we may trace an influence of the equilibrial centre (and probably of the canal impressions) on the centre for the movement of the eyes.

The wide variation in the position of organic disease that may cause nystagmus is scarcely surprising when we consider how wide and various must be the total connections of the functions of vision, of movement of the eyes, and of the maintenance of equilibrium; and that we can trace a connection between nystagmus and the derangement of each of these. Hence, some forms of nystagmus seem to be closely connected with vertigo. The manner in which nystagmus is produced by such varied disease suggests that its immediate pathological mechanism must be some tendency inherent in the centres concerned. It is possible that these centres have a tendency to rhythmical or intermittent action, which is normally counteracted, and that the counteracting influence is readily deranged. The subject needs far more systematic study than it has yet received.

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* I recorded some years ago (*Brain*, vol. ii) a case in which the reflex fixation of the eyes was brought into salience by disease. If the patient, looking at one object, was told to look at another at some lateral distance from the first, his head was instantly turned in the direction of the second object, but the eyes remained fixed on the first by a movement as rapid as that of the head, but in the opposite direction, and then they were slowly moved into the position corresponding to the second object. The patient was in the last stage of progressive muscular atrophy.

† Schwalbach, Hughlings Jackson, Pflüger (attempt to extract a polypus). Movements are also related to true auditory impressions, at least in so far as rhythm is concerned, as musical "marches" illustrate. In peculiar hysteroid states, Hogyẽs has found that nystagmus could be produced by bringing a vibrating tuning fork near the ear, and that the movements varied with the rapidity of the vibrations. He was also able to cause nystagmus by other sensory impressions (*Orvosi-Hetilap, 1886,* and *Cent. f. Nervenh.*, p. 526).

The practical significance of nystagmus in diagnosis is extremely great, not from its localizing value, but because it shows the presence of more than merely functional disease. It is often marked at the early stage of degenerative disease, when other symptoms are equivocal.

Spasm of the levator is very rare. It is usually due to some irritation in the region of the fifth nerve. Late in life it may come on as an independent affection, analogous to other muscular spasms, as torticollis and facial spasm. The contraction is usually tonic, an excessive degree of the normal tonic contraction of the levator, and the muscle is imperfectly relaxed when the eye is directed downward or the lids are closed. Hence, the eyelid is a little higher than the other when the eyes are directed straight forward; and on looking down the lid does not descend, so that a wide extent of sclerotic is exposed above the cornea. When the lids are closed, the upper lid is brought down by the orbicularis, but not to the same extent as on the other side, and hence the lids are not brought quite together. The exposure of the sclerotic above the cornea gives the impression of slight prominence of the ball. A long-continued spasm in the levator seems, indeed, to cause the eyeball to be very slightly more prominent than the other, in consequence of the origin of the levator being below the level of the upper part of the eyeball; but the apparent prominence due to the exposure of the sclerotic is greater than the real prominence. The retraction of the upper lid may cause a slightly greater fullness of its tissues than exists on the other side. Unless the affection is due to removable irritation of the fifth nerve, it is extremely obstinate.

Clonic spasm is also rare. It occurred from time to time in the case of reflex ptosis mentioned on p. 621. A very singular case of congenital spasm of the levator was exhibited at the Ophthalmological Society by Mr. Marcus Gunn. There was slight ptosis and slight myosis on one side, and the eyelid was raised by slight contraction of the levator whenever the external pterygoid of the same side was put in action.*

Spasmodic elevation of the upper eyelid also occurs from irritation of the cervical sympathetic. We have seen that this nerve supplies the plain muscular fibres of the orbit, which are indirectly connected with the eyelids, and that their paralysis causes slight drooping of the lid. It is probable that their spasm is the cause of the increase in the normal elevation, and of the defective descent of the lid in looking down, in many cases of exophthalmic goitre. Similar spasm is said to have been met with in pregnancy, as a reflex symptom.

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* *Ophthalmological Transactions*, vol. iii, p. 283. As one of a committee appointed to report on the case, I had an opportunity of carefully examining it. The simplest explanation for the condition seemed to be that some of the levator fibres of the third nerve arose from the motor nucleus of the fifth nerve. The small size of the pupil might be due to the influence of those cells of the third nerve nucleus which ought to have been, but were not, connected with the levator.

Treatment.—Little can be done for the treatment of these ocular spasms beyond the removal of their cause, as far as this can be effected. Hock's second case was apparently cured by specific remedies, his first case by tenotomy. Hysterical spasm can generally be removed by a small blister to each temple, if the potent influence of neglect is ineffective. The paroxysmal epileptoid form of ocular spasm is very obstinate. Bromides have little influence upon it, and tonics do more good than sedatives. Counter-irritation sometimes seems to produce a beneficial effect.

In the treatment of nystagmus the improvement of vision, if this is defective, and the removal of any cause that can be discovered, are the chief measures. The application of a feeble voltaic current, from the mastoid process to the closed eyelids, was recommended by Svetlin, but has failed in the hands of others.

Spasm of the levator is a most intractable affection. Irritation of the fifth nerve should be sought for, and, if present, removed. When no cause can be traced, the most varied treatment by counter-irritation, sedatives, and electricity usually fails.

THE FIFTH NERVE.

The fifth nerve, it will be remembered, has an extensive deep origin, not only from the middle nucleus at the level of its surface attachment, but also by descending fibres from beneath the corpora quadrigemina, and by ascending fibres, from the medulla oblongata (see p. 487). The latter are connected with sensory nerve cells, forming a tract of gray matter continuous with that from which the posterior cervical roots arise; the cutaneous distribution of the fifth nerve is continuous, on the head and neck, with these cervical roots and the central gray matter similarly continuous. This enables us to understand the radiation of pain from one nerve region to the other. The nucleus of the motor root is at the level of the origin of the nerve from the pons. The Gasserian ganglion lies in a hollow on the petrous part of the temporal bone, and thence the three divisions pass from the cranial cavity, the first by the sphenoidal fissure to the orbit, and the others by the foramina ovale and rotunda of the sphenoid bone to the spheno-maxillary fossa. The *first part* supplies the skin of the forehead and anterior part of the hairy scalp, the upper eyelid, and the bridge and tip of the nose; the *second part*, the lower eyelid, cheek, anterior part of the temples, side of the nose, upper lip, upper teeth, and upper part of pharynx, tonsils, soft palate, and uvula and roof of mouth; the *third part* supplies the rest of the temple, the anterior and upper part of the ear, the auditory meatus, the lower part of the cheek adjacent to the mouth, the lower lip, chin, lower teeth and gums, and the tongue, part of the mucous membrane of the mouth, and the salivary glands. The function of taste in the anterior part of the tongue is subserved by the lingual branch of the third division, but the fibres pass from this to the facial nerve and thence to the spheno-palatine ganglion and the second division, as will be explained immediately. The motor part supplies the muscles of the lower jaw, temporal, masseter and pterygoid, the mylohyoid, and the posterior belly of the digastric.

The connections of the fifth nerve are numerous, and some of them are of considerable importance. The first part receives, at the Gasserian ganglion, fibres from the sympathetic, which pass with it to the eye, and are the fibres that innervate the radiating muscle of the iris. The second part gives off, from the spheno-palatine ganglion, the Vidian nerve, which (after a connection with the tympanic branch of the glosso-pharyngeal) joins the facial. It is called the large superficial petrosal, after it has given off a branch to the sympathetic. The lingual branch of the third part gives off the chorda tympani, which joins the facial in the Fallopian canal, a little below its junction with the large petrosal (Vidian). There is strong reason to believe that most of the fibres of the chorda tympani pass into the petrosal (Vidian), and thus reach the spheno-palatine ganglion and the second part of the fifth nerve. These fibres not only conduct taste impressions from the front of the tongue, but probably also subserve some tactile sensibility, since this may be lowered by disease of the chorda tympani (see note on p. 642). Lastly, the otic ganglion of the third part gives off the small superficial petrosal nerve, which is connected with the facial nerve where the Vidian joins it, and ends in the tympanic branch of the glosso-pharyngeal.

PARALYSIS OF THE FIFTH NERVE.

Causes.—The course of the fifth nerve renders it liable to damage from disease in various situations, but its deep position protects it from some influences to which nerve trunks of superficial course are obnoxious, such as rheumatic neuritis, which is as rare in the fifth as it is common in the facial nerve. The chief causes of damage are the following:—

(1) Disease within the pons, especially focal lesions, hemorrhage, softening, tumors, and sometimes an islet of sclerosis at the level of the origin of the nerve, damaging either the root fibres or the nuclei. The most considerable symptoms are produced by damage to the root fibres. The nuclear origin of the sensory fibres is so extensive that disease never affects more than a portion of it. Degenerative disease is rare; the motor nucleus usually escapes even in widespread nuclear degeneration.

(2) The nerve is damaged by disease at the base of the brain, especially by tumors, chronic meningitis, and caries of bone. It is liable to suffer in disease of either the posterior or middle fossa, or of the petrous bone between the two.

(3) Each division of the nerve has a course that exposes it to special lesions; the first, in the wall of the cavernous sinus, may be damaged by growths in the pituitary region, or aneurism of the internal carotid, and within the orbit it may suffer from growths or inflammation, such as orbital

cellulitis; the second and third pass into the spheno-maxillary fossa, which is often invaded by tumors from the parotid region and adjacent bones.

(4) Traumatic injury, especially punctured and bullet wounds, through the mouth and nose. On the other hand, the nerve is rarely damaged in fracture of the skull.

(5) While secondary neuritis is common, arising by extension from bone or membranes, primary neuritis is rare, and when it has been found has occupied the Gasserian ganglion. The peculiar neuritis that seems to be the cause of herpes zoster is frequent in this nerve, but must certainly be distinguished from the ordinary form of inflammation.

SYMPTOMS—(A) *Sensory Portion.*—The chief symptom of an affection of the fifth or of its branches is loss of sensation in the region of the skin supplied by it, universal in severe disease of the trunk of the nerve, or when all three branches are damaged by a growth in the middle fossa of the base, but in disease of the branches limited to areas supplied by them. The loss of sensation is commonly preceded by symptoms of irritation, sharp, darting, burning pains referred to the region of its distribution, closely resembling those of neuralgia, and often accompanied by tender points in the course of the nerves. There may be increased sensitiveness, especially to pain. The duration of the stage of simple irritation varies according to the quick or slow progress of the disease; sometimes it is absent, and the anæsthesia is the first symptom. Tactile sensibility is usually lost first, and there is often sensitiveness to pain, when a touch cannot be felt. Ultimately both are lost. The muscles of the face are insensitive, but are not weakened, although the movements of the face have been observed to be a little slower than normal, apparently from defective sensation. The mucous membranes, as well as the skin, become insensitive. The conjunctiva can be touched and even pricked without discomfort or reflex action. The mucous membrane of the nose can no longer be irritated by snuff or ammonia. Odors can at first be perceived perfectly; after a time the sense of smell is blunted, in consequence of the dryness of the mucous membrane and secondary changes in its epithelial tissues. The anæsthesia extends over the mucous membrane of the lips, mouth and tongue, up to the middle line. When the patient drinks, the cup, felt only on the unaffected side, may seem broken. Food is not chewed on the paralyzed side because it cannot be felt, or because the muscles of mastication are weakened. Hence, fur accumulates on the anæsthetic half of the tongue; this accumulation has been ascribed to defective innervation, but it is common in all conditions which lead to one-sided chewing, and is probably merely due to the fact that the food no longer removes the epithelium. In some cases, at least, the back of the tongue, the anterior arches and the palate, soft as well as hard, are insensitive. In other cases, in which the extent of the cutaneous anæsthesia suggests disease of the whole of the fifth nerve, the loss of sensibility is confined to the anterior two-thirds of the tongue. It is not known whether this difference is due to individual variations in the distribution of the nerve, or to differences in the seat of the disease, but it is certain that disease of the fifth *may* cause anæsthesia in the root of the tongue and palate.

Another frequent symptom of disease of the fifth nerve is loss of taste. Disease of the root of the fifth nerve may cause complete loss of taste in all the gustatory region of the one side, tongue and palate. The effect is not, however, invariable; probably the exceptions are cases of partial disease, or disease within the pons, where the taste path has a separate course. Disease of the lingual, after the chorda tympani has joined it, causes loss of taste in the anterior two-thirds of the tongue; above the junction with the chorda tympani disease of the third division seems to cause no loss of taste. The evidence of these facts is stated in the appended section on affections of taste.

Trophic changes occasionally result from disease of the fifth nerve. Alterations in the vascularity of the face have been described, but are certainly rare. The secretions from the mucous membranes and special glands, lachrymal and salivary, are lessened in paralysis, and increased for a time in irritation. Rarely, there is swelling and ulceration of the gums. An accidental bite of the insensitive cheek, for instance, heals slowly and tends to ulcerate. In long-standing cases the teeth have become loose. But the most important disturbance in nutrition is that of the eye. Inflammation of the eyeball is a common result of section of the nerve in animals, and has been frequently observed in man. The cornea becomes cloudy, then opaque, and ulcers form upon it, which may perforate and lead to a destructive inflammation of the globe. The conditions that determine this "neuro-paralytic ophthalmia" (as it has been termed) have been the subject of much discussion. It is not simply the result of interruption of the sensory fibres, since such interruption, with complete anæsthesia, has been repeatedly observed without any ocular disturbance. In a patient under my care, complete paralysis of the fifth nerve, motor and sensory, has existed for seven years without a trace of ophthalmia. This fact, and the absence of inflammation in facial palsy, show that it cannot be due simply to irritation of the conjunctiva by foreign bodies, dust, etc., which are not felt. From various experimental and clinical facts it seems probable that the inflammation is due, not to mere interruption, but to irritation of the fifth nerve. An unsuccessful attempt to divide the nerve, which caused considerable irritation but no anæsthesia, has caused characteristic inflammation of the eyeball (Meissner), and electrical irritation of the ganglion causes ocular inflammation, which, although transient, is intense. It is probable, moreover, that such irritation is most powerful when it involves the Gasserian ganglion (especially the inner part, according to Meissner) or the nerve fibres in front of the ganglion. It is frequently absent in disease of the nerve within the pons and of its root, at the surface of the pons, and although it does occur when the disease is in this situation, it is probable that a greater degree of irritation is required to produce it than when the disease is at, or in front of, the Gasserian ganglion.

Herpes zoster is frequent in the region supplied by the fifth nerve, especially in that of the first division. The observations of v. Bärensprung and others make it probable that the eruption is due to inflammation of the Gasserian ganglion, or of the nerve trunks in front of this, although it is doubtful whether the cause is an ordinary neuritis. It is usually preceded or followed by much pain and hyperæsthesia, sometimes accompanied also by lessened tactile sensibility—evidence of irritation and damage to the conducting nerve fibres. In the old, the pain that follows herpes is often peculiarly enduring, and may last for months or years. Herpes has been observed to follow other lesions of the fifth, but is not common in such cases. Catarrhal herpes of the lips has been attributed to neuritis of peripheral branches of the nerve, but without the pathological evidence that is desirable in the case of an eruption that differs so markedly from zoster in its irregular bilateral distribution and in its common cause.

(B) *Motor Portion.*—The resulting loss of power in the muscles can be best recognized by placing the finger on each masseter or temporal muscle, and making the patient bring the teeth forcibly together as in the act of biting. The feebleness or absence of contraction on the affected side is then evident. When the loss of power is slight, the affected muscles may contract a little later than the others. The paralysis of the external pterygoid causes two characteristic symptoms: the patient cannot move the jaw toward the unaffected side, and when the lower jaw is depressed it deviates toward the paralyzed side, because, in depression, the external pterygoids draw the condyles forward, and this movement occurs only on the unparalyzed side. The mylohyoid and posterior part of the digastric act only with other muscles that are not supplied by the fifth, and hence their paralysis does not perceptibly impair the movement of the hyoid bone, which they help to raise. Although the tensor palati and tensor tympani are supplied from the fifth, no evidence of the paralysis of these muscles has been observed in cases of disease of this nerve. It has been assumed that the palsy of the tensor tympani would cause defect in hearing low notes, but I am not aware that the defect has been detected, and I have failed to discover it in cases that I have examined with special reference to this point. It is probable that the fibres to the tensor palati, although they may come from the fifth, are ultimately derived from one of the bulbar nerves. After a time the paralyzed muscles of mastication waste, the temporal and zygomatic fossæ become flattened, and ultimately a little secondary shortening of the muscles may limit, in a slight degree, the downward movement of the jaw.

Diagnosis.—The diagnosis of paralysis of the fifth nerve is easy when its degree is considerable, when the motor part is affected, and when the sensory part suffers without anæsthesia elsewhere, or with only paralysis of other cranial nerves. A diagnostic difficulty arises only when there is other sensory paralysis, or when the disease of the fifth causes only sensory irritation, and the resulting pain is like that of neuralgia. In hemianæsthesia the parts supplied by the fifth nerve, skin, and mucous membranes are insensitive, but so also are the back of the head, trunk, and limbs, and in many cases the special senses are impaired. As long as the only symptom of disease of the nerve is pain, due to irritation of the fibres, and referred to their distribution, the condition may be indistinguishable from ordinary neuralgia. The pain may be of the same character and seat, most intense in the same localities, and sometimes accompanied by the same tender points. Persistent hyperæsthesia of the skin is more marked and extensive in cases of organic disease than in neuralgia, and the pain radiates less frequently to other nerve regions, *e. g.*, to that of the cervical plexus. In the absence of other evidence of organic disease, such as is afforded by the affection of other cranial nerves, the presence of such disease is certain if anæsthesia develops, corresponding in range to the fifth nerve or one of its branches. This, as proof of definite arrest of conduction, is unequivocal evidence of an organic lesion. Another important symptom of such disease is loss of taste, which, as we have seen, may occur independently of anæsthesia. It should be carefully sought for in every case in which an organic lesion is possible.

The diagnosis of the seat of the disease depends on the extent of the symptoms, especially of the anæsthesia, and on the associations of the paralysis. When all parts are affected, the disease is commonly at the base of the brain or at the Gasserian ganglion. A lesion of the first part only is usually at the sphenoidal fissure, or in the orbit. If the symptoms are limited to the distribution of the second part the disease is probably in the spheno-maxillary fissure or the superior maxillary bone. Such symptoms are occasionally the first indication of a tumor of this bone. The third part is rarely diseased alone. An affection of the second and third divisions without the first is generally due to disease of the sphenoid bone or in the spheno-maxillary fossa.

The nerves to the eyeball are those most frequently associated with the fifth in disease at the side of the pons and in the middle fossa of the skull. In the former case all parts of the fifth suffer, in the latter only the first division. At the side of the pons the sixth nerve is more frequently associated with the fifth than any other, and disease of the sixth, and of all parts of the fifth, points conclusively to this position. Paralysis of one fifth, and of the arm and leg on the opposite side, if of sudden onset, is due to a lesion within the pons; if of gradual onset it may be due to disease in or outside the pons. The associated paralysis of the conjugate movements of both eyes toward the side of the lesion is conclusive evidence of disease within the pons.

Treatment.—The most important element, as in other cranial nerve palsies, is the removal of the morbid process as far as this is practicable. When there is reason to suspect inflammation, a blister should be applied to the side of the occiput, or behind the ear, but not to the temple, lest it set up ulceration. If the affection has followed exposure to cold, hot fomentations may be applied to the side of the head and face during the first three days, but not after the fourth day, or vesication may ensue. The pain is often very troublesome. Sometimes gelsemium relieves it. Cocain injected locally, by arresting peripheral impressions, may lessen the pain of organic irritation. Often, however, only hypodermic injection of morphia gives relief. A weak voltaic current occasionally lessens pain that is moderate in severity, but it is powerless over the more intense suffering. When there is anæsthesia without pain, stimulation of the terminal sensory fibres may be tried.. If the continuity of the nerve fibres is interrupted such stimulation is necessarily powerless, but in many cases of partial and stationary or regressive disease the fibres are slow in regaining functional activity, and this may be distinctly increased by their peripheral stimulation, which tends to overcome the resistance at the diseased spot. The best means of effecting this stimulation is the faradisation of the skin. A dry electrode influences the cutaneous nerves more than a moist sponge, and the wire brush is the most effective. The brush should be stroked over the anæsthetic areas, and the other electrode, which may be a wet sponge, should be placed behind the ear or at the occiput. The current should be strong enough, if possible, to cause some sensation, and it should be applied for two or three minutes. Faradisation is much better, for this purpose, than the voltaic current, since the latter, if strong enough to be effective, is apt to cause giddiness. Electricity is far better than irritating liniments, which readily set up trophic disturbance. The skin may, with advantage, be gently rubbed, so as to increase its vascularity, before the application.

SPASM OF THE MUSCLES OF MASTICATION.

Spasm of the muscles supplied by the fifth nerve, the "masticatory spasm" of Romberg, may be either tonic or clonic.

Tonic spasm keeps the jaws together so that the two rows of teeth cannot be separated ("lockjaw") or can only be separated for a short distance, a quarter or half an inch. Sometimes the teeth are pressed together with considerable force. The masseters and temporals can be seen to be prominent, and felt to be hard. An attempt to depress the lower jaw, and overcome the rigidity by force, causes pain in the muscles, and sometimes the spasm is itself painful. The muscular contraction is almost always bilateral. It is a conspicuous and early symptom in tetanus, traumatic and idiopathic, and is an occasional and late symptom in severe cases of tetany. It occurs also in hysteria; sometimes frequent brief paroxysms last a quarter of an hour or more, sometimes a more enduring form succeeds a hysteroid fit and usually lasts until another convulsion, which leaves the patient free.* This spasm was unilateral in one hysterical case recorded by Travers.

In rare cases tonic spasm has been produced by sensory irritation elsewhere, generally due to injury, and has passed away when the irritation was removed. Romberg has recorded several instances of this. Unhappily, trismus, so produced, is, in most cases, the first symptom of general tetanus. This is true, also, of trismus following exposure to cold, but in one case paroxysmal and transient spasm of the jaw and tongue, accompanied by a "dying away of the extremities," was produced by any considerable exposure to cold (Romberg). In a few cases, prolonged trismus, without other symptoms, has followed similar exposure, but in most instances the patients were young women, and it is possible that the cases were hysterical in nature.

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* There can be little doubt that the case recorded by Romberg ("Dist. Nerv. Syst," *Syd. Soc. Trans*, vol. i, p. 305), of trismus after "epilepsy," was an example of this form.

Another occasional and rare cause is irritation in the sensory region of the fifth nerve, due to carious teeth, ulceration in the mouth, and other causes. The tonic spasm has been observed to coincide with paroxysms of pain, and to be removed for a time by pressure on tender points in the branches of the nerve. It is said to be produced especially by irritation of the last molar, either from caries with alveolar abscess, or during the eruption of the tooth. In the latter case the spasm has been known to continue for several months (Germain).

Lastly, tonic spasm is an occasional symptom of organic disease of the pons, due to irritating disease near, but not in, the motor nucleus of the fifth. The spasm may be unilateral, but it is more frequently bilateral (although sometimes greater in degree on one side) even when the disease is one-sided. It is often permanent. A tumor is the most frequent cause of the symptom. Of recorded cases, in one (Marot) there was a small tubercle at the junction of the pons and medulla on the right side; in another (Wernicke) the tumor, also a tubercle, occupied almost the whole vertical extent of the left half of the pons, and had caused loss of the movements of both eyes to the left, paralysis of the facial nerve, extreme tension of the left masseter, and numbness of the right side of the head. Somewhat similar symptoms in a woman, aged forty-two, under my care, were probably due to syphilitic disease of the basilar artery; they developed in the course of two days and then remained stationary. On account of the spasm of the muscles, which was bilateral, the jaws could only be separated for a quarter of an inch; in this movement the jaw deviated quite a quarter of an inch to the left, and hence there was probably some weakness of the left muscles, although the voluntary contraction seemed equal. The sensory parts of the fifth nerve were unaffected, but there was entire loss of all lateral movements of the eyes, and limitation of the vertical movements; nystagmus, lateral in the right and rotatory in the left eye, which was strongly inverted; complete paralysis of all parts of the left facial nerve with degenerative reaction, and right hemiplegia, considerable in degree. Three years after the onset the spasm remained the same and the other symptoms presented only trifling improvement.

Partial tonic spasm, affecting only some of the muscles supplied by the fifth nerve, is extremely rare. In the case of a girl suffering from hysteria and chorea, recorded by Leube,* the jaw was fixed for several days in the position of lateral deviation, and as there was no spasm in the masseters or temporals the symptom was apparently due to the spasm in the pterygoid muscles of one side.

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* *Arch. f. klin. Med.*, *1869*, vi, 273.

Clonic spasm of the muscles supplied by the fifth nerve is met with in two forms: (1) quick contractions frequently repeated; (2) single sudden contractions occurring at considerable intervals.

1. The serial clonic spasm is the most common. It causes, if considerable, successive upward movements of the lower jaw, bringing the teeth together, sometimes with sufficient force to cause the sound popularly known as "chattering of the teeth."* If slight, there may be only a tremulous movement of the jaw, although the spasm of the muscles may be felt when the fingers are placed on them. The movement is almost always vertical; a lateral movement, from spasm in the pterygoids, has been described, but is extremely rare. Clonic spasm is bilateral in most cases. It is a conspicuous feature of many forms of general clonic spasm, as convulsion and rigor. It occurs also in some cases of paralysis agitans; in this disease the muscles on one side may be affected before those on the other, and this is, perhaps, the only condition in which such contractions are one sided.

As an isolated symptom such clonic spasm is rare, and the few recorded cases have occurred late in life and in women. Romberg relates an instance in a women aged sixty-five, who had previously suffered from facial spasm. The spasm affected the right masseter more than the left, and not the temporals. It caused a constant chattering of the teeth. Toward evening the movement became very forcible and violent; it ceased during sleep and also during mastication. Pain was felt only when the spasm was very violent.† I have met with one case in a woman aged fifty-five, in which similar spasm was associated with neuralgic "shooting" pain in the left side of the face, especially round the orbit, and about the malar bone, darting through the head, and sometimes along the lower jaw and down the neck. The pain was most intense in wet weather. The spasm appeared secondary to the pain, and was greatest when the pain was severe. Both masseters and temporals were involved. The spasm was not quite regular, but the frequency of the contractions was 72 to 80 per minute. At times it could not be felt. It ceased when the jaws were opened widely. It was rather stronger on the left side than on the right, and in gradually passing away (after some months of tonic and sedative treatment) it ceased on the right side sooner than on the left.

2. The second form of clonic spasm, in which there are occasional severe single contractions, sometimes occurs in chorea, but as an isolated affection it is very rare. The jaws are brought together with considerable force, and the tongue or cheeks are sometimes bitten. An instance of this form occurred in a girl aged twenty-six. It commenced suddenly; at first, whenever she tried to eat or began to talk, and also on going to sleep at night, the jaw would be suddenly jerked up, and her tongue was several times badly bitten. She had no other symptom except slight weakness of the lower part of the face on the left side. She presented no evi-

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* Probably modified, by false analogy, from "clattering of the teeth."

† Romberg, "Diseases of the Nervous System," *Syd. Soc. Trans.*, *1853*, vol. i, p. 301.

dence of hysteria. A heavy weight had fallen on the head about three months before the onset. The symptoms continued, decreasing in severity, for about a month, but for six months she had an occasional attack, leaving tenderness in the masseters, and there was some reason to think that on one occasion in the night she had an epileptic fit. I have known similar spasms to occur during sleep, chiefly in men during middle life. It wakes the patient, and the tongue, if it happens to be between the jaws, may be bitten. It appears to depend on enfeeblement of the nervous system, and ceases when this is strengthened.

Diagnosis.—The existence of the spasm in the muscles of mastication is readily recognized. The only conditions which simulate spasm are: (1) interference with the movement of the jaw by a tumor or inflammatory swelling near the ramus, conditions sufficiently obtrusive; (2) disease of the joint, usually rheumatoid, limiting the movement of the articular surfaces; here there is no tension of the muscles, and other joints are commonly affected. In one case, for instance, that of a man, thirty years of age, in whom the lower jaw could not be depressed for more than half an inch, a similar condition had fixed the articulations of the cervical spine so that the neck was as rigid as if it had been composed of wood. The prognosis of isolated spasm is good, except in the cases which are due to organic disease of the nerve centres. These are usually permanent. In other cases the spasm usually passes away, but only after a somewhat prolonged treatment.

Treatment.—The treatment of the forms of spasm that are part of diseases of wider nature is necessarily that of the primary disease, and is described elsewhere. Hysterical spasm is sometimes removed by a blister behind the ramus of each jaw. In the isolated form, it is important to search for and remove all sources of irritation of the sensory nerves, to lessen pain by sedatives, and to strengthen the nervous system by tonics, especially iron and quinine. When a carious molar is the cause of the spasm it may be necessary to relax the muscles by chloroform, that the tooth may be extracted. If the spasm is apparently due to cold, a hot air or vapor bath should be used in the early stage. Voltaic electricity has been said to do good in some cases, the positive pole being placed on the back of the neck, the negative on the contracting muscles, and if no result follows, the position of the poles may be reversed; but it is doubtful whether the current can reach the over-acting centres. The paroxysmal form of clonic spasm is certainly lessened by bromides, and they usually prevent the occurrence of nocturnal spasm, but they have no influence on the tonic form. Cauterization beside the cervical spine has been recommended for trismus due to cold (Petrone). In prolonged cases of tonic spasm in which the jaws are completely closed, the feeding of the patient is a matter of difficulty, and the extraction of a tooth may even be needed for the purpose.

AFFECTIONS OF TASTE.

The sense of taste, it must be remembered, includes only the recognition of the qualities known as "bitter," "sweet," "sour," and "salt," with certain metallic sensations. It does not include what are called flavors; these, as we have seen, are really perceived through the olfactory nerve. Gustatory sensation is subserved by the mucous membrane of the tongue, palate, and palatine arches, but in the fore part of the tongue the function is chiefly localized in the tip and edges, and is very slight on the upper surface. Each quality can be perceived in all parts of the gustatory region, but bitterness and sweetness are more readily appreciated at the back of the tongue, sourness and saltness at the tip and edges. Moreover, if minute areas are tested, there are found to be spots in which one quality can be perceived and not another. There seem also to be individual differences in the relative power of detecting the various qualities in the several regions. The sense is also influenced by age, being greater in the young than in the old.

In testing taste, substances should be used that do not appeal to any other sense—colorless solutions or white powders. Care must be taken that their action is limited to the spot examined. If powders are used, a little time must be allowed for their solution by the moisture of the mucous membrane, and slight friction facilitates the stimulation. Salt, sugar, citric acid and quinine answer very well; in spite of the slight volubility of solid quinine, its intense bitterness renders it readily perceived. The voltaic current affords a very convenient means of examination, applied by two insulated wires twisted together, so that their exposed ends are a few millimetres apart. A current of one or two cells causes a metallic taste wherever the function is intact. More cells should not be used, because pain is then produced, and obscures the sensation. The advantage of this test is that it influences directly the nerve endings.

There is much difference of opinion as to the cranial nerve or nerves that subserve the sense of taste, and in connection with which its disorder should, therefore, be described. Hardly any question in physiology has been the subject of more discussion and of more experimental research. But, from its nature, the subject is one on which experiment can give no very certain sound, and has far less significance than observations on the effect of disease on man. It is impossible, here, to do more than to state the more salient facts of the latter class. Two separate questions are involved in the problem: first, the nerve roots by which the taste fibres pass to the brain, and secondly, the course of the fibres from the mucous membrane to those roots.

With regard to the first of these questions we have two important facts. First, taste has been abolished by disease of the root of the fifth nerve. There are many observations of loss of taste in the front of the tongue from this cause. One unequivocal case has been recorded by Erb, in which the fifth nerve in the middle fossa was involved in a mass of inflammatory connective tissue, and the nerves of the medulla were normal.* In most of these cases, the state of taste on the back of the tongue has not been noted. In no less than six cases in which there were symptoms of disease of the root of the fifth nerve, either outside or just within the pons, I have found taste abolished entirely on the corresponding side, not only on the front of the tongue, but also on the back of the tongue and on the arches of the palate.* On the other hand, cases have been recorded, and I have seen two, in which there were symptoms of disease of the fifth nerve, without loss of taste. It is probable that, in these cases, either disease of the root is partial and the taste fibres escape, or else that the disease is within the pons, and that the path of the fibres for taste quickly separates from that of cutaneous sensibility, and so may escape when the latter is damaged. The latter hypothesis receives strong support from a case lately under my care in which there were symptoms of a small tumor within the pons near the level of origin of the fifth. In addition to palsy of the conjugate lateral movement of the eyes to the right, there was paralysis of the muscles of mastication on that side, and entire loss of taste on the same side of the tongue and palate, without any impairment of cutaneous sensibility. This case proves that the path of taste lies near, and may be affected with the motor fibres or nucleus of the fifth, and without the fibres for cutaneous sensibility, and it indirectly shows the possibility of an affection of the latter without the path of taste. The second fact is that there is no recorded case in which the roots of the glosso-pharyngeal nerve have been diseased, and indeed no case in which there were symptoms of disease limited to the nerve roots of the medulla, and in which there was any loss of taste.† These two facts constitute strong evidence that, in man, the fibres for taste reach the brain by the roots of the fifth nerve.

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* Erb, *Neur. Cent., 1882*, pp. 73 and 149. In a previous careful review of the subject,

The peripheral path of the taste fibres is, in part at least, strangely circuitous. It does not admit of doubt that the fibres from the front of the tongue are contained in the chorda tympani, which passes from the facial nerve to the lingual branch of the fifth. Apart from the results of experiment, it is only thus that we can explain the loss of taste in the front of the tongue

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("Handb. der Krankh. der periph. Nerven," 2d. ed., 1876, p. 220) he had come to the same conclusion, that the balance of evidence is in favor of the passage of the taste fibres of the chorda tympani to the brain by the fifth nerve. Schiff has arrived at the same opinion from experiments on animals, and has summarized his conclusions in a lecture published in the *Revue Méd. de la Suisse Romande, 1887*, No. 1. Vulpian found the fibres of the chorda degenerated after section of the fifth nerve within the skull, and not after section of the facial (*Gaz. Méd. de Paris, 1878*, No. 19).

* One case, in which there had been loss of taste for several years, with palsy of the fifth, and afterward slight weakness of the sixth, but no affection of the facial, auditory or any other nerve, was published in the *Journal of Physiology*, vol. iii, 1881. Since then I have met with five other cases. In all the loss was proved by electrical examination as well as by the ordinary tests.

† In a case of fracture of the skull, and symptoms of injury to the nerves of the medulla, with loss of taste, recorded by Lehmann (*Pflüger's Archiv*, xxxii, p. 194), direct injury to the nerves passing through the petrous bone was the probable cause of the symptoms.

that occurs in rheumatic facial neuritis, and in disease of the middle ear.* Moreover, that these fibres run from the facial nerve toward the lingual, and are distributed to the tongue with the latter, is shown by the fact that section of the lingual for neuralgia, below its junction with the chorda tympani, has abolished taste.† But disease of the facial nerve within the skull does not impair taste ; hence, the fibres that reach the facial by the chorda tympani must leave the nerve again, and the opinion is probably correct which assumes that they pass from the geniculate ganglion of the facial nerve, by the Vidian nerve, to the spheno-palatine ganglion. Thus again reaching the fifth nerve, they seem to ascend in the second division to its root and the brain.‡

Regarding the course of the taste fibres from the posterior part of the tongue and palate, we have far fewer facts. Indeed, as regards man, the facts are practically limited to two : the loss from disease of the roots of the fifth nerve already mentioned, and the curious fact that taste is often lost on the back as well as on the front of the tongue in caries of the middle ear.§ The spheno-palatine ganglion supplies the mucous membrane of the palate and its arches. Caries of the middle ear can scarcely influence more than the nerves that run through the petrous bone. How far its effect is due to damage of the chorda tympani or to the tympanic plexus, through which the glosso-pharyngeal nerve is connected with the otic ganglion of the fifth, we do not know. It is possible that taste fibres may be distributed with the glosso-pharyngeal, and yet pass to the brain by the tympanic plexus of the fifth. On the other hand, we need more facts as to the backward extent of the loss of taste from disease of the chorda tympani. In all observations on taste it is desirable that the three regions, front of tongue, back of tongue, and palate, should be specially tested. Opportunities for observation of the highest importance sometimes occur to surgeons, and it is very desirable that they should be utilized.

The peripheral nerve lesions that may cause loss of taste have been just mentioned. It may also occur, as part of general hemianæsthesia, in disease of the cerebral hemisphere, and also, in the same association, in the functional disturbance of hysteria. When the loss is partial, the perception of some qualities may be more impaired than that of others, but there is never such complete loss of some perceptions without impairment of others as could be compared to color blindness. It is said to be sometimes due to local disease of the mucous membrane, but, on account of the wide area in which this function is subserved, the loss is probably never complete. The evidence of the loss is inability to perceive the qualities mentioned on p. 641. Its *treatment* is chiefly that of the disease causing it. If stimulation of the nerves is thought desirable, this can be readily effected by the voltaic current. One rheophore may be placed behind the mastoid process, and the other, a flat piece of metal, on the surface of the tongue. A tongue depressor, insulated where it comes in contact with the lips, answers very well.

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* Apart from the overwhelming evidence that has been accumulated, a crucial observation has been recorded by Urbantschitsch. In a case of disease of the membrana tympani and middle ear, causing loss of taste and some diminution of tactile sensibility in the front of the tongue on that side, chemical and mechanical stimulation of the chorda tympani produced sensations of taste and of touch on the part of the tongue in which the sensibility was impaired (*Archiv f. Ohrenheilk.*, xix, p. 135).

† Inzani, *Meissner's Jahresbericht, 1864*, p. 555 ; Lussana, *Arch. de Phys., 1871*, p. 152 ; Mader, *Cent. f. med. Wissensch., 1879*, p. 395.

‡ In a case in which there were the symptoms of a lesion of the third division high up, above its junction with the chorda tympani, there was no loss of taste (Erb, *Neur. Cent., 1882*, p. 104).

§ Urbantschitsch, "Beob. über Anomalien des Geschmacks in Erkrank. der Paukenhöhle," Stuttgart, 1876. I have several times satisfied myself of the occurrence of the loss of taste in these cases.

Perversion of the sense of taste, which has been termed *parageusia*, sometimes occurs in neurotic maladies, especially in hysteria and insanity. A substance gives rise to some other sensation than that which it ordinarily causes. The symptom is often associated with morbid dislike to certain tastes. *Increased sensitiveness*, *hypergeusia*, is said to be met with occasionally under the same conditions as perversion of the sense, and so also are *subjective sensations of taste*, usually of an unpleasant character. These are also met with as a result of irritating disease of the nerves, such as disease of the ear, and they have been experimentally produced by electrical stimulation of an exposed chorda tympani. Such sensations sometimes occur as the aura of an epileptic fit, and as part of the hallucinations of the insane. They must, of course, be distinguished from actual sensations due to abnormal buccal secretions, and from those due to the presence in the blood of substances that can stimulate the gustatory nerves. The various subjective sensations or perversions of taste scarcely ever call for special treatment.

FACIAL NERVE.

The facial nerve, *portio dura* of the seventh pair, has a tortuous course both within the pons and through the wall of the skull. Its deep origin has been described at p. 486. At the surface of the brain and within the internal auditory meatus the facial and auditory nerves lie together and suffer together in disease. From the auditory meatus the nerve has a winding course through the temporal bone, passing first outward to the inner wall of the tympanum, and then backward above the foramen ovale, to pass down behind the tympanum to the stylo-mastoid foramen. It lies throughout this course in the bony "Fallopian canal," but the thickness of the lamina which separates it from the cavity of the tympanum varies in different persons. Moreover, two nerves pass from it into the cavity of the tympanum, the small nerve to the stapedius muscle, and the chorda tympani, which leaves the facial a few millimetres above the lower opening of the canal and courses across the upper part of the tympanic cavity and membrane, to pass again through the bone and join the third division of the fifth. An arterial twig also passes from the canal into the tympanum. These connections between the canal and the tympanum are important, since the nerve is often damaged by disease of the middle ear. At its bend in the temporal bone is a gangliform enlargement, the "geniculate ganglion," from which the large petrosal nerve passes (as the Vidian) to the spheno-palatine ganglion; and, as already stated (p. 632), there is reason to believe that most of the fibres from the chorda tympani leave the facial by the petrosal nerve and reach the brain through the fifth. The gangliform enlargement also receives a twig from the nerve (small petrosal) which connects the otic ganglion, through the nerve of Jacobson, with the glosso-pharyngeal. Outside the skull the nerve gives branches to the occipital part of the occipito-frontalis, to the external ear, to the stylo-hyoid and digastric, and then divides, opposite the posterior edge of the masseter, into numerous branches which pass to all the muscles of the face, and to the platysma myoides beneath the skin of the neck.

The path from the facial nucleus to the cerebral hemisphere crosses the middle line of the pons above the nucleus, so that if the face is paralyzed by one-sided disease above the middle of the pons, the paralysis is on the side opposite to the lesion, as in ordinary hemiplegia. The further course of this path has been described at p. 470.

FACIAL PARALYSIS.

Paralysis of the face results from any interruption to the path from the cortex to the muscles. The character of the paralysis differs according as the disease involves, on the one hand, the path above the nucleus (which we may term *supra-nuclear palsy*) or, on the other hand, the nucleus itself or the fibres of the nerve, whether within the pons or outside it (*nuclear and infra-nuclear palsy*). In the latter case all parts of the face are affected, the orbicularis palpebrarum and frontal muscle, as well as the muscles of the mouth. In the former case the upper muscles of the face are little or not at all affected, and the muscles which go to the angle of the mouth suffer chiefly. Another difference is that in supra-nuclear paralysis voluntary movements are often more impaired than emotional movements; in nuclear and infra-nuclear disease they suffer equally (see pp. 510 and 513).

Lastly, there is an important difference between the two forms in the reaction of the nerve and muscles to electricity. In supra-nuclear disease the reaction is normal, or presents only a trifling change, which is the same to the voltaic and to the induced current. Acute lesions of the nucleus or nerve, unless trifling in degree, cause secondary degeneration of the fibres with loss of all faradaic irritability, and increase of the voltaic irritability of the muscles.

It is customary to term paralysis from disease of the nerve "peripheral;" that from disease of the nucleus or root fibres in the pons is also sometimes termed peripheral, because it resembles that from disease of the nerve; sometimes it is termed "central;" paralysis from disease of the path above the nucleus is also sometimes termed "central," sometimes "cerebral." The latter term should be used only with the understanding that a similar paralysis may occur from disease of the crus or upper part of the pons. Paralysis from disease of the convolutions is termed "cortical."

The supra-nuclear palsy has been already considered in the chapter on hemiplegia; we have now to consider only that which results from disease of the nucleus or the nerve fibres.*

CAUSES.—(1) Within the pons the nucleus or root fibres may be damaged by various focal lesions, usually in association with the motor tract of the opposite limbs, or with the sixth nerve of the same side, round the nucleus of which the facial fibres course (Fig. 208, p. 486). In very rare cases of anterior poliomyelitis (infantile paralysis), the facial nucleus suffers; I have seen one instance of this. Primary degeneration rarely affects all parts of the facial nuclei, but the nerve cells related to the orbicularis oris often degenerate with the connected cells for the hypoglossal nerve, in chronic "labio-glossal" paralysis. Diphtheritic palsy involves the face in rare cases, and probably consists in a subacute affection of the cells of the nucleus and the fibres of the nerve.

(2) At the base of the brain the nerve may be compressed by tumors or damaged by meningitis. The auditory nerve usually suffers with it.

(3) Ear disease is a common cause, especially in children. In most cases there is caries of the bone and suppuration in the middle ear,† and the disease of the bone between the Fallopian canal and the tympanum may spread to the nerve. This may be merely inflamed, or may be destroyed by the spreading disease. The amount of ear disease that exists before the facial nerve is affected varies much, and the readiness with which the nerve suffers doubtless depends in part on the thinness of the protecting lamina of bone. It is said that the nerve may be paralyzed when only the lining membrane of the tympanum is inflamed and the bone is not diseased. In such a case we must assume an extension of inflammation along the chorda tympani, the nerve to the stapedius, or the arterial twig already mentioned. It is, perhaps, by this mechanism that facial paralysis has sometimes followed sore throat—simple, or such as occurs in some acute diseases.‡ In one case under my care, the paralysis came on as an attack of simple tonsillitis was passing away, and it was followed by severe pain below the ear for two weeks; taste was affected.

(4) Injury. Fracture of the base of the skull often passes through the petrous bone, and the nerve may be torn or bruised. The nerve or some of its branches outside the skull may be divided by wounds about the ramus of the jaw, and especially in operations for the removal of tumors in the parotid region. A simple blow on this part may also damage the nerve. An angry schoolmaster struck a boy, with the corner of a book, just below the ear. Much pain followed about the ear and the side of the head; in a day

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* It is often termed "Bell's paralysis," after Sir Charles Bell, who first explained its nature.

† It is not, however, a frequent consequence of caries, although the calculation of only 1 per cent. made by Bezold is, perhaps, too low. (Bezold, "Labyrinth-Necrose," 1886.)

‡ Wendt, *Archiv der Heilkunde, 1870.*

or two facial paralysis came on, complete, and, as it turned out, permanent, in spite of all treatment. The blow cost the schoolmaster a thousand pounds. Facial paralysis is sometimes, although rarely, caused during instrumental delivery, by the pressure of the blades of the forceps on the nerve over the jaw, and such paralysis has even been produced on both sides.

(5) Neuritis is the cause of the common facial paralysis, due to cold, and often termed "rheumatic." The majority of cases of facial paralysis seem to be of this nature, but very often no special exposure to cold can be traced. Nevertheless, their characters are so uniform, allowance being made for differences in degree, that we are justified in regarding the pathological condition as the same in all. There is little doubt that this condition is a neuritis usually within the Fallopian canal.* The inflammation probably affects chiefly the sheath of the nerve, which swells and compresses the fibres, expansion outward being prevented by the rigid bone. The truth of the hypothesis has, indeed, never been demonstrated post-mortem. and for a long time a very different explanation was given. It was supposed that cold acts by paralyzing the terminations of the facial nerve in the muscles. But there is no evidence that cold ever paralyzes intra-muscular nerve endings, and we know that paralysis of all fibres of one nerve and of no other fibres, always means disease of the trunk of the nerve.

Although the anatomical proof of Fallopian neuritis as the cause of the common form of facial paralysis has not been furnished, that proof has been supplied in the case of a perfectly similar paralysis recorded by May.† The patient was suffering from leucocythæmia, and the nerve within the Fallopian canal presented a fusiform swelling three-tenths of an inch long, due to an infiltration of lymphoid cells, and the nerve fibres at the spot were in process of destruction.

In more than half the cases a special and considerable exposure to cold can be traced; generally a draught of cold air has blown on the side of the face and head, as in sitting in a railroad carriage opposite an open window. The symptoms usually commence within twenty-four hours of such exposure. In other cases there has been more or less constant exposure, as by working in a draughty shop. Occasionally the sufferer presents other evidence of rheumatism, especially of the fibrous tissues, and a liability to a similar affection of other nerves, as in cases mentioned on p. 571. But many of the subjects of facial palsy present no other evidence of rheumatism, and the affection may be an isolated event in an otherwise healthy life. In some adult cases there is a history of gout, which may have predisposed to the disease. It is possible that inherited gout has sometimes an influence in younger persons. The neuritis sometimes arises by extension from external cellulitis; in one case the affection appeared distinctly due to such cellulitis, produced by chronic eczema of the skin in front of the ear, over the place of emergence of the nerve.*

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* It has been conjectured that the inflammation sometimes affects the nerve after its emergence, but no evidence of this (*e. g.*, swelling of the nerve) has been noted.

† E. May, *Aerztl. Intell. Blatt.*, *1884*, No. 31.

Grouping together all cases which may be due to Fallopian neuritis (excluding those that are the result of ear disease or syphilis) they appear to be more common in males than in females, no doubt from the greater exposure of the former. Of eighty cases of which I have notes, 50 were in males, 30 in females. The affection may occur at any age, but is rare under ten. Only two cases occurred in this period, and each was in the second year of life. It is most common between twenty and thirty (19 cases), and is equally frequent in each of the other decades between twenty and fifty; after fifty it becomes rare, but is occasionally met with even in old age (one case at 74). It occurs at all seasons, but is rather more frequent in winter than in summer.

(5) Syphilis is an occasional cause, but its frequency may readily be overestimated if the mere fact of preceding syphilis or of improvement under iodide of potassium, is admitted alone to be evidence of nature. We are only justified in regarding the paralysis as syphilitic when there is or has been other evidence of active syphilis, and when there is no other recognizable cause; the syphilitic nature of the case is more probable if the nerve is affected within the cranium. In many cases of facial paralysis in the subjects of syphilis the paralysis is distinctly excited by cold, and it is probable that the syphilis may have had a predisposing influence. The mechanisms by which syphilis causes facial paralysis are by meningitis, by a gumma on the nerve sheath or near it, and probably also by an interstitial neuritis.

(6) Hemorrhage into the nerve sheath or Fallopian canal is a rare cause. Fracture of the skull has been thought to cause facial paralysis by this mechanism, but the nerve can scarcely escape direct damage from an injury causing hemorrhage. There are cases in which facial paralysis comes on in a few minutes and is at once complete, and in which there is no indication of any central disease. Such cases, of which I have seen two, can hardly be otherwise explained than by the assumption of a hemorrhage into the Fallopian canal, at once arresting, by its pressure, the conducting power of the fibres. This is not altogether hypothetical; hemorrhage into the canal has been found after death.†

(7) Facial paralysis has been observed in rare cases of spinal disease, especially locomotor ataxy. Nothing is known of the mechanism by which it is produced. It is probably sometimes a coincident effect of syphilis. The facial nerve may also be paralyzed by an islet of disseminated sclerosis within the pons.

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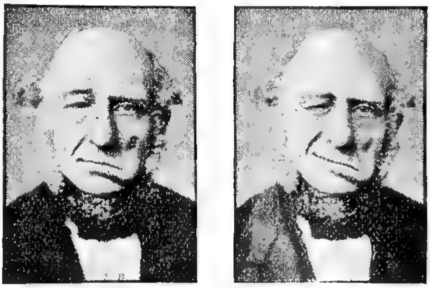
* It has been thought by some that cold affects the facial nerve by causing a catarrhal inflammation of the middle ear, which spreads to the nerve. But this seems improbable when we consider how frequent catarrh of the middle ear is without facial paralysis, and how rare are obtrusive signs of tympanic inflammation when the nerve is affected, and that slight inflammation of the tympanum may well be produced coincidently with neuritis by cold without the former being the cause of the latter.

† Wilks and Moxon, "Path. Anat.," p. 257, and Moxon, *Path. Trans.*, vol. xx.

Double facial paralysis is rare. Its causes are: (1) Disease of the pons Varolii; possibly a lesion in the middle of the pons, where the two facial paths cross; certainly lesions on both sides of the pons, such as the bilateral softening that is produced by disease of the basilar artery. (2) Disease, syphilitic or other, of the nerves at the base of the brain; by this usually other nerves are also damaged. In one case of this kind, in which there was also simultaneous paralysis of both auditory nerves (not aural), the onset occurred after exposure during sleep to a cold wind, but the patient had had syphilis. (3) Simultaneous double otitis.* (4) Diphtheritic paralysis. (5) Partial paralysis (of the lips on both sides) occurs from nuclear degeneration in labio-glossal paralysis, or symmetrical cortical lesions. Fallopian neuritis scarcely ever occurs on both sides simultaneously, although I have more than once known a second attack on the other side to occur within a few months of the first.

SYMPTOMS.—In complete facial palsy the muscles of the affected half of the face become toneless and immobile. In all movements, voluntary or emotional, the affected half of the face is still. The two sides of the face present a strange incongruity, and the smile or frown, deprived of half its range, loses more than half its character, so that it is difficult to recognize the expressional significance of the distorting contractions of cheek and brow which occur on the unaffected side. The influence of the loss of tone on the position of the features at rest differs according to the age of the patient; the smooth features of youth are largely moulded by the elasticity of the skin, and are little changed by the paralysis of the muscles; slight drooping of the angle of the mouth may be the only sign of paralysis during rest. But it is otherwise when time has scored the face with furrows. These are due to the loss of elasticity of the skin, and their position is chiefly determined by the muscular tension that is caused by and expresses the dominant emotion. With the loss of this tension the furrows change their place or vanish, as the flaccid skin adjusts itself in obedience to other influences (Fig. 271). The effect of the age-changes is seen conspicuously in the forehead and lower eyelid. The transverse furrows of the forehead cease suddenly at the middle line. In the young the lower eyelid is held by the elasticity of the skin almost as close to the eyeball as on the unaffected side. In the old it falls forward, and the tears, increased by the irritation of the unprotected eye, cannot reach their proper duct,

FIG. 271.



Facial paralysis in a man, aged sixty-five, showing the influence of the loss of tone on the inelastic skin. The figure on the right represents an attempt to close both eyes. (*From photographs.*)

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* Wright, *Brit. Med. Journ.*, Feb. 27th, 1869. A week separated the onset of the palsy on the two sides. The patient was a man aged sixty-five.

and the eye "waters." But in young and old the loss of movement is the same. The affected side takes no part in frown or smile. The eyelids cannot be brought together; in a strong effort to close them the eyeball is rolled upward, so that the cornea is beneath the upper lid, and only the sclerotic is visible. The patient often imagines, ostrich-like, that his eye is shut, because the cornea is covered. During sleep the eye remains open. The upper lid follows, in a normal manner, the movement of the eyeball, since the orbicularis takes no part in this descent. In the old the lower lid, on looking up, does not rise so well as on the other side, apparently because the lid is not kept in such apposition to the globe as is needful for the latter to raise it. In smiling and other movements, the mouth is drawn toward the unaffected side, the zygomatic muscles being unopposed by their fellows. From the displacement of the mouth the tongue is protruded to one side of the orifice, and thus may seem to deviate even when it is exactly in the middle line of the face. The lips cannot be pressed together on the paralyzed side, and hence the air cannot be so compressed within the mouth as to be expelled in a puff. The patient accordingly finds that he cannot blow out a candle. In drinking, the liquid runs out of the paralyzed corner of the mouth, and the patient has to incline his head to prevent this. Whistling is, of course, impossible, and smokers may first discover the paralysis by finding that the ejected saliva takes an inconveniently erratic course. Speech is frequently a little changed by the imperfect articulation of the labial consonants. The palsy of the buccinator permits food to get between the teeth and the jaws, and it is found more convenient to chew upon the other side. The palsy of the stylo-hyoid and posterior belly of the digastric does not cause recognizable symptoms. The platysma myoides is paralyzed, as may be observed by making the patient depress his lower lip, an action in which the platysma contracts. The dilator naris being inactive, the nostril does not expand on sniffing, and may even yield to the pressure of the air; smell, in consequence, is less acute than on the other side. The muscles of the external ear are also paralyzed in those persons in whom these muscles were under the influence of the will. All reflex movements are of necessity lost when the paralysis is due to disease of the nerve fibres or their nucleus, an important distinction from cerebral palsy, in which they are preserved.

Palate.—It is said that the palate is sometimes paralyzed on the same side as the face from disease of the facial nerve, and it has been supposed therefore that the levator palati is supplied by fibres which pass from the geniculate ganglion of the facial, by the large petrosal nerve and Vidian, to the spheno-palatine ganglion. The indications of the paralysis of the palate are said to be a lower position on the affected side than on the other, an inclination of the uvula toward the sound side, and deviation of the palate on movement. My own observations have led me to entertain considerable doubt as to the occurrence of paralysis of the palate from disease limited to the facial nerve, in any part of its course. I have noted the condition of the palate in more than a hundred cases of facial paralysis, due to disease of the nerve in various situations, and found in only one of them evidence of paralysis, and this was on the side opposite to the palsy of the face. Deviation of the uvula was occasionally observed, but its inclination was as frequently from as toward the paralyzed side. This has been noted by others, who have expended considerable ingenuity in explaining the difference of position on the hypothesis of unilateral palsy. But obliquity of the uvula is not uncommon in those who present no facial palsy, and sometimes is considerable in degree. I have seen the uvula like the quadrant of a circle in a patient who was suffering from no nervous disease. That this is the true explanation of the obliquity observed in most cases of facial paralysis seems certain from the fact that (with one exception, to be referred to presently) the two sides of the uvula, on movement, contracted equally, and the obliquity at once disappeared, even in the cases in which it was most marked. This fact absolutely excludes paralysis as a cause of the deviation. A difference in the height of the arches of the palate was observed in a few cases, but that on the side of the palsy was the higher in some cases, the lower in others; in no case was there a difference of movement, and in every case the difference in height persisted unchanged after the facial nerve had recovered. The exception to the equality of the movement of the uvula was a case of facial paralysis from cold, in which the deviation of the uvula toward the unaffected side was increased on movement, but other symptoms rendered the case complex. I think that these facts justify the conclusion that the paralysis of the palate is a much rarer accompaniment of facial palsy than is commonly assumed, and that further observations are necessary to determine its significance.* The best established fact regarding paralysis of the palate is that it is produced by disease at the side of the medulla, damaging the hypoglossal and spinal accessory nerves.

Taste.—In disease of the facial nerve between the origin of the chorda tympani and the geniculate ganglion, taste is lost in the anterior part of the tongue on the affected side. It is not lost when the lesion is in the root of the facial nerve,† or of the nerve fibres within the pons. It is lost in about half the cases of paralysis from cold, doubtless because the neuritis extends in the canal to the origin of the chorda. When the nerve is damaged outside the skull, taste is generally unaffected; the exceptions are probably due to an ascending neuritis. The affection of taste may persist after the paralysis, or pass away before it. Sensation in the face is unaffected, but I have several times found an area of anæsthesia on the front and back of the concha, in the region of skin supplied by a nerve given off by the facial as it emerges, and which is probably derived from the fifth nerve.

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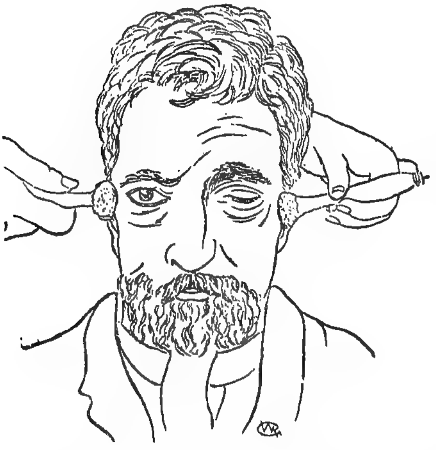
* Dr. Hughlings Jackson has also stated that he has never seen paralysis of the palate due to disease of the facial nerve (*Lancet*, April 2d, 1887). He made this remark in connection with an instructive case of paralysis of the face and palate, which at first sight seemed to prove the association, but further investigation showed conclusively that the palsy of the face was of cerebral origin.

† Certain recorded cases of disease of the root and loss of taste, opposed as they are to the usual rule, are explicable on the assumption that the descending degeneration was attended, as it sometimes is, by descending neuritis, and that this invaded the taste fibres at or below the geniculate ganglion.

Hearing is often impaired in cases of facial paralysis. In most cases the impairment is due to preceding ear disease, which has caused the affection of the facial nerve. Less commonly the two symptoms are the effect of the same cause; the facial and auditory nerves may be affected together by basal disease, or the cold that produces a Fallopian neuritis may also set up a catarrh of the middle ear. Tinnitus often attends the onset of facial palsy from cold. In consequence of the palsy of the stapedius, the tensor tympani, being unopposed, often increases the sensitiveness of the ear to musical notes, especially to those of low tone, and the auditory hyperæsthesia may be distressing. A subjective high-pitched sound has been referred to the same cause.

The electrical reactions in facial paralysis are those characteristic of peripheral palsies—the degenerative reaction fully described on page 66. The charts there given are from cases of disease of this nerve. In a severe case the nerve rapidly loses its irritability to faradaic and voltaic stimulation, and this may be extinct in the course of ten days or a fortnight. In slighter cases the irritability of the nerve is lowered, but not lost; a strong current still excites it. In most cases the fall in irritability does not commence for three or four days after the onset of the paralysis, and in slight cases may be delayed for a week or ten days. During the first few days an increase of irritability may often be found, preceding the diminution. In extremely slight disease of the nerve, I have several times found an increase corresponding to that which precedes the fall in severe cases, but followed by no diminution. This initial increase is usually the same to both forms of electricity, but it is often more marked to the isolated faradaic shock than to the serial current.* In the muscle the initial increase may also be recognized, and depends on changes in the intramuscular nerve fibres. This increase is followed by a gradual fall in the faradaic irritability of the muscles, corresponding in degree to that of the nerve, and due to the fact that the faradaism only stimulates the muscles through the intramuscular nerve endings. With this loss of faradaic irritability, that to the voltaic current is preserved and increased; to it the

FIG. 272.



Right facial paralysis, due to ear disease in childhood; simultaneous faradisation of both facial nerves. (*From a photograph.*)

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* It is said that in the slightest cases there may be no change in irritability, and the statement is doubtless true. But in the cases that I have seen in which the irritability to both currents was normal, I have seldom failed to find a distinct increase of irritability to the isolated induction shock.

muscle fibres themselves respond, and respond with undue readiness, so that they contract with a weaker current than is required by the muscles of the unaffected side. The form of response to increasing currents is often (but by no means always) changed, and instead of 1 KCC;[*] 2 ACC, AOC; 3 KOC, we may have such order as the following, which I take from my notes of actual cases. 1 KCC = ACC; 2 AOC; 3 KOC, or 1 ACC; 2 KCC, AOC; 3 KOC, or 1 KCC, AOC; 2 ACC; 3 KOC. A continuous contraction (tetanus) is also readily produced during the passage of the current, as in 1 KCC; 2 ACC = KC Tet.; 3 AC Tet.

With the recovery of the nerve the irritability of nerve and muscle slowly returns to the normal, but the nerve fibres often recover some conducting power (*i. e.*, the paralysis lessens) before they regain their local excitability. For a long time this remains below the normal. The muscles lose their excessive degree of irritability before the order of response becomes normal.

In rare cases, the reaction in nerve and muscles is that of the mixed form ("middle form." Erb), in which, with only slight diminution in the irritability of the nerve, the muscles present the increased and qualitative change characteristic of degeneration. The varieties in the course of the reactions are illustrated in the charts on pp. 68–70 (Figs. 33–38).

The electrical reaction presented by one muscle, the orbicularis oris, merits special attention. The muscle sometimes illustrates in a striking way the increased but slow muscular response to the voltaic current which results from nerve degeneration. The voltaic current diffuses itself widely; applied to the angle of the mouth on the unaffected side the closure of the circuit causes a quick, sharp contraction in the normal half of the orbicularis, distinctly *followed* by a slower contraction on the paralyzed side. If the strength of the current be then reduced until it is too small to cause contraction in the normal muscle, it may still cause the sluggish contraction on the opposite, paralyzed, side. This has been mistaken for a reflex contraction; it is no doubt the result of the diffusion of the current.

In cases in which the paralysis of the face remains absolute, the paralyzed half of the orbicularis usually regains a little power. This is doubtless due to the fact that, the muscle being circular, the two halves are continuous, and the nerve influence from one side extends beyond the middle line. It is indeed probable that the nerves of the two sides join, and some regeneration from the sound side may be possible. I have occasionally found, in old-standing cases, otherwise complete, that faradaism applied to either angle of the mouth caused contraction of the whole of the orbicularis, a fact difficult to explain on any other hypothesis than that of nerve regeneration from the healthy side.

Muscular wasting follows the nerve degeneration in the face as elsewhere, and in those who have but little subcutaneous fat the loss of tissue may be perceptible. But in most cases the atrophy is not revealed by any change in the contour of the face, because the thin muscles constitute but a small part of the subcutaneous tissues, and the nutrition of these is unaffected. True facial hemiatrophy never results from disease of the facial nerve.

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[*] KCC, kathodal closure contraction; KOC, kathodal opening contraction; Tet., tetanic contraction (see p. 67).

The loss of the protective movements of the eyelids exposes the conjunctiva to various irritant influences, but inflammation rarely results. The increased secretion of tears, although a source of annoyance to the patient, doubtless supplements the defective action of the lids in removing foreign particles. In the old there is occasionally a slight conjunctivitis of the lower lid, induced by the exposure which is the consequence of its recession from the eyeball (see p. 649). I have once known a small ulcer to form on the cornea, but it quickly healed.

The characters of the facial palsy are not influenced by either the seat or character of the disease of the nerve. In partial lesions some fibres may suffer more than others, with a corresponding variation in the relative degree of paralysis in the several parts of the face. But paralysis due to disease of the nucleus presents an important difference from that which is produced by disease of the nerve trunk. The fibres for the orbicularis oris are dissociated from the other facial fibres in their nuclear origin, and are connected with the fibres for the tongue; the two suffer together in nuclear degeneration (labio-glossal paralysis). On the other hand, in the case of facial palsy from nuclear inflammation (part of infantile paralysis), the orbicularis oris had escaped entirely, although the rest of the face was absolutely paralyzed.*

Certain concomitant symptoms occasionally attend the onset of facial paralysis, and are due, for the most part, to the cause of the nerve lesion. In neuritic cases, pain about the ear is very common, and may be felt for several days before and after the onset. It sometimes extends over the whole of the corresponding side of the head. A little swelling in front of the ear may often be found, if looked for, in the early stage; it is sometimes considerable and extensive. Auditory symptoms have been already mentioned. In one case the patient complained, at the onset of the paralysis, of a phosphorus-like taste in the back of the tongue, explicable, on the hypothesis stated at p. 649, by irritation of the nerve of Jacobson or the tympanic plexus. Slight giddiness not unfrequently attends the onset, and may be due to a disturbance of the labyrinth; it is not due to paralysis of the stapedius. Sometimes the giddiness is intense, especially when there is disease within the skull, probably from injury to the nerve fibres from the semicircular canals, or to the middle peduncle of the cerebellum. In some cases there are the signs of more wide-spread disturbance, nervous or general, from the exposure to cold that causes the paralysis; besides severe neuralgia, I have met with transient dilatation of the pupil and derangement of the movements of the eyeball on the same side, general febrile disturbance, and transient albuminuria.

Course.—The onset of facial paralysis is usually rapid but rarely sudden.

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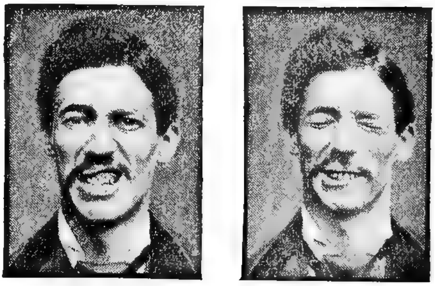
* The connection between the fibres for the orbicularis oris and the tongue is very close (see note on p. 487).

The palsy commonly reaches its height in from four hours to two days. Sometimes the affection comes on during the night.

Its duration varies according to the degree and character of the nerve lesion. This may be severe from any cause, but the most trifling as well as the most severe cases are met with from cold and ear disease, especially from the former. In the slightest form the paralysis may last only for a week or ten days. Often the affection lasts for several months. Two or three months is the average duration in cases of moderate severity. Those of greater degree last for six or eight months, and it is very common for the ultimate recovery to be incomplete. Indeed, whenever there is nerve degeneration, some trace of the paralysis usually remains, sometimes to be seen only on close scrutiny. In rare cases no recovery takes place; the paralysis remains absolute.

Secondary Over-action.—In all severe cases, some muscular contracture comes on, usually as voluntary power returns. When the palsy remains absolute, the contracture is absent; the muscles remain toneless. The first trace of contracture usually manifests itself about four or six months after the onset of the paralysis; it slowly increases for eight or twelve months, and then remains stationary or lessens. It is most marked, at rest, in the zygomatic muscles, and reproduces the lost naso-labial wrinkle, often in deeper degree than on the other side, so that, on looking at the face, the first impression is that the healthy side is the weaker. The impression may seem to be confirmed by a slight movement of the face, for this is more marked on the affected side than on the other.* But if the movement is considerable, the side that moved first is seen to move much less than the other. With this contracture is constantly combined a tendency to associated over-action. A smile is, in health, accompanied by a slight contraction in the orbicularis palpebrarum, and this contraction is increased on the affected side, so that the eye may almost close. Conversely, when the eyes are closed by a strong contraction of the orbicularis, the zygomatici on the affected side over-act and draw outward the angle of the mouth (Fig. 273). The contractions may also be readily excited by reflex action through the fifth nerve. In many cases another symptom is added after a time—spontaneous twitchings, isolated spasmodic contractions, recur-

Fig. 273.



Old paralysis of the right side of the face, late contracture and associated over-action. The figure on the left shows an attempt to raise the upper lip; that on the right closure of the eyes. In the latter it is seen that while the patient's right eye is closed less firmly than his left, the naso-labial furrow is rendered much deeper on that side by the associated over-action of the zygomatici. (*From photographs.*)

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* It is possible that there is not, as there seems, an undue initial movement, but that the previous contracture renders the effect of an equal voluntary contraction more conspicuous on the affected side.

ring at irregular intervals, and affecting chiefly the zygomatici. These various spasmodic symptoms are often considerable when the amount of regained power is slight.

The effect of the muscular contracture on the aspect of the face at rest varies according to the age of the patient, in a manner opposite to the effect of the palsy. In the old, it reproduces the lost naso-labial furrow, and restores the symmetry to the lower face. In the young, it develops a furrow that has no counterpart on the other side, and so adds a deformity at rest to the distortion during movement, and is a source of annoyance scarcely less than the original palsy. The condition is very persistent. When slight, and succeeding a trifling paralysis, it may pass away in time, but when considerable, and after a severe paralysis, it usually lasts unchanged for years, and although it may subsequently lessen, it rarely passes away entirely. It has been attributed to the use of electricity in the treatment of the palsy; faradaism and voltaism have both been blamed, but the contracture and over-action develop in as marked degree in patients who have not had electrical treatment as in those who have.

Second attacks of facial paralysis are rare. I have notes of only five instances. In two the second attack was on the same side. In each the first attack was slight. The second attack in one case was four years, in the other, two months after the first. In the latter case it was distinctly excited by a fresh exposure to cold. In three other cases the second attack was on the other side of the face. I have also twice known a slight facial paralysis, which was improving, to relapse.

PATHOLOGY.—The evidence of the nature of these cases has been already stated, and the minute changes in the nerves in such disease have been previously described.

The exact cause of the late over-action and spasm is uncertain. The contracture always coincides with some recovery of power; in a case of absolute and lasting paralysis there is no contracture. Since the associated over-action and the clonic spasm accompany the contracture, it is reasonable to refer all to the same cause. It is, on the whole, probable that they are due to changes in the facial nucleus induced by the long-continued interruption of the nerve, and constant stimulation of the centre by efforts to move the face. The resistance between the cells becomes lessened, so that they act with undue readiness on each other, and on the fibres. Their tonic influence on the muscles is increased, the action of one part of the nucleus spreads too widely through it, and the cells are liable to spontaneous discharge.*

DIAGNOSIS.—The existence of facial paralysis is only too conspicuous. When the face has partly recovered, but is the seat of late contracture, it is easy to mistake the side affected, unless attention is paid to the strength of contraction, and the significance of the associated over-action is remembered. A mistake is sometimes facilitated by an impression which patients are apt to have, that the smooth, unwrinkled half of the face is natural, and that the side which is distorted by movement is unnatural. No reliance can be placed on the statements made by patients or their friends as to the side affected.

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* This hypothesis is essentially the same as that put forward by Jacobi ("Inaug. Diss.," Marburg, 1877) in regard to a case of similar spasm after palsy of the nerves of the arm. Hitzig has suggested that the disease of the nerve leads to a state of "irritation" in the centre.

The distinction of paralysis due to disease of the nerve fibres from that due to disease of the motor tract from the hemisphere (infra-nuclear and supra-nuclear palsy) rests especially on the distribution of the paralysis, as stated on p. 513. The most important guide is the condition of the eyelids. If there is such persistent paralysis of the eyelids that they cannot be perfectly closed, the lesion is probably in the nucleus or nerve fibres. Cortical disease may, for a few days, paralyze the orbicularis, but this condition is distinguished by another important indication—that an emotional smile is less impaired by cerebral disease than is voluntary movement; in disease of the nerve the two are equally affected. In the latter, reflex action is lost, in the former it is unchanged.

In nuclear and infra-nuclear disease the reaction of degeneration is found in the nerves and muscles; in lesions of the facial path above the nucleus the reaction is normal or nearly so. Thus the degenerative reaction is proof of a lesion in the nucleus or nerve, but a normal or nearly normal reaction does not prove that the disease is above the nucleus, since it occurs also in very slight lesions of the nerve.

The further diagnosis of the seat of the lesion in the nerve rests on the associations of the paralysis. If no nerve except the facial is affected, and taste is unimpaired, the lesion is probably either outside the skull or in the lowest part of the Fallopian canal. If taste is lost on the front of the tongue, the lesion is in the canal, and involves the nerve between the geniculate ganglion and the origin of the chorda tympani. It does not, however, prove that the disease began in this part; disease may spread to the chordal region. Paralysis of the palate is generally thought to show disease of the geniculate ganglion or nerve above it, but this indication needs further investigation. Slight deafness is of little localizing value; complete deafness, without ear disease, coming on at the same time as the paralysis of the face, indicates disease in the internal auditory meatus or at the base of the brain, commonly the latter, since disease within the meatus is extremely rare. Paralysis of the facial and sixth, without the auditory, is probably due to disease in the posterior part of the pons, where the facial fibres curve round the nucleus of the sixth nerve. It is possible, however, as I have seen, for the sixth nerve to be paralyzed by a simultaneous rheumatic neuritis. If the orbicularis oris is quite unaffected, and all other parts of the nerve are paralyzed, the facial nucleus is diseased; if the orbicularis only is paralyzed and the rest of the face is free, the disease is in the neighborhood of the hypoglossal nucleus, and the tongue is almost certain also to be involved.

PROGNOSIS.—The cause of the paralysis is of less prognostic significance than are its characters. The chief etiological indication is that the prognosis is good in *pure* syphilitic cases, in the paralysis from pressure during birth, and (if the patient lives) in paralysis after diphtheria. It is said to be better in ear disease if the tympanum is not perforated (Dalby), probably because perforation is almost invariable when there is bone disease. But in ear disease and all other causes, cases of slight and severe character are met with, and the prognosis must be mainly founded on the electrical reaction. It is true that signs of improvement, and its rate, may enable an estimate to be formed of the likelihood of recovery, and of the probable duration of the affection. But if, as is often the case, there is no recovery of power when we have to form an opinion, we should have no prognostic guide were it not for the information electricity affords us. By its means we can ascertain the state of function of the nerve fibres and infer the state of their nutrition, and thus we can gain more information than even the microscope could supply. If at the end of ten days the irritability of the nerve is not below the normal, the face will probably be well in a few weeks. If, on the other hand, at the end of a fortnight the irritability of the nerve is absolutely lost, the paralysis will certainly last for several months. If, at the end of a fortnight, the nerve irritability, although lowered, is not lost, recovery will probably occur in about two months. Between these forms, intermediate gradations of severity occur, for which the prognosis must be correspondingly modified. When the faradaic irritability has been absent for some weeks, any return of excitability in the nerve fibres indicates a speedy return of some power in them.

TREATMENT.—The first element in treatment is to arrest and remove, as far as possible, the morbid process that is damaging the nerve. If there is even a possibility that this may be syphilitic, iodide of potassium should be given, and if there is no speedy improvement, mercury should be added. If there is ear disease, free exit for any discharge should be secured. When exposure to cold is the apparent cause, or in similar cases in which no cause can be traced, if the paralysis has existed for a few days only, fomentations should be applied to the region in front of and below the ear, as hot as can be borne, for half an hour, and repeated every three hours for two or three days. In all cases, except those of very trifling degree, a blister should be applied behind the ear, over the mastoid process, if the nerve is diseased in the canal, or on the side of the occiput if it is at the base of the brain. In rheumatic cases, the patient should take a smart purge and avoid alcohol. An alkaline diuretic mixture (bicarbonate of potash, or acetate of ammonia, and nitric ether) may be given at the onset. If there is evidence of general rheumatic catarrh, a hot bath, or better still, free diaphoresis, may be employed. It is important that the patient should, if possible, keep indoors. If this is impossible, the side of the head should be protected. Subsequently the blister may be repeated, and small doses of iodide of potassium may be given, with quinine and strychnine.

There is no evidence to show that electricity has any influence over the process of nerve degeneration or recovery. But the voltaic current excites the muscles to contraction when they are otherwise absolutely inert. It is reasonable to conclude that such functional stimulation of the muscles helps to keep up their nutrition. The nerve fibres may recover functional power after months of interruption. During this time the muscles, left alone, undergo partial atrophy, and it is probable that this is less if galvanism has been sedulously employed. If a case has not been galvanized, after several months of absolute paralysis scarcely any contraction may be obtained the first time the current is applied, but after a few applications the muscles may respond vigorously, and it sometimes happens that the first indication of returning voluntary power follows such a "waking up" of the muscles. The positive electrode should be placed below the zygoma, and the negative stroked along the course of the muscles, along the zygomatici, the orbicularis, in the upper and lower lip, the levator anguli oris, across the frontalis and the eyelids, the upper eyelid being lowered. The strength used should be the minimum to which the muscles will respond; usually from four to eight cells of an active battery are sufficient, the current used for the eyelids being weaker than for the other muscles. It has been said that the positive electrodes should be applied to the muscles, because these respond more readily to this than to the negative pole. But this is not always the case; sometimes the response to the negative pole is the more ready; often it is the same to each pole, and the irritability to the negative is scarcely ever more than a single cell behind that to the positive. The rule given above is, therefore, on the whole the best. The application should be made for a quarter of an hour once or twice a day. It can be made perfectly well by the patient himself, seated before a looking-glass. As the face recovers, the increased voltaic irritability lessens and faradaic irritability returns, slight at first, but gradually increasing. It has been advised that faradaism should then be substituted for voltaism, but it is very doubtful whether faradaism does good. The voluntary power which is then regained constitutes a stimulus to the muscles far greater and better than that of faradaism, and it influences the same muscular fibres. If any electrical treatment is continued it is better to still use the voltaic current, which not only stimulates the nerve fibres as surely as faradaism, but also any muscular fibres of which the nerves have not yet recovered. When there are indications of commencing late contracture, the use of faradaism is distinctly contra-indicated, and it is, perhaps, better to stop all stimulating electrical treatment. Although this is certainly not the cause of contracture, it may yet tend to increase it, and when this stage is reached electricity has probably effected all that it can achieve.

For the contracture and over-action little, unfortunately, can be done. I have never been able to observe any beneficial result from either drugs or local treatment. The use of a weak, unbroken voltaic current has been recommended, but its influence is inappreciable. Warm douches, or steaming the face, and the internal administration of bromide of potassium have been recommended (Rosenthal). Daily gentle shampooing of the face, by moving the finger, with gentle pressure, along the course of the muscles from origin to insertion, may be adopted as having an influence in the right direction, and at least incapable of doing harm. Faradisation of the muscles of the sound half of the face neither has nor can have any influence on the contracture, beyond a tendency to increase it by reflex stimulation through the fifth nerve.

FACIAL SPASM.

Spasm in the muscles supplied by the facial nerve is sometimes termed "*mimic spasm*," from the semblance of emotional expression which results, and also, after the French, *convulsive tic*.*

Facial spasm may affect only a few muscles or almost all those of one side. Either form may be unilateral or bilateral. It may be primary or secondary to paralysis. The secondary form has been already described (see p. 656). The spasm is usually clonic; if there is tonic spasm there is almost always clonic spasm as well. Simple tonic spasm is extremely rare.

The orbicularis palpebrarum is more prone to spasm than other muscles of the face, no doubt because the motor mechanism for this muscle is more sensitive, in consequence of its important and energetic reflex action. Hence spasm, ultimately general, often commences in the eyelids, and partial spasm affects them far more frequently than any other part of the face. This eyelid spasm is termed "blepharospasm," and will be separately described, while the habit spasm of childhood, a totally different disease, is considered in connection with chorea.

Causes.—The cases of facial spasm, considered from the point of etiology, fall into two classes: first, those in which an organic lesion is certain or probable; secondly, those in which there is no indication of organic disease, and the general causal influences that can be traced render it probable that the spasm is idiopathic. Each form is met with only during adult life, after twenty years of age. Only the spasm that succeeds paralysis occurs in childhood and youth. Ordinary facial spasm usually commences between thirty and sixty, rarely between twenty and thirty or after sixty. An inherited neurotic tendency can sometimes be traced; direct heredity is very rare.† Women suffer far more frequently than men, constituting three-quarters of the cases (twenty-three out of thirty-two); indeed, the idiopathic form is almost confined to females. It does not seem to have any special relation to hysteria. Anæmia and general defective nutrition of the nervous system conduce to its occurrence, but the cause of the idiopathic form that can be most frequently traced is mental anxiety, sometimes a sudden shock, more often prolonged grief. In this connection it is instructive to note, on the one hand, the intensely disturbing influence of such emotion on the nervous system, and, on the other, the relation of contraction in the facial muscles, and especially of those in which the spasm often begins, to the process of weeping. Such emotion preceded the onset in five-sixths of the cases that have come under my own observation. In many, the disease begins about the climacteric period. Another occasional cause is irritation of the fifth nerve, to which the facial has a special reflex relation. The eye is the most common seat of the irritation, and the spasm begins in the eyelid and may afterward become general. Irritation of other branches of the fifth, as from decayed teeth, has been present in some cases. Rarely, neuralgia in the neck has preceded the spasm, but neuralgia, however violent, seldom sets up spasm of independent course, in spite of the fact that muscular contraction often accompanies paroxysms of severe pain. Irritation of the intestines or uterus has been alleged as a cause, but on insufficient evidence. Severe cold acting on the face and head has apparently caused the disease in a few instances, perhaps also by a reflex mechanism, since in some of these cases, although not in all, pain has preceded the spasm. It is to be noted that the effect of these influences is not always immediately manifest. Thus, a man's face was scorched, and his eyelashes and eyebrows were burned, by the explosion of a cartridge; a month later, when the face was well, spasm commenced in the muscles of the eyelids and nose, and continued for years. A soldier during the Franco-German War suffered from severe neuralgia of the right side of the face, induced by cold; two years afterward, this side of the face became the seat of spasm (Bernhardt).

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* This is the original meaning of the word "tic," concealed in part by its use as an abbreviation of the more common *tic douloureux* (neuralgia), to which it was applied, either metaphorically, from the twitch-like character of the pain, or else on account of the spasm that may accompany severe paroxysms of neuralgia. The original French form *ticq* is supposed by Skeat to be allied to *tukken*, Low German, *to twitch* (modern German, *zucken*).

† Rosenthal has described the affection of five members of a family, but he does not state the form of spasm. In most cases of direct inheritance the affection is not true facial spasm, but a kind of habit spasm.

Organic disease causing facial spasm has been in one of two situations, in the facial nerve or the facial centre in the cortex of the opposite hemisphere. In several cases spasm has been produced by a small tumor pressing on the nerve at the base of the brain, a small sarcoma in one case,* a cholesteatoma in another (Schuh), while in a third it was due to the aneurism of the vertebral artery shown in Fig. 274. It does not seem to be caused by disease in the petrous bone, in spite of the frequency with which the nerve suffers in this situation. In organic lesions of the pons, spasm has only been observed as a transient symptom, preceding paralysis, in consequence of an abscess. On the other hand, persistent facial spasm has been produced by a small stationary lesion of the ascending frontal convolution in the position of the facial centre, opposite the inferior frontal sulcus. Fig. 275 shows a point of softening in this situation, which caused persistent clonic spasm, limited to the zygomatic muscles.† I have seen one case in which it was highly probable that facial spasm was the result of a cortical injury during birth.

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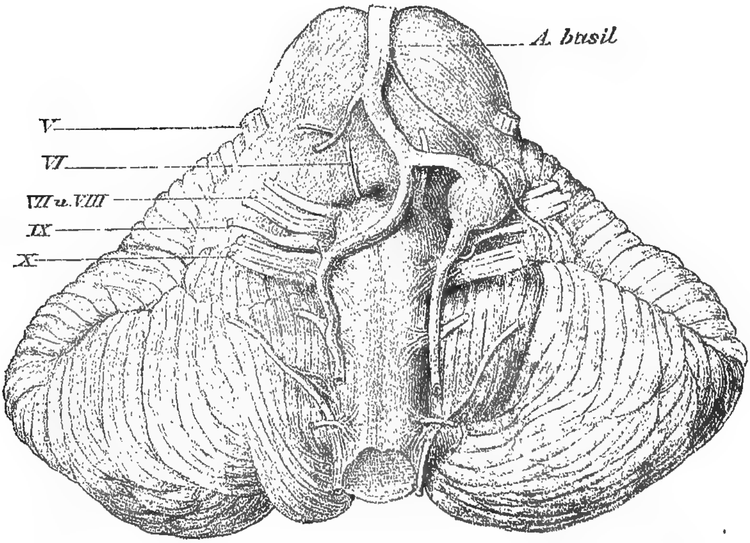
* Moos, *Archiv f. Augenheilk.*, *1874*, Bd. iv, Abth. i, p. 179.

† Berkeley, *Medical News*, July 15th, 1883. The case recorded by Allen Starr, *American Journal of Med. Science*, *1884*, Case 51, is the same.

In other cases the spasm has followed a fall on the head, and a slight injury to the surface of the brain, such as often results from contusion, was highly probable. Lastly, it should be mentioned that in many cases, at least one quarter, no probable cause for the spasm can be ascertained.

SYMPTOMS.—In some cases of facial spasm, the muscles of one side of the face present frequent momentary contractions, resembling those produced by the stimulation of the nerve with the faradaic shock. In others there is a single contraction, which recurs at irregular intervals of seconds or minutes. Often there are brief paroxysms in which there is both tonic and clonic spasm, and the latter may have a quick, quivering character. In other cases, again, the paroxysms are longer and more violent, lasting several minutes, and consisting of quick contractions, two or three per second, with imperfect relaxation between them.

FIG. 274.



Aneurism of the left vertebral artery, compressing the facial nerve, and causing facial spasm. (*After Schultze.*)*

The orbicularis and zygomatici are always more affected than the other muscles, so that the eye is half closed, the angle of the mouth is drawn outward, and the naso-labial furrow is deepened. Sometimes the spasm involves the corrugator supercilii, but scarcely ever the frontalis. Both corrugators may be involved, although the spasm is, in other muscles, one-sided. The elevator of the upper lip is often affected, and so are occasionally the depressor of the angle of the mouth, the levator menti, and the platysma myoides. The latter sometimes seems to become hypertrophied, from the continued over-action (Fig. 276). The effect of the preponderant contraction in the orbicularis and zygomatic muscles is a curiously-mixed emotional aspect, a sort of whimpering smile. In connection with the escape of the frontalis, it may be noted that its action is opposed to that of the orbicularis palpebrarum, and, in emotional expression, to the zygomatics, and that it is

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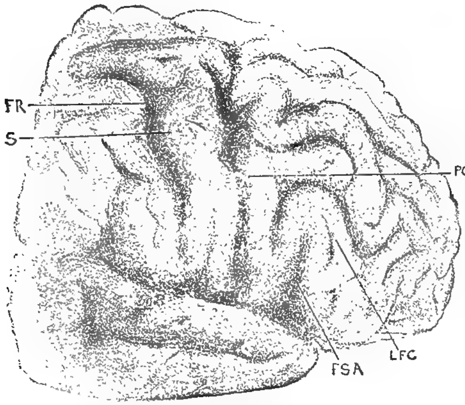
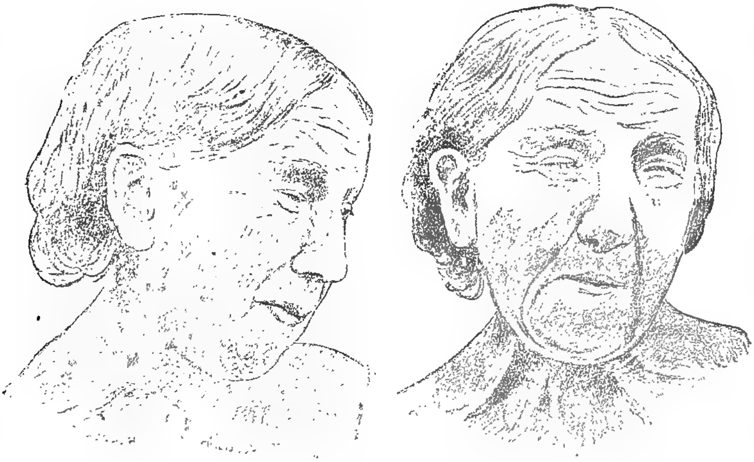
* The patient was a man, aged fifty-six, who had received an injury to the head ten years before. For a year before death he had suffered from short, quick contractions in the left side of the face, increased by any movement of the jaws or face. All muscles were affected except the frontalis; the palate did not move. There was no pain. No morbid changes could be found in the nerves on naked-eye or microscopic examination. (Schultze, *Virchow's Archiv*, Bd. 65, p. 385.)

FIG. 275.



Small focus of superficial softening, S, in the ascending frontal, opposite the origin of the fissure between the middle and lower (L F C) frontal convolutions, which had caused persistent clonic facial spasm. P C S, precentral sulcus; F R, fissure of Rolando; F S A, anterior limb of fissure of Sylvius. (*From a photograph* kindly furnished by Dr. Berkeley, of Baltimore.)

FIG. 276.



Bilateral facial spasm in a woman of sixty-one, due to grief. Over-action and apparent hypertrophy of platysma.

often associated in spasm, as in normal action, with the muscles at the back of the neck (see Torticollis). The orbicularis oris is rarely involved. Its action is also opposed to that of the zygomatics.

The spasm may be confined to the muscles of one side, and is almost always so confined at first. Often in the severer paroxysms there may be slight spasm on the other side, of the eyelids or angle of the mouth, or of the levator menti, and in rare cases, after a time, the spasm becomes altogether bilateral. Spasm limited to the muscles about the eyes is, indeed, often bilateral, involving chiefly the orbiculares, sometimes the corrugators, but it is not common for the lower muscles to be equally affected on both sides.

The spasm is usually lessened by rest, physical and mental. It is always increased by emotion, and by movement of the face, whether in speaking or chewing; in slight cases it may occur only on movement. Frequently the spasm is increased by light and by cold. The influence of light is intelligible, since the orbicularis palpebrarum is constantly involved, and a strong light produces reflex contraction in this muscle under normal circumstances. Conversely the spasm is lessened by warmth and by darkness.

Facial spasm causes no pain; when pain is present it is independent and often primary. As a rule, there is no paralysis. Voluntary movements may be interfered with by the spasm, but are otherwise unimpaired. Distinct loss of power only exists in cases of progressive organic lesions, the effect of which is to cause, first, spasm, and then paralysis. Most, if not all non-progressive cases, in which considerable loss of power co-exists with spasm, are cases of peripheral paralysis in which contracture and spasm have supervened. The electrical irritability of nerve and muscle is, as a rule, normal. I have found, however, in one case a distinct increase of irritability in the nerve.

The spasm very rarely affects the muscles of the palate, even when the movement of the face is violent and general. In the case already mentioned, in which the spasm was due to the pressure of an aneurism on the nerve trunk, the palate was unaffected. A few exceptions are on record. Thus, in a case of one-sided spasm, there were clonic contractions of the uvula, drawing it to the side affected, and synchronous with the spasm in the face (Schütz). In another case of bilateral spasm the uvula was drawn up by spasm of both sides, so that in the stronger contractions it almost disappeared (Leube).

Symptoms referable to spasm of the stapedius muscle have been very rarely noted, except in cases of tonic spasm of the orbicularis (blepharospasm). In these a continuous noise has been noted, and has persisted after the blepharospasm ceased.* Many persons can produce a quivering noise in the ear by a strong contraction of the orbiculares, and most readily if they try to turn the eyes up at the same time. In one case there was giddiness during the height of a paroxysm, with conjugate deviation of the eyes to the right, symptoms which Moos, who has recorded the case, attributes to spasm of the stapedius, suddenly lowering the pressure in the labyrinth and semicircular canals.

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* Gottstein found that in such cases the sound could be arrested by pressure on the front of the mastoid process, or by electrical irritation of the skin at the same spot. (*Archiv. f. Ohrenheilkunde. 1880.* Bd. xvi. p. 61.)

Subjective sensations of taste have not been noted, but taste was lost on the front of the tongue in one case, in which there was probably damage to the nerve. Equally rare is disturbance of the secretion of saliva, which was for a time excessive in the case of bilateral spasm in which the palate suffered. No other vasomotor or trophic disturbance has been observed.

Occasionally, the spasm spreads beyond the limit of the facial nerve, to the muscles of mastication, the tongue, and the muscles of the neck, and even of the arm.* It may begin in the tongue and spread to the face.† Extension to the eyeball muscles has not been hitherto recorded. In a case of my own there was slight deviation inward of the eye on the side of the spasm during the paroxysms, apparently from spasm of the internal rectus or inhibition of the sixth nerve. In the case of Moos it is assumed that the deviation was secondary to derangement of the equilibrium centre by the labyrinthine disturbance.

Tender points in the fifth nerve are rare in general facial spasm, although they have been noted when the spasm was limited to the eyelids.

Tonic spasm is common after paralysis, and exists in slight degree in the zygomatic muscles in some cases of old hemiplegia, and on both sides in paralysis agitans, in some cases of tetany, and in considerable degree in tetanus. Primary tonic spasm is said to have occasionally resulted from exposure to cold, to be unilateral or bilateral, and sometimes associated with trismus. Deep furrows are persistent; the mouth deviates toward the paralyzed side; the palpebral fissure is narrower than on the other side, the eyebrow is raised, there is a sense of tension in the face, and movements are hindered.

Tonic spasm of the face may be produced in the cataleptic condition of hysteria. A curious form came under my notice in a man who, after sleeping for over eighteen hours, woke up in the following condition: there was some weakness of the left arm, none of the leg, no distinct paralysis of the tongue, and no paralysis of the face, but conspicuous over-action of the left side of the face, induced by the slightest excitement, coming on even when he entered a room, and passing away only after he had been perfectly quiet for some time. Sometimes it would occur without any exciting cause. The chief contracture was in the zygomatic muscles, causing a very deep naso-labial depression. There was also slight contracture of the orbicularis palpebrarum. He was readily excited to tears, and then the angle of the mouth was drawn still further outward, and the left eye almost closed. There was also a little rigidity of the muscles of mastication. The condition presented no change during five months that the patient was under observation.

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* Keen, *Trans. American Surgical Association*, May 1st, 1886.

† Remak, *Berl. kl. Wochenschr.*, *1883*, No. 34.

Course.—Facial spasm usually begins gradually; generally in the eyelids. Months or even a year may pass before the lower part of the face is involved. Less commonly it begins in the lower part of the face, or in all parts simultaneously. The course of the affection is very irregular. Sometimes for weeks or months the face is comparatively or absolutely free from spasm, and then the movements return, spontaneously or after some emotion. The same proneness to relapse is seen when arrest has been obtained by treatment. The duration of the affection is always long; it generally lasts for years, and not unfrequently persists to the end of life.

PATHOLOGY.—The spasm may probably result from deranged action of the gray matter of the motor facial centres, either the cortical gray matter in the ascending frontal convolution, or the facial nucleus in the pons. We have seen that organic disease in either of these situations may cause spasm. We have seen, also, that it may result from pressure on the nerve trunk, but we do not yet know how such pressure acts. It may be by disturbing the downward nerve currents from the facial nucleus, which in some degree are probably constant even during rest. It is also conceivable that the pressure on the nerve may modify and derange the action of the facial nucleus itself; or, on the other hand, that the mechanical irritation of the nerve fibres may excite them directly, as electrical stimulation does. When facial spasm is due to a reflex cause, as neuralgia or exposure to cold, it is reasonable to regard the nucleus as the part chiefly affected. In the cases that are excited by depressing emotion, or develop slowly without apparent cause, there is nothing to show whether the deranged action is at the cortex or at the nucleus. The nature of the cause suggests that the morbid state is a change in the mode of action and nutrition of the nerve cells, and is not the result of any disease outside them.

DIAGNOSIS.—The chief point in diagnosis is the distinction of primary spasm from that which follows paralysis, and this rests on the history or the presence of weakness, and on the existence of persistent contracture and over-action, as well as of occasional spasm. In rare cases of chorea the face is much affected, and the limbs but little, and it is probable that such a case has been mistaken for, and even published as, a case of simple facial spasm. Attention to the condition of the limbs will prevent error, since slight movements may always be observed in them.

A more urgent question is the seat and nature of the disease. In forming an opinion on this point, we must be guided by the considerations already mentioned in the sections on causation and pathology. More facts are needed regarding the relation between the character of the spasm and the seat of the disease. It is probable, however, that when the spasm affects both corrugators, and other muscles only on one side, it is cortical in origin. Partial facial spasm, however, limited to the zygomatics, may also be cortical (Berkeley). Any tender points, or source of irritation in the fifth nerve, or a history of pain, suggest a reflex origin. Indications of weakness, secondary in time, suggest a progressive organic cause.

PROGNOSIS.—The prognosis is grave in all cases except those that are of recent origin, and are distinctly due to a reflex cause. In cases which have lasted more than a few months the probability is against recovery, although the fact that in a few instances, even of years' duration, recovery has taken place, justifies an effort to relieve. Only when the symptom is due to progressive organic disease has it any intrinsic gravity. But, trivial as it is in other cases, it causes more annoyance than many diseases of far more serious nature.

Treatment.—In a recent case, apparently excited by cold, free diaphoresis should be employed, and the face and side of the head bathed frequently with hot water. If there are indications of organic disease, the nature of this must be ascertained, and, as far as possible, treated. All causes of reflex irritation must be sought for and removed; decayed teeth should be extracted, especially if they are on the same side as the spasm. Any derangement of the general health should also be treated, and in the cases that are excited by depressing emotion tonics are usually required. So-called nervine tonics and stimulants, zinc, nitrate of silver, asafœtida, valerian, have all been recommended, but in the vast majority of cases they conspicuously fail. In only one typical case have I known recovery to occur under such treatment, which consisted of arsenic, bromide and Indian hemp. Hypodermic injections of strychnia are said to have cured one case (Sanders).

Sedatives applied to the skin seldom exert any influence over the spasm. Hypodermic injection of morphia has done good in some cases, alone and combined with atropine. From the results obtained in torticollis, it is probable that a permanent effect may be produced by keeping up the influence of morphia for many months; but this course of treatment may easily have results more serious than the malady itself. In the use of morphia for the relief of spasm it is probably more desirable to inject it in or near the seat of spasm than in the case of its anodyne use. The temple is the most convenient locality near the face, but if injections are long continued, some, at least, must be made in distant parts.

Electricity has been largely used, and has been highly praised by some of its advocates, but in nine-tenths of the cases it fails even to relieve. Only a weak voltaic current should be employed, in a "stabile," *i. e.*, uninterrupted manner. Various methods of application have been recommended, and may be tried. One is to place the anode in front of the ear, the kathode on the muscle, so that a descending current may pass. Another is to place the anode on the nerve, or behind the ear or at the occiput, the kathode on some indifferent place, so as to obtain the simple sedative influence of the positive pole. Another is to place a pole on each mastoid process. If the fifth nerve presents any tender points, the anode may be placed on these.

Counter-irritation, as by a blister behind the ear, generally causes some diminution of the spasm for a time, perhaps by the inhibitory influence of the sensory irritation, but no lasting amelioration results. Similarly, the spasm has been observed to cease during the existence of a painful affection of the conjunctiva (Bernhardt). Counter-irritation along the cervical spine by the actual cautery is said to have cured one case (Remak).

Stretching of the facial nerve has been of late adopted in these cases, but unfortunately with imperfect realization of the hopes that were entertained when the operation was introduced, and that seemed to be justified by its immediate effect. Slight stretching of the nerve seldom has any influence.* Vigorous stretching causes paralysis of the face, lasting for weeks or months, with degenerative reaction in the nerve and muscle. The paralysis in time passes away. In many cases the spasm has returned with voluntary power. In others the face has remained free from spasm for some time after the recovery of the nerve, but the spasm has ultimately returned, either spontaneously or on some fresh exciting cause, such as an emotional shock. Of thirteen cases collected by Godlee,† in only one (recorded by Southam‡) was the patient free from spasm two years after the operation, and in a more recent collection of twenty cases by Keen, the only other case of cessation for a longer period than six months is that of Zeiss, mentioned below.§ In some cases the spasm became as severe as ever, in others it was distinctly less intense than before the operation and remained so. As a remedial measure, the operation clearly deserves little confidence, and the prospect of amelioration seems scarcely sufficient to justify its performance. At any rate, it should be limited to cases in which there is reason to refer the spasm to a functional condition of the nucleus, and not to organic disease, and it would be desirable not to trust to the operation alone, but to follow it up by a course of treatment, such as the hypodermic injection of arsenic and morphia, which is known to have some power of lessening such spasm; this should be commenced when the palsy is passing away. The operation may perhaps have an indirect influence on the nerve cells of the nucleus.

Blepharospasm.—Spasm confined to the eyelids, and showing no signs of extension to other parts of the face, is distinguished from other forms of facial spasm also by its causes and its bilateral character. It occurs in *tonic* form in connection with photophobia, as a result of painful affections of the eye, and sometimes of other branches of the fifth nerve. It is an excessive action of the reflex mechanism that normally guards the eye. When thus set up it may persist as a troublesome affection, in excess of its cause, and may continue in the dark, and even exist in an eye that is blind. *Clonic* spasm of the eyelids is sometimes dignified by the name "nictitation," and sometimes occurs in hysteria, and also in children as part of "habit chorea," which is elsewhere described. The most important elements in treatment are the removal of nerve irritation, for which, in the case of conjunctival disease, cocain is useful, and the diminution of the central over-action by sedatives, such as bromide and belladonna, and by tonics, such as iron and quinine. Cold douches to the eye should also be used.

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* In a case recorded by Zeiss, *Wien. med. Wochenschr.*, *1884*, No. 2, and 1885, No. 27, stretching insufficient to cause paralysis was followed by slow diminution in a spasm of eight years' duration, which disappeared seven weeks after the operation, and was still absent two and a half years later.

† *Trans. Clin. Soc.*, vol. xvi, p. 220. In a previous paper by the same author (ib., vol. xiv, p. 44) the method of performing the operation is fully described. It is also described by Keen, loc. cit. (p. 665).

‡ *Lancet*, August 27th, 1881, and a later note in Godlee's table.

§ The only case of relief for longer than three months is one operated on by himself, in which the spasm was still absent five months after the operation.

AUDITORY NERVE. DISTURBANCE OF HEARING.

It is probable that the fibres of the auditory nerve that go to the cochlea and vestibule on the one hand, and those that supply the semicircular canals on the other, differ in function, and that only the former are excited by sound.[*] The latter seem to convey impressions from the canals, generated by the pressure or movement of the endolymph in various postures and movements of the body. Hence the latter fibres have been distinguished by Cyon as the "space nerve" (*Raumnerv*), and, although the term is awkward, a better one has not hitherto been suggested. The two sets of fibres unite in a common trunk, in which they cannot be distinguished. Within the internal auditory meatus and at the base of the brain, the auditory nerve is adjacent to the facial. The deep origin of the nerve has been already described (p. 485), and we have seen that many fibres pass directly to the cerebellum, with which also the nuclei are probably connected. The former probably convey impressions from the canals to the equilibrial mechanism in the middle lobe. The upward auditory path seems to pass by the superior fibres of the tegmentum of the crus[†] to the internal capsule, but whether it reaches the tegmentum through the pons or through the cerebellum has not yet been ascertained. There is certainly a decussation above (or at the level of) the nuclei, so that the auditory path from each ear passes to the opposite hemisphere. The cortical centre for hearing is seated in the first temporo-sphenoidal convolution (see p. 464), and that of the left side subserves the auditory use of words.

Disturbance of the functions of the auditory nerve are much more often due to disease of the ear than to affections of the nerve trunk or centres. Our knowledge of the symptoms that are of primary nerve origin is still very imperfect, because they are for the most part identical with those due to lesions of the labyrinth. Exact aural diagnosis stops at the middle ear, and its present state resembles that of the diagnosis of affections of the eye before the invention of the ophthalmoscope. Labyrinthine and nerve lesions are now confused under the term "nervous deafness," just as affections of the retina, choroid and optic nerve were included under the common term

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[*] This assumption is, however, at present unverified. It is practically certain that the semicircular canals give rise to impressions other than those of sound, and there is no evidence that, in man, they are concerned with hearing, but the latter function is not *excluded* by present evidence.

[†] See p. 486.

"amaurosis." Indeed, since labyrinthine diseases cause symptoms by their influence on the nerve fibres, the confusion seems almost inevitable.

Five kinds of symptoms may result from disease of the auditory nerve in its course or termination. (1) Loss of function, deafness. (2) Increased action, auditory hyperæsthesia, "hyperacusis." (3) Irritation symptoms in the auditory function of the nerve, causing subjective sensations of sound, "tinnitus aurium." (4) Disturbance of equilibrium, or sensations of such disturbance, due to derangement of the fibres from the semicircular canals. (5) Connected with the last are certain involuntary movements that have been observed in rare cases of disease of the nerve within the ear. Pendulum-like oscillations of the head were present in two cases recorded by Moos.* The fourth class of symptoms, which constitute "labyrinthine" or "auditory vertigo," can be considered more conveniently in connection with other forms of vertigo, while the forced movements are too rare to deserve detailed description. Here only the derangements of the auditory function of the nerve can be briefly considered in their medical relations. For a fuller description of the symptoms, the reader is referred to special works on Diseases of the Ear.

DIMINISHED FUNCTION; NERVOUS DEAFNESS.

CAUSES.—(1) By far the most common cause is disease of the labyrinth, either hindering the passage of the vibrations to the structures in which the nerve fibres end, or else damaging those structures and the nerve endings themselves. Such disease may be primary in the labyrinth, or may extend to it from the middle ear. Our knowledge of the nature of the morbid processes is limited to the occurrence of acute inflammation, chronic inflammation, syphilitic disease,† and degenerative processes. Acute inflammation is sometimes bilateral, and thus causes deafness on each side. It is probable that the membrane of the labyrinth, in the old, undergoes degenerative changes, since these may be observed in the membrana tympani. In those who are gouty, symptoms sometimes suggest that the internal ear may be the seat of changes similar to those met with in other fibrous tissues in this disease.

Certain drugs cause deafness having the characters of labyrinthine deafness, and probably such, because congestion of the internal ear has been found to accompany the symptoms. A loud noise has been known to cause permanent deafness. Lucae suggests that the mechanism may be a hemorrhage into the labyrinth, since he has found an extravasation in the middle ear from this cause.‡

(2) Much less common than disease within the internal ear, are lesions of the nerve trunk in the internal auditory meatus or at the base of the

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* *Zeitsch. f. Ohrenheilk.*, xii, p. 101.

† Moos, *Virchow's Archiv*, Bd. 69, p. 313 (with autopsy); see also McBride, *Glasgow Med. Journal*, *1885*, p. 172.

‡ Lucae, "Subjectiv. Gehörs-empfindungen," 1884.

brain. The nerve suffers chiefly from disease commencing outside it, especially morbid growths or meningitis, of syphilitic or other nature, and aneurisms. It may also be compressed in consequence of thickening of the cranial bones, narrowing the meatus. Disease arising in the nerve itself is rare; tumors (neuromata), interstitial hemorrhage, and calcareous nodules have been met with. Primary inflammation has not been proved to occur, although an auditory neuritis analogous to optic neuritis has been suspected in cases of cerebral tumor. In cases described as rheumatic neuritis, an affection of the labyrinth was more probable.

Primary degeneration of the nerve occurs occasionally in locomotor ataxy (Erb, Wernicke). A case is mentioned on p. 297, in which the progressive limitation of the range of hearing made such atrophy of the auditory nerve highly probable, but, as is shown by another case there referred to, this morbid process has certainly been sometimes assumed to exist without sufficient grounds, and the symptoms of "nervous deafness" in tabes are no proof of a primary atrophy. Such degeneration has also been met with as an isolated change, chiefly in the old, as in a woman eighty-nine years of age, in whom this atrophy was found by Lucae. Concretions of phosphate of lime have also been observed in the substance of the nerve (Moos). Long-continued disease of the nerve, in consequence of disease of the middle ear, has been supposed to lead to atrophy, but the evidence is inconclusive.

(3) The nuclei within the pons may be damaged by hemorrhage, softening or tumor, but seem to escape more frequently than other nuclei.

(4) Deafness is sometimes produced by disease above the nuclei. It may be caused (*a*) by disease which damages the superficial layer of the tegmentum, such as a tumor of the corpora quadrigemina;* (*b*) by disease of the internal capsule; it is then associated with hemianæsthesia; (*c*) by disease of the cortex in the upper part of the temporo-sphenoidal lobe, or white substance within it.†

(5) Loss of hearing may be of functional origin. In hysteria it is common, as part of hemianæsthesia. In anæmia there may be some loss of hearing, which may pass away when the general health is improved. Considerable loss of blood has been followed by absolute deafness, analogous, perhaps, to the blindness that results from the same cause; after death no morbid condition of ear or nerve has been discovered.

SYMPTOMS.—Deafness, the indication of lessened function of the auditory nerves, is due to so many morbid states of the ear, that alone it is of little value as an indication of an affection of the nerve. For a full description of the methods of examination of the ear, the reader is referred to special works.‡ Some points of medical importance may be here mentioned.

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* See p. 486.

† Deafness in the left ear, with left-sided loss of sensation, was produced by a tumor of the right hemisphere in a case reported by Strümpell, *Neur. Centralbl.*, *1882*, p. 361. See also Sharkey's case, Fig. 185, p. 463.

‡ An examination of the external meatus should, of course, never be omitted, and I may

When the deafness is due to obstruction of the meatus, or to disease of the middle ear, *i. e.*, whenever there is impaired conduction of vibrations to the internal ear, and no disease of the labyrinth itself, there is deafness to vibrations that reach the ear through the air, while those that are conducted through the bones of the skull can still be perceived. A vibrating tuning fork held opposite the meatus is inaudible, but it is heard at once if placed in contact with the bone of the skull. In cases of slighter deafness, in which the tuning fork can be heard through the air, defect of conduction can be demonstrated in another way. Normally, as the vibrations lessen, they can be heard through the air longer than through the bone; when the fork in contact with the skull ceases to be heard, the sound can still be perceived if the instrument is held opposite the external meatus. This is because the receptive mechanism is more sensitive to vibrations that reach it through the tympanum and chain of bones than to those which it receives from the bone directly. If the tuning fork cannot be heard longer through the air than through the bone, there is impaired conduction through the meatus or middle ear.*

Sounds conducted through the bone are intensified by closure of the external meatus. This is due to the circumstances that vibrations escape by the open meatus† (Mach), and that the closure of the meatus intensifies the vibration by converting the cavity into a resonant chamber (Lucae). The intensification shows that hearing through the bone is normal, *i. e.*, that the labyrinth is sensitive. But it is a less convenient test than simple contact, because unobservant persons often fail to recognize the increase. If there is some deafness to sounds through the air, and yet the tuning fork is heard longer through the air than through the bone, *i. e.*, the hearing through air

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in passing, mention the very great suitability of the ophthalmoscope for this purpose. A three-inch lens should be held over the speculum, slightly tilted to get rid of the reflection. The lens concentrates the light on, and at the same time magnifies, the membrana tympani. A light vulcanite speculum answers best.

* It is assumed that vibrations pass directly from the bone to the labyrinth. There is, however, reason to believe that some of the vibrations reach the labyrinth from the bone through the tympanum. Gellé maintains that all the bone vibrations thus reach the labyrinth, and that what is termed bone deafness, even when complete, may, and commonly does, result from disease of the stapes and of the foramina ovale and rotunda. He relies especially on the fact that conduction through the bone may be lessened and even extinguished by compression of the air within the meatus, which, he assumes, arrests the movement of the ossicles. Hensen, therefore, has proposed to call conduction through the bone "cranio-tympanic conduction." But that vibrations may pass to the labyrinth directly seems proved by the fact (observed by Lucae) that when the membrane and bones of the tympanum are congenitally absent, sounds can still be heard through the bone. The effect of compression on which Gellé relies is susceptible of another explanation—that it acts by increasing the pressure within the labyrinth, and so preventing the vibration of the structures connected with the nerve.

† If a stethoscope or otoscope is applied to one meatus, sounds entering the other ear can be distinctly heard; the old adage, "in at one ear and out at the other," having thus a basis of literal truth.

and bone have their normal relation, the deafness is due to the nerve or labyrinth.

The high-pitched short sound of a watch furnishes a test of considerable value and delicacy, but caution is needed in drawing inferences from the results obtained. The best method of using it is to close the meatus by pressing back the antitragus, and first to hold the watch near, but not in contact with, the root of the zygoma, and then to press it firmly against the bone. The observation may be repeated against the mastoid process. If the watch is heard better in contact than when not in contact, the function of the labyrinth is not impaired. If it is not heard better in contact, further observations may be made with the tuning fork. If the watch is heard through the bone, it is not likely that the tuning fork will reveal impairment. If the sound is heard through the bone, but not so loudly as normal, we cannot infer disease of the labyrinth or nerve, because simple anchylosis of the stapes will reduce bone conduction, although no disease of the middle ear will extinguish it (see note on page 672). But during the degenerative period of life, after fifty or sixty years of age, it is very common for the watch not to be heard through the bone, although there is no other evidence of impaired function. The loss apparently depends on the labyrinthine changes incidental to age already mentioned. During the first half of life it is rare to find bone deafness, and its existence then may reasonably be regarded as evidence of a pathological condition, and this is true at any age if the defect exists on one side only. Even after middle life a bilateral loss is sometimes of significance.

The state of hearing through the bone is of special importance to the physician, because it indicates the functional state of the internal ear and nerve, and eliminates the morbid states of the conducting media, the meatus and tympanic cavity. It is assumed that the vibrations pass from the bone directly to the labyrinth.

Another indication of impaired function of the nerve is an altered electrical reaction, first ascertained by Brenner, and since studied by Erb and others. But this is little employed in diagnosis. The attempt to obtain it often causes vertigo; an alteration occurs in morbid states of the middle ear as well as in affections of the nerve, and, in the latter, is sometimes absent.

We have no means of distinguishing between disease of the labyrinth and nerve, except by the associated symptoms. In each case the deafness is the same, and is often associated with symptoms of irritation—subjective sounds. If the facial nerve is paralyzed and there is no disease of the middle ear or bone, we may feel sure that the nerves are affected at the base of the brain or in the internal meatus.* A basal affection is also probable if any other nerve, near the auditory (as the sixth) suffers with it. In rare instances the auditory nerve is damaged without the facial at the base of the brain, perhaps because the resisting power of the *portio mollis* is less than that of the *portio dura*.

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* If, with this combination of symptoms, there is also disease of the middle ear, it is probable that the facial nerve and the labyrinth are affected by extension of disease from the tympanum, but this diagnosis is not certain, as a case recorded by Schwartze shows. In spite of disease of the middle ear, the deafness and facial paralysis were found to depend on a tubercular tumor springing from the dura mater, and compressing the trunks of the facial and auditory nerve ("Beiträge zur Path. des Ohres," 1870).

The symptoms of disease of the auditory nuclei in the medulla are still little known. In several recorded cases, disease of the nuclei on one side has caused deafness in the ear on the same side. But we do not know whether this relation is invariable, whether unilateral disease always causes deafness on the same side, or whether the connection of nucleus and ear is in part crossed.* Moreover, extensive damage to the nuclei has been found when no deafness was noted. Nuclear disease may be suspected if deafness comes on suddenly, together with other symptoms of a lesion of the pons or medulla, especially with weakness of the limbs on the opposite side. Sudden deafness, alone, is of no localizing significance, because either the trunk of the nerve or the labyrinth may be the seat of sudden hemorrhage. Deafness of gradual onset, in association with bulbar symptoms, is also of little significance as regards disease of the nucleus, since it is more frequently due to pressure on the nerve. Deafness, with hemianæsthesia or hemiplegia on the same side, is probably due to disease of the cerebral hemisphere.

Bilateral deafness may be due to various causes: (1) Symmetrical disease of the labyrinth, which is common ; acute inflammation is sometimes, and chronic degeneration is often, bilateral; double otitis has sometimes been mistaken for meningitis; (2) symmetrical lesions of the two auditory nerves, which are very rare; (3) possibly disease of the medulla; (4) a tumor in the corpora quadrigemina, damaging the crustæ of the crura cerebri; (5) symmetrical disease of each temporo-sphenoidal lobe; syphilitic gummata caused this effect in a case recorded by Wernicke and Friedländer.

Treatment.—For a description of the little that can be done in the treatment of disease of the labyrinth, the reader is referred to special treatises. The treatment of disease of the nerve trunk or centres is, for the most part, that of the morbid process, and must be conducted on the same principles as that of similar lesions in the other cranial nerves. In cases of acute deafness, counter-irritation, as by blisters, is often of great service, and even in chronic cases may do some good. The auditory nerve may be stimulated by electricity, but the results obtained by most of those who have employed this method have been slight and disappointing.

AUDITORY HYPERÆSTHESIA.

The term is applied to an increased or perverted action of the auditory nerve or centres. While deafness is much more frequently due to diseases of the ear than to morbid states of the nerve, the opposite is true of hyperæsthesia, which is commonly due to altered nerve function.

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* See a curious case of tumor of the right cerebellar amygdala (Wolf, *Archiv f. Ohrenheilk.*, Bd. xvi, p. 157), which compressed the right auditory region, and is said to have caused deafness of the left ear and left facial paralysis. The latter, however, might have been due to a tumor that existed in the ascending parietal convolution.

(1) *True hyperæsthesia,* increased keenness of hearing, "hyperacusis," is a rare morbid state in which sounds are heard with undue loudness, and even such as are inaudible to other persons are distinctly perceived. It occurs chiefly in hysteria, usually in association with augmented acuteness of other senses, and is probably, in such cases, of central origin. It has also been observed at the onset of acute cerebral and general diseases.

From true hyperæsthesia we must distinguish certain forms of hyperacusis due to disorder of the conducting mechanism. In paralysis of the stapedius muscle, from disease of the facial nerve, low notes may be heard with undue loudness.

(2) *Auditory Dysæsthesia,* "*Dysacusis.*"—Sounds, although not heard with undue loudness, cause discomfort. This is common in cerebral affections, functional and organic, during attacks of headache, in many cases of meningitis, etc. The pain in the head seems to be intensified by the noise. There is no evidence that irritation of the auditory fibres ever produces pain directly. Politzer has indeed recorded a case of labyrinthine disease in which chords played on a harmonium caused a distinct sensation of pain, while single notes did not; but the case stands alone, and it is probable that the pain was produced in some indirect manner.

The treatment of these forms of auditory over-action is that of the morbid state with which they are connected, and the nature of this is usually obvious. If direct treatment is needed, full doses of bromide of potassium have most influence in diminishing the morbid excitability.

IRRITATION OF THE AUDITORY NERVE; TINNITUS AURIUM.

Subjective sounds are of varied character and still more varied origin. They constitute a common ailment, and one that is often most distressing; an ailment, moreover, that often baffles all efforts to relieve it. Its frequency is not surprising. When we consider, on the one hand, how exquisitely sensitive the organ of hearing is, how slight a vibration will affect it, and, on the other hand, that around and even within it blood is in constant, and muscle in frequent, motion, the marvel may well be the habitual silence rather than the occasional sound. It has indeed been thought, with reason, that the silence is due to cerebral inattention and not to peripheral inactivity. In a quiet room, continuous auditory sensations may be noticed, of which we are normally unaware, just as in the dark the field of vision may be found full of moving points of light. Almost any morbid process, in any part of the ear, may cause subjective sounds; they may result also from sound-producing processes in and about a normal ear, as well as from irritation of the auditory nerve and centres within the skull.

(1) The blood currents within and near the ear, which normally give rise to no sound, may be so changed that they cease to be noiseless. In anæmia, vibrations occur in the blood with undue readiness, and a pulsating murmur may be heard, probably produced in the carotid artery. A

similar murmur may also arise in an intracranial aneurism. Vasomotor paralysis of the labyrinthine vessels has probably been the cause of a subjective sound observed in a case of disease of the cervical spine, pressing on the vertebral artery along which the sympathetic fibres run. A similar mechanism may have been effective in another case in which there was enlargement of the glands of the neck adjacent to the sympathetic; the noise ceased when the glands became small.* Perhaps dilatation of the small arteries within the ear may give rise to a similar sound, and if tinnitus is ever, as is asserted, of reflex origin, it must be thus produced. Pulsating sounds are, as a rule, arrested or lessened by compression of the carotid artery in the neck.

(2) Tinnitus may attend every form of ear disease—accumulations of cerumen in the external meatus; inflammation of the middle ear, catarrhal, plastic, or suppurative; spasmodic contractions of the tympanic muscles, and various morbid states of the labyrinth. The precise mechanism by which the sounds are produced is a matter of conjecture; vascular congestion, increased labyrinthine pressure, and irritation of the nerve endings or their connected structures have all been assumed, and may all be operative in different cases.

(3) Organic changes that irritate the fibres of the auditory nerve or its centres may cause subjective sensations of sound, and these may therefore be present as a symptom of any of the morbid states already mentioned as affecting these parts, and producing loss of hearing, degeneration, concretions, etc.

(4) Intense stimulation of the auditory nerve, as by a loud railway whistle, has been known to set up a lasting subjective sound, but by what mechanism is unknown. Habitual exposure to sounds may cause tinnitus, usually slight in degree; musicians, piano tuners, smiths, etc., are said to suffer occasionally in this way.

(5) Subjective sounds are sometimes due to a functional disturbance of the auditory centres. In migraine paroxysmal sounds sometimes occur, although they are a rare symptom. Such sounds are more common as the aura of an epileptic seizure; it is probable that they are due to disturbance in the cortical auditory centre, since organic disease in this position may cause such a sound as the warning of a convulsion (see p. 464). That continuous tinnitus sometimes results from central functional disturbance is also probable, on account of the elaborate character of the sounds that some patients experience. In various conditions of nervous exhaustion and excitability, tinnitus may arise without impairment of hearing. Of the pathology of these cases nothing is certainly known, but it is probable that they are due to defective nutrition of the nerve elements, central or peripheral.

SYMPTOMS.—The sounds that are heard are extremely various in character and intensity. Slight sounds may be low pitched, a low rumble like a distant wagon, or a faint murmur such as may be heard when a shell is held up to the ear. Louder sounds are rarely low in tone. They may be humming, hissing, rushing, or roaring noises. A common simile is that of stone-sawing, or the hissing of a kettle or a gas jet, while still louder noises are compared to that of a steam engine or a waterfall, or to a room full of machinery in motion. Others again, less common, are like the sound of a bell, or of many bells jangling at a distance, while still more elaborate sensations may resemble music or voices. Definite words are seldom heard, except in cases of insanity and epilepsy. An important difference in the more simple sounds is their continuity. Some remit or intermit, and are synchronous with the pulse; others are continuous and unvarying. The significance of the intermission is that the sound is directly due to arterial pulsation, but its ultimate cause may be either an increase of this pulsation or an increased sensitiveness of the nerve structures. Hence the sound is intermitting, not only in aneurism, but in anæmia, and also in many diseases of the middle ear, and in some labyrinthine affections, and even occasionally in morbid states of the centre.* Sounds of central origin are generally continuous; but continuous sounds may also be due to ear disease, labyrinthine or tympanic, and even to accumulations of wax in the meatus. Thus this feature of tinnitus affords little help in the diagnosis of its cause. Elaborate sounds are generally of central origin. A lady for twenty years has heard the sound of music; no actual tune can ever be distinguished, but she states that were it not for its persistence the sound would be pleasant; no affection of the ear can be discovered. Auditory hallucinations of the insane, although of cerebral origin, are sometimes determined by the presence of ordinary tinnitus, which existed before the onset of the hallucinations.

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* These two cases are recorded by Brandeis, *Zeitsch. f. Ohrenheilk.*, xi, 1882, p. 294.

At the beginning of the affection, the sound, especially if slight and of a familiar character, may be thought to have an actual objective origin. This is especially the case when the sound is elaborate. One patient sent a message to his next-door neighbor, asking that a clock might be moved, the loud striking of which annoyed him; he had no idea that the sound was subjective until informed that there was not a striking clock in his neighbor's house. In most cases, however, the real nature of the sound is quickly recognized. The sound is usually referred by the patient to the ear; sometimes it seems to be in the head generally; the difference depends in part on the loudness of the sound, and partly on its bilateral character. One patient, for instance, said that the sound, as a rule, seemed to be in the ears, but when it became more intense it seemed to be within the skull. Under normal circumstances a sound conveyed simultaneously to both ears by a double tube is referred to some region in the middle line of the skull, the precise locality varying in different persons.

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* That intermittent sounds may be of central origin is certain, from the facts of epilepsy. I have elsewhere recorded a case in which part of the aura was an intermitting hissing, evidently synchronous with the pulse, and succeeded by two bright lights, which seemed to approach the patient by jerks, of the same rhythm as the preceding sounds, "Epilepsy, etc.," p. 67.

The sounds may vary from time to time in their intensity. Such variations may be distinctly dependent on the general health of the patient; the sounds may lessen or even cease during good health, and may increase when nervous tone or general strength is lowered. Occasionally tinnitus is slight or absent at certain periods of the day, or absent on one day and loud on another, without any obvious cause for the variation. A sudden paroxysmal increase is rare except in association with attacks of vertigo; these may be heralded by increasing intensity of sound, compared by some patients to the whistle of a swiftly-passing train.

Slight tinnitus is heard only when there is external quiet, and even loud sounds may be inaudible in a considerable noise, as that of a railway carriage. Occasionally external sounds intensify tinnitus, especially that which arises from continued noise.* It is rare for hearing to be interfered with by the tinnitus *per se;* it may, indeed, be normal to every test, even when the subjective noise is loud, but more frequently there is some deafness; the morbid process that irritates the auditory nerve also impairs its function, or hinders the passage of vibrations through the ear. In some cases of progressive disease the deafness gradually increases, while the noises become less, and they may cease altogether when the patient is entirely deaf. In other cases the noises continue in spite of absolute loss of hearing. When the sounds are loud, the distress they occasion is very great, and has driven more than one patient to commit suicide.

A sound produced in an intracranial aneurism (of the internal carotid or vertebral) may be heard by another person on listening through a stethoscope applied to the skull. In extremely rare cases sounds, apparently of intra-aural origin, have also been audible, on auscultation, as in the case of a boy eight years of age, recorded by Greene,† in whom an aneurism was most unlikely. In another case a pulsating sound, resulting from some effect of a blow two weeks before, could be heard eight inches from the patient.‡

The sound of muscular contraction within the ear has a peculiar vibratory character, and is probably only produced by the stapedius muscle. This is supplied by the facial nerve, and its central connections are related to those for the orbicularis palpebrarum; hence, in many persons a strong contraction of the orbicularis is accompanied by this peculiar sound in the ear. The same sound may accompany facial spasm (see p. 664). The function of the stapedius, like that of the orbicularis, is to guard the sense organ—to prevent the base of the stapes being driven too far into the foramen by an excessive movement of the membrana tympani. Clicking sounds are probably due to the action of the muscles connected with the Eustachian tube. They are sometimes audible by another person.§ In one case clonic spasm of the levator palati gave rise to such a sound, repeated 120 times a minute, and audible twenty feet from the patient. It ceased only during sleep.*

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* Lucae, "Subject. Gehörsempf.," 1884, p. 487.

† Greene, *Trans. American Otological Society, 1878.*

‡ Poosten, *Monatsbl. f. Ohrenheilk., 1878,* No. 4.

§ Backer, *Zeitschr. f. Ohrenheilk.,* xiv, 1885, 237.

Diagnosis.—The characters of the tinnitus thus afford little help in diagnosis except that in the rare cases in which sound is elaborate it is almost certainly of central origin. An aneurism is probable if the sound is audible on auscultation, and certain if this coincides with the symptoms of a basal tumor. But in all cases the chief indication of the cause of tinnitus is afforded by associated symptoms. The most important of these is deafness, which is present in the majority of cases. The cause of the deafness is, as a rule, the cause of the tinnitus. The position and nature of the disease interfering with hearing must therefore be ascertained on the principles already described. It must be remembered that disease of the meatus or middle ear can only cause tinnitus by its influence on the labyrinth, either by increasing the labyrinthine pressure or by the extension of disease. If there is evidence of disease, not of the middle ear but of the labyrinth or nerve trunk, we can only distinguish between the two latter by associated symptoms; if these are absent we must be guided by the fact that disease of the labyrinth is far more frequent, and therefore in any given case more probable, than disease of the nerve; and that this is especially true if deafness coexists. When tinnitus is due to organic disease of the centres, other definite symptoms are rarely absent and indicate its seat and nature.

Prognosis.—With the exception of the cases in which the noise is due to removable ear disease or to easily remedied constitutional disturbance, the prognosis is unfavorable. In many cases the noises persist in spite of all treatment, but considerable relief is sometimes obtained, and occasionally the symptom is removed.

Treatment.—The first element is the treatment of any discoverable morbid process on which tinnitus may depend, directly or remotely. This causal treatment comprehends, first, the removal, as far as possible, of any ear disease by which the symptoms may have been produced; and, secondly, the treatment of any general condition with which it may be connected directly or remotely—anæmia, defective nerve power, gout, syphilis. Tinnitus in gouty persons is often lessened by alkalies and free purgation. In all cases the general health should be carefully attended to and all influences that increase the noise should be avoided. Those who suffer in consequence of habitual exposure to sounds, should obtain rest in a quiet place and protect themselves by obstructing the meatus. If such causes are not discoverable, or such treatment fails, the symptom itself must be treated, and our power of influencing it is unfortunately very small.

The noise is sometimes diminished by sedatives that lessen nervous overaction. Of these bromide has more influence than any other drug. In many cases its effect is very marked, although less than on the giddiness which often accompanies the noise. It should be given in scruple doses two or three times a day. Hydrobromic acid has been recommended, but seems to have no advantage over bromide, into which it must be converted as soon as it enters the alkaline blood, while its acidity interferes with its administration in adequate doses.* The effect of bromide is sometimes increased by the addition, to each dose, of tincture of belladonna (♏x) or tincture of Indian hemp (♏iij–v). Morphia by hypodermic injection lessens the tinnitus for a time, but is only suitable as an occasional palliative when violent paroxysms occur. No other sedative has an appreciable effect on the noises.

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* Williams, *Zeitschr. f. Ohrenheilk.*, xiii, 1884, p. 99.

Counter-irritation is unquestionably useful. A blister behind the ear often causes the sound to be less loud for a week or ten days after the application, and repeated blisters sometimes produce a permanent diminution in the intensity of tinnitus, although they rarely cause it to cease. The effect is manifest in cases of long duration, as well as in those of recent origin, and cannot therefore be ascribed merely to an influence on inflammatory processes.

Drugs that are known to interfere with the functions of the internal ear have been given in the endeavor to alter the morbid action, and in the hope that the disturbance produced may be antagonistic to that of the disease. The only evidence of such an influence is the production of deafness, tinnitus, and vertigo. These are symptoms common to morbid processes of varied character and seat. Politzer noted long ago that subjective noise was often temporarily reduced, together with hearing power, by quinine. Charcot has given quinine in sufficient doses to produce cinchonism, and found that in some cases, when the toxic effect had passed away, the noises were less. I have tried this and also salicylate of soda (15 grains three times a day) in the same way and have found the influence of the latter more distinct than that of quinine. Of course, it is only in chronic and stationary cases that this treatment is admissible, since these drugs cause actual hyperæmia of the labyrinth and are capable of increasing acute disease.† Lucae has advocated the treatment of tinnitus by exposing the patient daily for a short or long time, according to circumstances, to a sound of the opposite character to that which he hears—to a high tone if the subjective tone is low, and *vice versâ*. He employs an electro-magnetic tuning fork with resonaters.

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* The dose of the solution of hydrobromic acid usually prescribed is 20–60 minims, and this is equal only to 3–8 grains of bromide of potassium.

† It has been objected that this treatment is homœopathic. It is not more homœopathic than the treatment of psoriasis by irritants, or the administration of alcohol to reduce the rapid pulse of fever, or the application of an alum lotion in conjunctivitis, or the treatment of the constipation of colic by opium. Besides, to make the production of like symptoms a systematic ground of rejection of treatment is only less irrational than to make it a systematic ground for the adoption of treatment. To adopt the former system would be to verify the epithet of "allopath," at present an untrue name, invented to conceal, by a factitious contrast, a greater absurdity.

GLOSSO-PHARYNGEAL NERVE.

The origin of the glosso-pharyngeal nerve is described at p. 484. It leaves the surface of the medulla near the highest fibres of the pneumogastric. Its fibres are ultimately distributed to the back part of the tongue, the soft palate, tonsils, upper part of the pharynx (mucous membrane and muscles), to the Eustachian tube and to the tympanic cavity. The connections of the nerve are important. The tympanic nerve of Jacobson (arising from the enlargement on the glosso-pharyngeal, termed the "petrosal ganglion"), forms, with the sympathetic, the tympanic plexus in the wall of the middle ear, and gives two branches, one to the large superficial petrosal nerve, from (Meckel's ganglion to the facial nerve), and the other (the small petrosal nerve) to the otic ganglion. Thus the glosso-pharyngeal nerve is connected certainly with the otic and perhaps also with the spheno-palatine ganglion of the fifth nerve. Connections with the facial nerve are effected by a branch from the small petrosal nerve to the gangliform enlargement of the facial, sometimes also by a filament to the trunk of the nerve from the digastric branch of the facial, possibly by the connection of the nerve of Jacobson with the large superficial petrosal. With the pneumogastric, the glosso-pharyngeal nerve is connected at the petrous ganglion, and in the pharyngeal plexus.

Our knowledge of the functions of the glosso-pharyngeal nerve, and of the symptoms of its paralysis, is less certain than with regard to any other cranial nerve. This is due to the circumstances that the experimental study of its functions in animals is extremely difficult, and that in man it is scarcely ever diseased alone. Hence its functions have to be inferred from its anatomical distribution, and the connection with other nerves lessens considerably the value of conclusions thus reached, because the functions suggested by the termination of its fibres may be in part due to its connections, and may not represent the functions of its root.

The muscular fibres of the upper part of the pharynx are supplied by the pharyngeal plexus, and opinion is divided as to whether the motor fibres come from the glosso-pharyngeal or from the pneumogastric. The glosso-pharyngeal nucleus, however, contains large nerve cells, motor in aspect, a fact which suggests that it may furnish the fibres for these muscles. Most anatomists think that the fibres to the stylo-pharyngeus come from the facial, by the twig from the digastric branch of the latter. Whether the glosso-pharyngeal innervates any palatine muscle is not known. It is probable that it supplies sensory fibres to the upper part of the pharynx and perhaps also to the tympanic cavity. It is not probable that it is the sensory nerve for the front of the soft palate, palatine arches, or back of the tongue, since these are rendered anæsthetic by disease of the root of the fifth nerve. But it is generally believed that nausea is produced through the agency of this nerve. It is commonly regarded as the nerve of taste for the back of the tongue, palate, and the fauces. Some even believe that it subserves taste in the front of the tongue, although the unquestionable relation of this to the chorda tympani, renders it necessary to assume that this nerve derives its taste fibres from the glosso-pharyngeal by the connection between the large and small petrosal nerves (see p. 642). The fact of most weight connecting the function of taste with the glosso-pharyngeal is that its fibres have been traced to the circumvallate papillæ, the nerve structures in which, believed to subserve taste, undergo degenerative changes after division of the nerve trunk. On the other hand, there is no instance on record of loss of taste at the back of the tongue from disease of the roots of the glosso-pharyngeal nerve, while there is evidence, as mentioned on p. 642, that disease of the root of the fifth nerve causes loss of taste on the back as well as the front of the tongue, and also on the palate and palatine arch. Hence it is difficult to resist the conclusion that if some of the terminal fibres of the glosso-pharyngeal nerve subserve taste, such fibres come ultimately from the fifth nerve. Their path must then be from the otic ganglion, through the tympanic plexus, to the petrous ganglion of the glosso-pharyngeal, a circuitous path, but scarcely more so than that which the taste fibres of the chorda tympani undoubtedly take. This view is supported by the fact (mentioned on p. 643) that taste on the back of the tongue may be lost in disease of the middle ear, a fact explicable on no other hypothesis.

The nerve may be diseased by any of the intracranial processes that damage the nerve roots—meningitis, tumors, etc., and its motor fibres participate in the central degeneration, etc., that produces labio-glossal paralysis. It is probable that the pharyngeal symptoms of chronic and acute bulbar paralysis (labio-glosso-pharyngeal paralysis) are due chiefly to interference with the functions of this nerve. Of the symptoms of its isolated paralysis nothing certain is known, but it is probable that the upper part of the pharynx is rendered insensitive and weak, so that deglutition is difficult.

PNEUMOGASTRIC AND ACCESSORY NERVES.

Of all the cranial nerves, the pneumogastric has the most extensive distribution, supplying the pharynx, larynx, lungs, heart, œsophagus and stomach, and even, in part, the intestines and the spleen. In some of the so-called functional diseases of the organs which it supplies, its action is conspicuously deranged. The symptoms of its disease are thus very extensive, and it will be well first to describe them generally, and afterward to consider in detail those that merit separate description.

Some of the functions subserved by the trunk of the pneumogastric depend on the fibres which it derives from the spinal accessory; it is convenient to consider disease of these fibres in connection with that of the root of the pneumogastric, and to describe separately the derangement of the spinal fibres of the accessory nerve, which supply the muscles of the neck.

The pneumogastric, it will be remembered, arises from the side of the medulla, between the glosso-pharyngeal above and the spinal accessory below, and to the outer side of the hypoglossal. Its origin is described at p. 483. The trunk of the nerve, after receiving fibres from the spinal accessory, and giving off some small branches (of which the most important is one to the external ear), passes down the neck, behind and in the same sheath with the carotid artery; enters the thorax on the right side, over the subclavian artery, and on the left, between the subclavian and the carotid; passes through the thorax beside the œsophagus; and ends in branches to the stomach, spleen and intestines. The most important branches are the pharyngeal, which, with the glosso-pharyngeal, forms the plexus of the same name; the superior laryngeal; the recurrent laryngeal, which passes back, the left around the arch of the aorta, the right around the subclavian artery; branches to the œsophagus; pulmonary branches which, by means of the pulmonary plexus, supply the lung; and branches which form the cardiac plexus for the heart.

The vagus nerve, besides containing motor fibres for the pharynx and larynx, is the chief afferent nerve for the respiratory centre. It contains accelerating and inhibitory fibres for this centre, but the former preponderate, so that experimental division of the nerve in an animal renders the respiration less frequent, but deeper, while stimulation of the divided (central) end quickens the respiration, and may even arrest it in tetanic standstill. The inhibitory fibres are contained chiefly in the superior laryngeal nerve, and their stimulation arrests the respiration, the muscles being relaxed. It is the inhibitory nerve of the heart; slight stimulation increases the diastolic periods, and stronger stimulation arrests the action of the heart. On division of the nerve the cardiac contractions are accelerated. It has been said to contain trophic fibres for the heart and lungs, but this is not certain. The pneumogastric is an afferent nerve for the vasomotor centre, the action of which is lowered by its stimulation, so that the arteries throughout the body are relaxed. It is the motor and sensory nerve for the œsophagus, the sensory nerve for the stomach, and partly also the motor nerve for the stomach and intestines.

DERANGEMENT OF THE PNEUMOGASTRIC GENERALLY.

Causes.—The deep position of the pneumogastric and its branches preserves it from some forms of damage, although its extensive course renders it liable to suffer from many causes. The nucleus in the medulla may be damaged by local softening, hemorrhage, or slow degeneration; but in all these cases other adjacent nuclei suffer also. The nerve, at its origin from the medulla, may be compressed by thickening of the meninges, growths from the meninges or bones, or aneurism of the vertebral artery. Derangement of its function, due to syphilis, is almost always the result of meningeal disease in this situation. Other adjacent nerves commonly suffer at the same time. The trunk of the nerve is sometimes, but rarely, implicated in punctured or gunshot wounds; incised and lacerated wounds in its position are usually immediately fatal from injury to the large blood vessels to which it is contiguous. In surgical operations the trunk and branches of the nerve occasionally suffer. The trunk has been tied in ligature of the carotid, and divided in the removal of deep-seated tumors. In such operations in the lower part of the neck it is often also difficult to avoid injury to the recurrent laryngeal. In excision of an enlarged thyroid, both recurrent laryngeals have been repeatedly divided, from the time of Galen down to the present. Sarcomatous and other tumors, and enlarged glands, may compress or involve the nerve in almost any part of its course; and interference with its function especially occurs from such disease in regions limited by rigid structures, as in the upper part of the neck near the skull, and in the upper part of the thorax. Aneurisms may compress the nerve or its branches; and the recurrent laryngeals suffer from this cause with especial frequency. Other causes of paralysis of the recurrent laryngeals are described in the section on paralysis of the larynx. The vagus is, in rare cases, the seat of neuromata. Neuritis of the trunk of the nerve, due to cold, is supposed to be an occasional cause of symptoms; such cases are extremely rare, but in some acute forms of multiple neuritis it has certainly been involved. Symptoms of its derangement are occasionally produced by toxic influences, which may act on its nucleus, but more probably have led to neuritis. The vagus may also suffer in diphtheritic paralysis.

SYMPTOMS due to paralysis of the vagus are more frequently met with than those which result from its irritation. Occasionally both are combined. Laryngeal spasm and vomiting are the irritative symptoms most commonly met with, but occasionally cardiac inhibition occurs. Czermak, for instance, was able at will to arrest his heart for a few beats, by pressing a small tumor of the neck against his pneumogastric. Concato had a patient in whom a similar inhibition could be caused by pressure on the right nerve. The increased frequency of pulse which corresponds to paralysis of the vagus has been several times noted, and has occasionally been associated with diminished frequency of respiration, although the laryngeal paralysis, also resulting, has usually obscured the effect on the respiratory movements. Roux tied the trunk of the vagus with the left carotid; instantly respiration was arrested, but the pulse was also retarded; the ligature was immediately relaxed, but the patient died in half an hour. Robert also tied the nerve with the carotid; the patient, who was conscious, immediately called out, "I am suffocated!" and his voice became hoarse; he recovered, but the hoarseness continued for six months. An instructive example of interference with the functions of the vagus has been recorded by Guttmann. A lad, after diphtheria, presented paralysis of the palate and of one sterno-mastoid. His respiration quickly became reduced to twelve per minute, and very labored, while his pulse rose to 120, and he died in a few hours. In many other cases a similar change in the pulse and respiration has been noted, and even a pulse rate of 160–200. In the face of these observations, and of experiments on animals, it is not easy to understand a fact observed by Billroth, who excised half an inch of one pneumogastric, which was implicated in a tumor, without any resulting symptoms.

The important central relations of the vagus, above alluded to, cause derangement of its function to form part of many so-called functional disorders of the central nervous system. Its nucleus forms part of, or is connected with, the respiratory centre, which is conspicuously disturbed in hydrophobia and some other diseases. The phenomena of "Cheyne-Stokes breathing," or "respiration of ascending and descending rhythm," are probably the result of lowered action of the respiratory or pneumogastric centre (see p. 557). The central connections of the vagus, in the hemispheres, extend to, or are connected with, those parts which are concerned in emotion, and it is probably through the agency of this nerve that the heart's action is affected in excitement and fear. In many epileptic fits the central representations of the nerve are the parts through which the consciousness is first affected, and hence the so-called "epigastric aura."

A similar disturbance seems to be the cause of the globus hystericus and of the laryngeal spasm, which are conspicuous in some epileptic and hysteroid seizures. The nerve is closely connected with the centre or nerves for equilibration, so that severe vertigo, on whatever dependent, is often followed by vomiting. The nucleus is contiguous to the internal auditory nucleus, and part of the auditory nerve, that which comes from the semicircular canals, is known to be concerned in the process of regulating the maintenance of equilibrium. In the vertigo that results from disease of this nerve or of the canals (labyrinthine or auditory vertigo) vomiting is very common, and the nausea and retching of sea-sickness are probably due to the deranged action of the semicircular canals in consequence of the motion, and this deranged action further affects the pneumogastric centre. It is possible that the connection of the vagus with the equilibrial nerves is by means of the cerebellum, disease of which so constantly causes vomiting, although this connection has not yet been definitely traced. Conversely, gastric disturbance of the vagus is often accompanied with vertigo, especially when combined with preëxisting imperfect action of the auditory nerve.

PHARYNGEAL BRANCHES.

Branches of the pneumogastric form, with the glosso-pharyngeal, the "pharyngeal plexus." From this plexus the muscles and mucous membrane of the pharynx are supplied. The special distribution of the branches of each constituent nerve is not known.

The most common cause of paralysis of the pharynx is disease of the origin of the nerve in the medulla; such disease commonly also involves adjacent nuclei. Paralysis may, however, result from meningeal disease outside the medulla, and from disease of the bones of the base of the skull, but it is scarcely ever due to disease outside the skull. It occasionally forms part of diphtheritic paralysis.

Paralysis of the pharynx is manifested by difficulty of swallowing; food, entering the pharynx from the mouth, lodges there instead of descending to the œsophagus. Small particles or liquid may enter the larynx, and cause spasm and even actual choking. Pulpy food can be swallowed better than solids or liquids. If the paralysis is limited to the superior constrictor, liquids may, it is said, be forced into the nose by the contraction of the middle constrictor, but it is doubtful whether this occurs unless the soft palate is also paralyzed. The affection of the nerves on one side causes only slight difficulty in deglutition, no doubt on account of the circular arrangement of the muscular fibres.

Paralysis of the pharynx can be confounded with other conditions only in consequence of imperfect observations. I have known the difficulty in swallowing to be attributed to malignant disease, but such a mistake ought not to occur. If any doubt exists as to whether dysphagia is due to paralysis or organic obstruction, the passage of a sound will at once exclude the latter. From pharyngeal spasm, paralysis is distinguished by its continuous character.

SPASM of the pharynx is always part of "functional" disturbance, and does not result from organic disease. It is commonly due to hysteria, and probably occurs in the more severe form of hysterical "globus." It is usually associated with other hysterical symptoms, but sometimes occurs alone. As an instance may be mentioned the case of a gifted but highly nervous man, who for many years could only swallow food when alone; in the presence of others spasm of the pharynx always prevented deglutition. Similar spasm occurs during the paroxysms of hydrophobia.

LARYNGEAL BRANCHES.

PARALYSIS OF THE LARYNX.—Paralysis of the larynx is a large subject, of which only the more important outlines can be given here. The organ is innervated by two branches of the vagus. (1) The superior laryngeal, which arises high up in the neck. It is the sensory nerve of the larynx above the vocal cords, and supplies the crico-thyroid muscle and the deflectors of the epiglottis. (2) The inferior or recurrent laryngeal, which arises in the upper part of the thorax, and passes up to the larynx, between the trachea and œsophagus. It gives sensibility to the larynx below the vocal cords and to the whole of the trachea; and it supplies all the muscles of the larynx except the crico-thyroid and the epiglottidean muscles. All the motor fibres for the larynx come from the spinal accessory; only the sensory fibres pass to the medulla by the roots of the pneumogastric itself.

In order to understand the symptoms of laryngeal paralysis, it is necessary to have a clear conception of the anatomy of the larynx and of the action of the muscles. It may be well, therefore, first to enumerate, in brief outline, the facts which are of the most salient importance.

The glottis is opened or closed by the movement of the posterior extremities of the cords only (the anterior remaining always fixed), and this movement is effected chiefly by the motion of the arytenoid cartilages. These cartilages are attached to the cricoid cartilage by an articulation that permits free movement. Each has the form of an irregular pyramid, prolonged at the base into two processes, an anterior or vocal process, from which the vocal cord extends to the thyroid cartilage, and an external or muscular process, to which the muscles are chiefly attached. When the latter, which is at right angles to the vocal process, is moved back, the vocal process moves outward, away from its fellow; the cord is abducted, and the glottis opened. When the muscular process is moved forward, the vocal process is moved inward toward its fellow; the cord is adducted, and the glottis closed. But these movements of the cords are further aided by the movement of the arytenoid cartilages away from or toward each other.

The most important muscles of the larynx and the effects of their palsy may be briefly described.

Crico-thyroid.—The fibres, outside the thyroid cartilage, pass downward and forward to the cricoid, which they draw back and slightly tilt, lowering the posterior part of the cartilage with the attached arytenoids, and thus they elongate and make tense the vocal cords. Isolated paralysis is singularly rare, and there is much uncertainty about its exact symptoms. It is said to impair the production of high notes, but if so it is probably because, without the influence of the crico-thyroid, the internal thyro-arytenoideus cannot act with effect. It is probable that paralysis influences the height of the cords during the production of high notes, but this effect can only be recognized when the paralysis is one-sided; the arytenoid cartilage and vocal cord are higher on the paralyzed side than on the other, on account of the obliquity of the cricoid cartilage, and the posterior part of the glottis is displaced toward the paralyzed side (Riegel).

Thyro-arytenoid.—The fibres pass backward, from the posterior surface of the front of the thyroid cartilage, close to and parallel with the vocal cord. The inner fibres are connected with the cord, and seem to influence the distribution of tension. The outer fibres pass to the muscular process of the arytenoid, and, if this cartilage is free to rotate, they draw the outer process forward and the vocal process inward, thus adducting the cord. The inner fibres, and also the outer, if rotation is prevented, shorten the cord. Paralysis of the inner fibres renders the cord atonic with a concave edge. Paralysis of the outer fibres doubtless lessens the power of adduction, although in a degree that is difficult to recognize.

Lateral Crico-arytenoid.—The fibres pass from the side of the cricoid backward and upward to the outer process of the arytenoid cartilage, and, drawing this forward, move the vocal process inward and adduct the cord. It is the chief adductor. Isolated paralysis is very rare, but would certainly lessen considerably the power of adduction; it is probable that the cord could still be brought to the middle line by the thyro-arytenoid, unless there was secondary contracture of the abductor.

Arytenoid.—This, passing between the arytenoid cartilages behind, draws them together and thus adducts. In isolated palsy the vocal cords are brought together, except behind, between the arytenoid cartilages, where a small triangular space remains.

Posterior Crico-arytenoid.— The fibres of the "posticus" muscle pass from the posterior surface of the cricoid cartilage outward, upward, and ultimately forward, over the edge of the cricoid cartilage to the muscular process of the arytenoid cartilage. They draw this back and the vocal process outward, and thus abduct the cords and open the glottis. It is the only special abductor, and is thus a muscle of great importance. When it is paralyzed alone, abduction is impossible and the vocal cord is in the middle line, adducted in consequence of the unopposed action of the adductors.

Thus the vocal cord is abducted chiefly by one muscle, but the widening is increased by the separation of the arytenoid cartilages themselves, effected probably by the simultaneous action of the posterior fibres of the lateral and outer fibres of the posterior crico-arytenoids. These fibres draw the arytenoid cartilage downward and outward on its convex articular surface. The cord is adducted, and the glottis closed, by several muscles; by the lateral crico-arytenoid and the outer part of the thyro-arytenoid, which rotate the cartilage, and by the arytenoids, which bring the cartilages together. The vocal cords are lengthened and made tense by the crico-thyroid, shortened by the thyro-arytenoids, and made either lax or tense in parts, according as the inner fibres of this muscle are inert or active.

The muscles must, however, act in very complex combinations. The different fibres of each muscle have not all the same direction and cannot have the same action. The difference may even be such that some fibres of one muscle may have an effect opposed to that of the rest, if they act alone or with fibres of another muscle. This is illustrated by the fact already mentioned, that the posterior fibres of the lateral crico-arytenoideus, acting with the outer fibres of the posterior crico-arytenoideus, may aid in abduction by drawing the arytenoid cartilage downward. On the other hand, if the other glottis closers are acting powerfully, the highest (horizontal) fibres of the posterior crico-arytenoideus, the chief glottis opener, may even aid closure by helping to approximate the arytenoid cartilages. If the action may be thus complex in simple opening and closing the glottis, how much more complex must it be in the delicate and varied actions by which is produced the infinite variety of vocal sounds!

Causes.—Paralyses of the larynx fall into several widely different categories, whether they are considered in reference to their symptoms or to their causes. The chief causes are:—

(1) Organic disease of the centres, or of the nerves outside the larynx. According to the seat and character of the disease the palsy may involve one or another group of muscles, and may be unilateral or bilateral.

(*a*) The central causes are usually nuclear degeneration, involving the cells of the highest roots of the spinal accessory. In rare cases the lesion is acute central softening. In either case the symptoms are usually associated with paralysis of the tongue and lips as "labio-glosso-laryngeal paralysis," chronic or acute; it will be remembered that the highest part of the spinal accessory and the hypoglossal nuclei are contiguous, the former behind, the latter in front of the central canal of the cord. Palsy from chronic degeneration is often associated with wasting in other muscles. Degenerative paralysis of the larynx occurs sometimes in disseminated sclerosis, in general paralysis of the insane, and especially in locomotor ataxy. After diphtheria, also, the larynx may be paralyzed, probably in consequence of central changes. The palsy in most cases is bilateral, and is often abductor; it is unilateral only in rare instances.

(*b*) Damage to the roots of the nerves is also an occasional cause, commonly of syphilitic origin, sometimes the result of an aneurism or other simple tumor. Paralysis from this cause is usually one-sided, and often affects half the tongue and palate as well as the vocal cord.

(*c*) The long course of the nerve trunks exposes them to damage from many morbid processes and injuries, which have been enumerated in the section on general causation. The superior laryngeal nerve is far less liable to suffer than the recurrent. Disease of the latter is the most frequent cause of laryngeal palsy. Aneurism of the vessels round which the nerves turn frequently compresses them, and the left suffers from this cause more frequently than the right, on account of its course round the aorta. Other causes of pressure are growths, and enlarged glands in the thorax, cancer of the œsophagus, and enlargements of the thyroid. Paralysis has been met with in some cases of chronic lung disease, and has probably been produced through the agency of enlargement of the glands; the paralysis has been unilateral or bilateral. In all these cases, except disease of the superior laryngeal, many muscles are of necessity paralyzed, although the resulting symptoms are not always the same.

(2) The laryngeal muscles are often weakened by processes that, involving no organic change in nerve or centre, and passing away completely in many instances, are termed "functional." The morbid process may be central or local in its conspicuous relations, although we cannot affirm that the conspicuous relation always corresponds to the seat of the morbid process. The chief central causes are hysteria, and the nervous weakness that attends anæmia and follows prostrating maladies. The local causes are congestion and inflammation of the mucous membrane, and over-use of the larynx, especially in public speaking. We cannot altogether separate the two classes, since local and central disturbance may coincide in cases that appear to be of local origin; hysterical aphonia, for instance, is frequently excited by a transient laryngeal catarrh. Paralysis of the larynx has been thought to be occasionally reflex in origin. Thus, abduction palsy, necessitating tracheotomy, has developed without traceable cause during pregnancy and has passed away after delivery.*

(3) Paralysis of a single muscle on one side results only from local disease affecting the minute branches to the individual muscles. Laryngeal growths, cellulitis, and ulceration of the cartilages are occasional causes; but palsy of this nature is not common. Cold has been supposed to excite a rheumatic neuritis of these branches, but the evidence of this is not conclusive.

Symptoms.—The larynx is the organ of voice and the gate of the air passages. By the aid of the laryngoscope its action in these two functions can be directly observed. The evidence of paralysis is correspondingly threefold. (1) Voice may be changed or lost; (2) the entrance of air in respiration may be impeded; or the closure of the glottis in cough may be impaired; (3) the defective movement can be, in part at least, directly observed. The phonic and respiratory functions are subserved by the same muscles and the same nerves, but by centres that no doubt differ in anatomical connection if not in locality.

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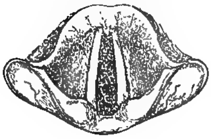
* Aysaguer, *L'Union Méd.*, March 31st, 1885.

The vocal cords assume after death a position of slight abduction from the middle line—a little nearer together than they are during ordinary breathing. This position must, therefore, be regarded as that of muscular relaxation—of the rest that, during life, they may approximate, but never actually attain. During phonation the cords are brought together and made tense; the degree of approximation and tension varies according to the note produced. In breathing, the cords are separated during inspiration, the extent of abduction being proportioned to the force of the inspiration; during expiration they are brought a little nearer together than the cadaveric position.

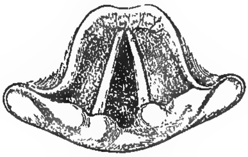
If there is complete paralysis of all the muscles, or (what is the common condition) of all except the crico-thyroids, the vocal cords assume the cadaveric position, from which they cannot be moved (Fig. 277). They are not approximated on an attempt to phonate, nor do they recede on deep inspiration. Hence, vocal sounds cannot be produced. In deep inspiration the current of air may bring the cords a little nearer together than normal, and may cause slight stridor. Instead of the natural explosive cough there is only a sudden rush of air through the glottis. If only one cord is paralyzed, this cord alone is motionless in the "cadaveric position" (Fig. 278), the other moves normally. Some phonation may still be possible, because the unaffected cord may be over-adducted beyond the middle line, but the voice is low pitched, and often hoarse. The abduction of the healthy cord during inspiration prevents stridor. But, unless the palsy is slight, the glottis cannot be closed with sufficient firmness to effect an explosive cough. Complete paralysis is met with in central disease, in disease of the trunk of the vagus, or in disease of the recurrent laryngeal. The escape of the crico-thyroid in the latter case does not materially modify the condition of the larynx.

FIG. 277.



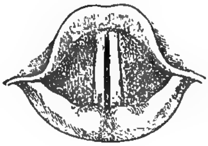
Total palsy of both cords.
Cadaveric position.

FIG. 278.



Similar palsy of left vocal cord.

FIG. 279.



Bilateral abduction paralysis.

In other cases of paralysis, instead of complete loss of movement, with the cords in the cadaveric position, they are nearer together and cannot be abducted, even as far as the cadaveric position (Fig. 279). They can be brought closer together in phonation and cough, and when the effort ceases they recede a little, but the normal, wide recession during inspiration does not take place. There is paralysis of the abductors; the posterior crico-arytenoids. The slight recession that may occur is due to the elasticity of the attachments of the cords. The position of the cords, and the amount of recession, depend on the duration of the paralysis. The adductors, unopposed, undergo secondary contracture, and with this the glottis becomes permanently narrower, until there may be only, at widest, a narrow chink between the cords. The tensors are still active as well as the adductors, and hence voice is little affected. The cords are indeed always in the position for phonation. The chief symptom is the effecton respiration.* The normal recession of the cords during inspiration does not take place, and they are even brought still nearer together by the pressure of the in-rushing air. Hence, inspiration is accompanied by a whistling stridor, often very loud; the hindrance to the entrance of air brings into action the extraordinary muscles of respiration, and inspiration occupies a longer time than normal. On account of the shape of the cords expiration is unimpeded; the current of air even tends to separate them. The absence of any affection of the voice often misleads the diagnosis, and the obstruction is referred to the trachea. But the symptoms differ from this in the absence of expiratory stridor, and the movement of the larynx up and down during breathing is greater than is ever seen in tracheal stenosis. The symptoms are indeed so characteristic that the diagnosis can be made with certainty, even without the aid of the laryngoscope, and they are so striking that, once witnessed, they can scarcely be mistaken, and can never be forgotten. The urgent dyspnœa and loud stridor, accompanied as they may be with lividity of the face and coldness of the extremities, are alarming. The danger is not apparent only; the slightest catarrhal swelling of the cords suffices to occlude the narrowed glottis, and prompt laryngotomy alone may save the patient from death by suffocation. This account is true, however, only of bilateral palsy. If the paralysis is unilateral, the affected cord is near the middle line and motionless; the other recedes during respiration, sometimes to a greater degree than normal; the symptoms are slight and may even be absent. The recession of the unaffected cord prevents both stridor and dyspnœa.

This paralysis of abduction may be due to central disease or to local causes. It has been known to follow a simple laryngeal catarrh. In some cases its etiology is uncertain. Both posterior muscles have been found destroyed by degeneration when all the other laryngeal muscles were healthy. It is possible that these obscure cases are also of central origin. But a similar paralysis of abduction may be produced by disease of the recurrent laryngeal, although this nerve contains fibres for the adductors as well as the abductors. Instead of the cadaveric position of the cord and the complete immobility found in many cases of disease of the recurrent, the cord is near the middle line; further adduction is still possible, but there is no abduction, and if both nerves are affected the symptoms above described are present in characteristic degree. Much ingenuity has been expended in attempting to explain why the abductors should suffer chiefly from disease, such as the pressure of an aneurism, which presumably affects all the fibres of the nerve. Some have suggested that the adductors must be supplied also by the superior laryngeal, an hypothesis unsupported by anatomy. It has also been suggested that the fibres for the abductors may be superficial in the nerve, and so suffer first, or have some mysterious special proclivity to disease.* Others have remarked that the abductors suffer most, just as the abductors and extensors of a limb suffer more in hemiplegia than the adductors and flexors—a doubtful analogy which affords no explanation.

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* Hence this palsy was termed by Türck "respiratory paralysis," as distinguished from "phonic paralysis," in which the voice is affected, while the conditions in which both functions are disturbed have been termed "mixed paralysis." This somewhat loose division hardly deserves the wide adoption it has obtained.

The fact that disease of the recurrent laryngeal, affecting all fibres apparently equally, may influence abduction more than adduction, must be taken in connection with another fact, that electrical stimulation of the recurrent nerve causes adduction of the cord. In the former case we are justified in presuming that all the muscles supplied under-act, in the latter case it is certain that all over-act; in each case there is adduction. It is probable that the explanation will be found in the relative bulk and arrangement of the two sets of fibres. The adductors are more numerous than the abductors, are probably more bulky, are certainly more varied in arrangement, and perhaps act at greater mechanical disadvantage. The direction of the fibres of the posterior crico-arytenoids seems less favorable to their action on the arytenoid cartilages than that of the lateral muscles, and when the cords are adducted the relative disadvantage of the posterior muscles must be increased. Thus an equal reduction of absolute power may cause a far greater impairment of the abductors than of the adductors. Hence it is better to speak of this condition as an impairment or loss of abduction, rather than as paralysis of the abductors. The abduction palsy with narrow glottis probably results from damage to the recurrent nerves, which is incomplete in degree, although not necessarily partial in distribution. The complete palsy with cadaveric position of the glottis is the consequence of complete paralysis of the nerve. Thus, the latter has been observed to follow the former as the disease progressed (Schech, Rosenbach).

It has been suggested (first by Riegel) that the adduction in disease of the recurrent might be the result of secondary contracture in the unopposed crico-thyroid, which, as a tensor and elongator of the cords, must tend to bring the cord and vocal process into a straight line. The chief difficulty in accepting this explanation is that the adduction is not invariable, as it should be were it the result of a secondary contracture of the unopposed crico-thyroid. Nevertheless, secondary contracture may play a part in the phenomena of some cases. If the narrow glottis ever persists after palsy has become complete, it may be from secondary contracture and shortening of the adductors, perpetuated by tissue changes in them.

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* See on this subject Semon, *Archives of Laryngoscopy*, *1881*, vol. ii, No. 3.

Disease of one recurrent nerve usually affects only the corresponding vocal cord. Paralysis of both cords, when produced by organic disease, is usually due to disease of both recurrent nerves or to central changes. But three cases are on record in which both cords were paralyzed, although only one recurrent nerve was found to be compressed or otherwise diseased.* In two cases the paralysis was greater on the side of the lesion than on the other. The paralysis of the opposite cord is supposed to be due to an influence on the centre.

Paralysis of abduction also occurs in hysteria, although rarely; it is always bilateral. The characteristic symptoms are present, inspiratory stridor, often intense, with unimpaired phonation. Most recorded cases have recovered, but the inspiratory dyspnœa may be alarming in its intensity, and it is probable that death has occurred in more than one instance. In a case of morphia habit and hysteria† the patient suffered from loud inspiratory stridor, absent during expiration, not interfering with phonation, and due to abductor paralysis. It disappeared entirely while the patient was under the influence of morphia, but reached an alarming intensity when morphia had been withheld for twelve hours, the stridor being audible all over the house. Amaurosis and some mental derangement coincided with the stridor. She recovered rapidly under treatment, which consisted in a modification of the Weir-Mitchell system, the gradual withdrawal of morphia, and the hypodermic injection of strychnine. The abductor paralysis has been mistaken for spasm, on account of the stridor that accompanies it; but the circumstance that expiration is noiseless, or nearly so, and speech unaffected, effectually distinguishes it from spasm. It is probable that many cases of so-called hysterical spasm of the glottis have been really abductor palsy.

In adductor paralysis the cords are apart and cannot be brought together. The state in which the cords are in the cadaveric position, and cannot be moved, should not be spoken of as adductor paralysis, because, although these muscles are paralyzed, all the other muscles are paralyzed also. The term should be applied only to the cases in which there is still the power of abduction in deep inspiration, but no power of bringing the cords nearer together than the cadaveric position. Thus defined, adductor paralysis is rarely due to organic disease of the nerves or centres.‡ It is most common as a partial paralysis; the cords are not brought together in phonation, and the patient is therefore voiceless, but they can be brought together in coughing. Hence this has been termed "phonic paralysis" (Türck). It is common in hysteria, in which it causes the so-called "hysterical aphonia," and it results also from over-use of the voice, and from catarrhal laryngitis.

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* Bäumler, Johnson, Sommerbrodt. The case of Sommerbrodt was one of traumatic damage.

† Seen with Mr. W. L. Winterbotham, of Bridgewater. The diagnosis of the laryngeal condition was confirmed by Dr. Morell Mackenzie.

‡ Navratil, *Berlin. klin. Wochenschrift*, *1869*, Nos. 36 and 37. The same fact has been recently emphasized by Dr. Semon, *Arch. of Laryngology*, loc. cit.

It is also said sometimes to result from cold without laryngitis (Bose). In hysterical aphonia the patient is sometimes able to sing, although she can only speak in a whisper (v. Bruns).

Another form of partial adductor palsy is due to loss of power in the arytenoideus; the result is defective closure of the posterior part of the glottis, and hoarseness or loss of voice. Little is known of the symptoms and causes of paralysis of the tensors of the vocal cords, beyond the fact that palsy of the internal fibres of the thyro-arytenoideus causes the edge of the cord to be concave. The effect on the voice is similar to that of the preceding palsy, and the two are sometimes associated.

For the exact diagnosis of these laryngeal palsies the examination of the larynx by means of the laryngoscope is essential. But it may be useful to compare the symptoms presented by the chief forms of paralysis, since they often suggest very clearly the nature of the affection. In the following table the symptoms are enumerated in the first column, the condition seen with the laryngoscope in the second, and the form of paralysis in the third. The inability to effect an explosive cough is of great significance as evidence of a palsy of organic origin, in the absence, of course, of organic disease of the larynx. Entire loss of voice, as well as of cough, suggests bilateral palsy of grave organic nature, but without loss of cough it indicates unimportant adduction palsy. Loss of cough without loss of voice suggests paralysis of one cord. Loud inspiratory stridor without loss of voice means abduction paralysis.

SYMPTOMS.	SIGNS.	LESION.
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive, little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Anæsthesia of the larynx is rare. It results from disease of the superior laryngeal nerve, or the roots of the vagus; it is occasionally met with in degenerations of the medulla, but is then usually incomplete. It may form part of hemianæsthesia of hysterical or cerebral origin. In the latter case reflex action is unimpaired; in disease of the nerve or medulla it is lost. General anæsthesia about the entrance to the larynx is said to have been met with in hysterical aphonia.

SPASM OF THE LARYNX.

The common form of spasm of the laryngeal muscles is that of the adductors. The muscles that close the glottis are more powerful than those that open it, no doubt because firm closure is necessary for the process of coughing, and in order to fix the thorax during muscular effort. Moreover, the reflex mechanism is connected chiefly with the glottis closers, doubtless to protect the air passages from foreign bodies or to aid in the expulsion, by coughing, of any irritant substances that have gained an entrance. Hence, any nerve irritation, direct, central, or reflex, causes closure, and it is not surprising that spasm accompanies a large number of laryngeal diseases, varying in its prominence according to the irritative nature of the disease and the excitability of the reflex mechanism. The latter is far more intense in children than in adults, and hence in them the slightest laryngeal catarrh gives rise to spasm. Attacks occur especially at night, when the reflex mechanism, released by sleep from the control of the higher centres, is in its most active state. Spasm may occur from irritation, not only of the superior laryngeal nerve, but also from that of the vagus below, as when the latter is compressed by a tumor in the upper part of the chest; the afferent impression is due to irritation of the sensory fibres from the trachea. Reflex spasm is always bilateral. Direct spasm from irritation of one recurrent laryngeal usually involves only one vocal cord, but in a few cases spasm so excited has been bilateral. This may be explained either by the irritation of some afferent fibres, or (according to Krishaber) by spasm of the arytenoideus, which is a bilateral muscle.

Simple spasm occurs in rickety children, in whom the nervous system is in a condition of excessive reflex excitability. In this form, which is termed "laryngismus stridulus," the vasomotor and cardiac centres of the medulla are also deranged; the child, on some exciting cause, as a start, a peripheral impression, or even without apparent cause, suddenly turns pale, is unable to get its breath for a few seconds, and then, the spasm relaxing, air is drawn through the slowly-opening glottis with a crowing noise. Paroxysmal attacks of laryngeal spasm sometimes occur in adults, usually in the night. They are apparently analogous to attacks of asthma, the spasm affecting the larynx instead of the bronchial tubes. The sufferer wakes up with a feeling of suffocation, intense difficulty of breathing, and loud laryngeal stridor, which after a few minutes passes away. During the duration of the spasm the distress may be extreme, the patient tears open his clothes, and may seem at the point of death. Such occasional attacks have been known to recur from time to time during many years. They have been known to replace attacks of migraine (Liveing), and are occasionally met with in the subjects of locomotor ataxy, in whom they have been termed "laryngeal crises" (see p. 301).

The paroxysms of laryngismus stridulus probably differ only in degree from the general convulsions that are also common in rickety children. In most epileptic convulsions, laryngeal spasm also occurs, and determines the character of the initial "epileptic cry." During the paroxysms of hydrophobia there is also spasm of the glottis.

Lastly, spasm of the larynx is met with in certain general neuroses, in tetany* rarely, in hysteria occasionally. In the latter it may occur in paroxysmal or more continuous form. The paroxysmal form constitutes one variety of hysteroid convulsion. Instances of this are described in the chapter on hysteria. The continuous form is very rare. There is stridor with inspiration and expiration, the voice is feeble, and there may be hysterical rapid breathing. The diagnosis from hysterical abduction paralysis rests on the fact that the stridor in spasm accompanies inspiration as well as expiration, and the voice is more altered than in abduction palsy, in which also the loud whistling inspiration contrasts with the almost noiseless expiration. The laryngeal symptoms usually partake of the character of the other disturbances that may be present; the spasm is accompanied by other spasmodic or convulsive symptoms; the paralysis, by loss of power or of sensibility. It is probable that some cases of supposed spasm have been really instances of abduction paralysis.

A rare condition of functional spasm has been described,† in which spasm is excited by attempts to speak. It is, so to speak, the converse of phonic paralysis. In the latter the cords cannot be brought together in speaking; in the functional spasm they are brought together too forcibly. Either the patient cannot speak or speaks at first in an altered voice, which ceases altogether when a greater effort is made, on account of the increased spasm that the effort induces. Apart from attempts to speak there is no laryngeal disturbance. It has been termed "spastic aphonia" or "phonic laryngeal spasm" by Schnitzler, "spastic dysphonia" by Schech, and "coördinated laryngeal spasm" by Nothnagel, because he observed it to accompany other voluntary movements of the larynx besides those of speech, while it was absent in all involuntary and automatic movements. The spasm may be attended with pain in the larynx and even in the upper part of the thorax (Jurasz). The affection has been compared to writers' cramp, but differs from this in its general etiological relations, and in its greater amenability to treatment. A closer analogy to writers' cramp was presented by a case reported by Gerhardt, in which the patient had actually suffered from writers' cramp and, at the age of fifty, learned to play the flute. The act of blowing the flute brought on laryngeal spasm and an unintended voice sound, accompanied by muscular contractions in the arm and angle of the mouth.

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* Killian, *Monatsschr. f. Ohrenkr., etc., 1884.*

† Schnitzler (1875), Schech (1879), Nothnagel (1881), Fritsche, Jurasz (1880).

PULMONARY BRANCHES.

The influence of general disturbance of the pneumogastric on the respiratory movements, and the spasm that results from irritation of the afferent pulmonary nerves, have been already described. The muscular fibres of the bronchi are supplied by this nerve, and their paroxysmal contraction in asthma has been thought to be produced through its agency. It has also been asserted that the plain muscular fibres, said to exist in the pulmonary tissue, are supplied by the pneumogastric (Gerlach), and their contraction has been assumed to explain a peculiar form of emphysema observed in a case of compression of the pneumogastric (Tuczek); but the compression caused also deep breathing of a costo-superior type, and the emphysema may have been merely the result of the energetic movement consequent on the stimulation of the respiratory centre. The pneumogastric is commonly believed to contain vasomotor fibres for the vessels of the lungs, but Brown-Séquard and Franck have separately shown that these fibres are contained, not in the vagus, but in the sympathetic. Vascular lesions of the lungs have, however, been observed after section of the vagus. Michaelson noted rapid congestion and hemorrhage; it is possible that this may have been of reflex origin, produced through the agency of the sympathetic. But in man, acute lesions of the pons sometimes causes rapid vascular changes in the lungs; in one case of hemorrhage into the pons, fatal in two hours, I found intense congestion with extravasation into the left lung, and hemorrhages in the left extremity of the stomach. After section of the vagus, animals die from chronic pneumonia, and hence the vagus has been supposed to be a trophic nerve for the lungs. But the changes have been accounted for by the entrance into the bronchi of food from the pharynx, in consequence of the obstructive paralysis of the œsophagus, and the paralysis of the larynx (Traube, Steiner). All admit that this is one cause of the pulmonary affection, but opinions differ as to the extent of its influence.

CARDIAC BRANCHES.

The inhibitory effect of irritation, and the acceleration of the heart's action that results from lessened action of the vagus, have been before alluded to. Increased frequency has been several times observed in cases of local disease of the vagus in the thorax, compression by mediastinal tumors, etc. In a case of phthisis, for instance, in which the pulse was at first occasionally, and afterward constantly, frequent (130–148), Meixner found the left vagus enclosed in a mass of enlarged glands in the upper opening of the thorax. The vagus is also the afferent nerve from the heart, and although we are normally unconscious of the cardiac action, some of the disordered sensations of disease are apparently produced through its agency. In some anginal attacks the heart's action is, for a time, arrested or retarded, and in a few cases these symptoms have been found associated with organic disease of the cardiac plexus. Thus, in a case in which, during paroxysms of intense anginal anguish, the heart's action was arrested for four or six pulsations, Heine found a tumor involving the cardiac plexus. In a case recorded by Blandin anginal attacks were associated with a small tumor of the vagus. Further, there are afferent fibres from the heart, inhibiting the action of the vaso-motor centre, and these are probably disturbed in some anginal seizures.

After disease or injury of the vagus, the heart has been found in a state of fatty degeneration, and hence it has been thought that the vagus contains trophic fibres for the cardiac substance.

GASTRIC BRANCHES.

The branches to the œsophagus are rarely diseased except in cases of affection of the nerve trunk or of the centre. In very rare cases such disease has caused difficulty in swallowing, simulating stricture. Spasm of the œsophagus is far more frequent. The vagus is the sensory nerve for the stomach. Its fibres are very sensitive to any local irritation, and not rarely the seat of spontaneous neuralgia. Hunger is generally believed to be a pneumogastric sensation, and complete loss of the sensations of hunger and thirst was noted in a case of softening of the root of the vagus from an aneurism of the vertebral artery (Johnson). Appetite, however, is not always lost in animals when the pneumogastrics have been divided (Reid). In some cases of disease of the nerve excessive appetite has been present. This symptom, for instance, was noted in one case, in conjunction with dyspnœa, noisy breathing and vomiting of unaltered food; post-mortem both pneumogastrics were found atrophied (Swan). In another case of insatiable appetite, small neuromata were found on the nerve. It is possible that the symptom may be partly the result of the defective digestion of food.

The pneumogastric is also in part the motor nerve of the stomach; after its section the contractions of the organ are lessened, although not altogether arrested. Vomiting is probably produced through its agency, by varied reflex and central irritation. In the latter case (as in meningitis) the vomiting is sometimes extremely rapid. I have known paroxysmal vomiting to result from the intermitting pressure of a tumor on the vagus; and Boinet, having exposed the vagus in an operation in the neck, noted that whenever he touched the nerve the patient vomited.

The vagus accelerates the contraction of the intestines, but no intestinal symptoms have been observed from its disease.

Prognosis.—The prognosis in every case depends chiefly upon the nature of the disease that interferes with the function of the nerve. It is good only when the disturbance is not due to organic disease. In the diseases in which the local symptoms form part of a wider functional disturbance, as in the case of hysteria, the prognosis is described in the account of those diseases.

Diagnosis.—The chief symptoms on which the diagnosis of disease of the vagus, in any given case, depends, are the laryngeal paralysis, retarded respiration, accelerated or retarded action of the heart, and vomiting. The diagnosis of the seat of the disease rests upon the distribution of the symptoms, and on the associated disturbance. Disease of the trunk of the vagus is much less common than disease of its branches or roots. Paralysis of one vocal cord, for instance, is almost always the result of pressure, either on the recurrent laryngeal or on the roots of the spinal accessory at the medulla. In the former case there are indications of disease in the thorax; in the latter other nerves suffer, especially the hypoglossal. Bilateral paralysis of the larynx, if considerable, suggests central disease—degeneration of the nuclei in the medulla; if slight, it may be of local origin and independent of a lesion of the nerve itself, as in the case of the "phonic" adduction palsy. Disease of both recurrent laryngeals is very rare, but it must be remembered that disease of one recurrent has been known to influence both cords. In most cases of pressure on the nerve, the compressing disease causes other obtrusive indications of its presence and position, the chief exception being deeply-seated tumors of the thorax.

TREATMENT.—The chief element in treatment is to remove the morbid process by which the damage to the nerve is produced; the measures to be employed must vary according to the nature of the process, and have been already fully described in connection with the other cranial nerves. The frequency with which a lesion of the nerve roots is due to syphilis must be especially remembered. Central degenerative processes are, for the most part, beyond the influence of drugs; their treatment, as far as it is practicable, is described in the section on glosso-labial paralysis.

The chief division of the pneumogastric for which special treatment may be necessary is that for the larynx. The causes of organic damage to the recurrent nerves are generally grave progressive diseases, such as cancer or aneurism, beyond the effective range of medicine or surgery. The fact that scrofulous enlargement of lymphatic glands is an occasional cause of compression of the recurrent nerves should be remembered, and cod-liver oil, iodide of potassium and arsenic should be given whenever it is probable or even possible that the disease is of this nature. Moreover, here also syphilis may be at work.

In paralysis secondary to inflammation of the larynx or to cold, the appropriate treatment for the laryngeal disease should be combined with stimulating applications or blisters to the exterior of the larynx. The insufflation of strychnine has been recommended, but the amount absorbed is uncertain and variable, and dangerous symptoms have ensued. It is probable that all the good which strychnia can accomplish is to be obtained from its administration by the stomach or the skin. Hypodermic injections are unquestionably useful in laryngeal palsy; gr. $\frac{1}{60}$—$\frac{1}{30}$ of the nitrate may be injected daily. It is of especial value in hysterical paralysis, in phonic paralysis and in diphtheritic palsy.

Electricity has been used in various ways: (1) One electrode has been introduced into the larynx and placed near the paralyzed muscle, with the guidance of the laryngoscopic mirror, the other electrode being placed outside the larynx. (2) A double laryngeal electrode has been used, with two small rheophores close together. (3) One electrode has been placed in the pharynx at the back of the larynx, the other outside. (4) Both poles have been placed outside the larynx, and the current passed through it. The intralaryngeal application, even in the most skillful hands, is not pleasant to the patient, and can only be borne for a few seconds at a time. The percutaneous method is painful, although far less so than the other; a stronger current can be borne, and for a longer time, and if the current cannot be localized with the same precision, the muscles can be more effectually stimulated. The positive rheophore may be pressed behind the jaw, and the negative pressed firmly and moved down the side of the larynx and trachea. In thin persons, at least, the superior and inferior nerves may be thus directly stimulated. Either faradaism or voltaism may be applied externally; in organic disease of the nerve the latter only will influence the muscle. But the value of electricity in organic disease of the nerves is probably not great. In functional palsy, phonic paralysis and the like, the nerves not being degenerated, faradaism can be employed and is often distinctly useful. It is highly probable that the chief results obtained by electricity are due to the stimulation of the sensory nerves, and to the indirect influence thus exerted on the centres.

In the gravest form of laryngeal palsy, paralysis of the postici, the utility of electricity is practically limited to hysterical cases, and even in them it must be employed with some caution. The separate stimulation of the postici can only be effected by the intrapharyngeal method, the electrode being placed at the back of the cartilage, a little on one side of the middle line. Any other method of application will stimulate the adductors far more than the abductors, and so increase the perilous constriction of the glottis. Even the intrapharyngeal method is not free from this danger. All stimulation of the sensory nerves has a reflex action chiefly on the adductors, by virtue of the central reflex mechanism for guarding the air passages against the entrance of foreign bodies. Hence, even the intrapharyngeal application is in danger of doing more harm indirectly than good directly.

In some cases of phonic paralysis, careful laryngeal gymnastics have been found of service, as in making the patient utter or sing certain simple vowel sounds without complicating articulation. Treatment by manipulation of the larynx was proposed some years ago by Oliver,* and has been found useful by Gerhardt and others. It consists in pressing firmly with the thumb and forefinger on each side of the thyroid cartilage, at the upper and hinder part. During the compression the patient is made to utter a simple sound, and then is often able to speak, at first in a weak, and afterward in a stronger voice. Vocalization once effected, the patient is often able to continue to speak. Laryngoscopic examination during the compression shows that the manipulation approximates the arytenoid cartilages and the vocal cords, and at the same time makes them tense. The treatment of hysterical aphonia is described in the chapter on that disease.

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* *American Journal of Medical Science*, April, 1870, p. 305.

In the treatment of *laryngeal spasm* the most important thing is the removal of the condition on which it depends, by the treatment of any local laryngeal irritation and of any diathetic state which exalts the irritability of the central nervous system. Bromide of potassium or ammonium at once lessens the irritability, and cocain applied as a spray has a like action, but these are, in most instances, palliative only. Useful as they are in removing spasm for a time, it is necessary also to strengthen the nerve centres by tonics in order to prevent a recurrence. The spasm is usually removed for a time by the inhalation of chloroform, and often by nitrite of amyl. Ten-grain doses of chloral have been recommended by Johnson as a substitute for chloroform, but chloral is, as a rule, inferior to bromide. Spasm due to local laryngeal irritation is often relieved by a necklet of ice; a long narrow tube is made with gutta-percha tissue, the edges being stuck together by means of chloroform, and this is filled with small pieces of ice and placed round the neck.

SPINAL ACCESSORY NERVE.

(EXTERNAL PART.)

Of the two parts of which the spinal accessory nerve consists, the "accessory" part (which arises from the medulla oblongata, and, joining the pneumogastric, supplies the larnygeal muscles) has been already described. The "spinal" portion, as it is termed from its origin, or "external portion," as it is sometimes called from its distribution, is virtually a series of fibres of the motor cervical nerves, that have the unusual course of ascending to the cranial cavity and leaving it again with one of the cranial nerves to be distributed to the cervical muscles. The root fibres arise from the middle of the lateral column of the cord, but the fibres pass through this, and spring from the nerve cells of the anterior cornu, just as do the anterior roots of the cervical nerves. As the fibres have the same origin as the motor cervical nerves, so they have the same distribution—to two cervical muscles, the sterno-mastoid and trapezius, which are supplied in part by this nerve, and in part by the cervical nerves. The nerve usually perforates the sterno-mastoid, and supplies this muscle almost entirely, the other nerves to it being unimportant branches from the second and third cervical pairs. The fibres can be traced almost down to the lower border of the trapezius, but this muscle receives a larger supply from the cervical and upper dorsal nerves. Only paralysis of the nerve is here described. The muscles supplied by it are frequently the seat of spasm, which produces the condition known as "torticollis." But the spasm often passes beyond the distribution of this nerve, and is therefore more conveniently considered, in connection with other forms of spasm, at a later page.

Causes.—The nuclear gray matter from which the nerve arises may participate in central degeneration, causing wasting in these muscles, associated with more extensive muscular atrophy (see p. 350). The nerve may be damaged in the neighborhood of the foramen magnum by local meningitis and compression, and both nerves may be thus affected. At the side of the medulla the external part often suffers with the hypoglossal and with the fibres for the larynx. Outside the skull it may be damaged by wounds, by deep-seated tumors, by caries of the higher cervical vertebræ, and by abscesses springing from the cervical glands, and sometimes, although rarely, by rheumatic neuritis.

SYMPTOMS.—The effect of disease of the nerve is paralysis of the muscles supplied by it. If the trunk of the nerve is diseased, the sterno-mastoid suffers much more than the trapezius, and the latter chiefly in its higher parts. Wasting almost always accompanies the loss of power. The paralysis of the sterno-mastoid is shown by an absence of the normal prominence of the muscle in movements of the head, and by defective power of rotation of the head to the side opposite to the paralysis. Paralysis of one sterno-mastoid does not cause any deviation of the head when at rest, but such deviation may arise from secondary contraction of the unopposed muscle on the other side. There is no such thing as a pure paralytic torticollis.

The only part of the trapezius that is completely paralyzed by disease of the spinal accessory nerve is the highest portion, which descends from the occipital bone to the acromion. Instead of the nearly straight contour which this muscle gives to the outer side of the neck in the normal condition, the neck presents a concave curve, and the difference between the two sides is brought out strongly by a deep inspiration, the action in which this muscle is chiefly employed. The weakening of the second part of the trapezius allows the shoulder to fall a little; the scapula recedes from the spine and is rotated, the lower angle inward, in consequence of the unopposed action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is also impaired, because the deltoid has lost some of the support from which it acts. But the middle part of the trapezius is never completely paralyzed from disease limited to the accessory nerve, by reason of the additional innervation from the spinal nerves.

In bilateral paralysis of these muscles the power of supporting the head in the upright posture is impaired. If both sterno-mastoids are affected, the head tends to fall backward; if both trapezii, it readily sinks forward, so that the chin rests on the sternum. Such defective power of support of the head is not uncommon in children in consequence of chronic meningitis about the foramen magnum, damaging both spinal accessory nerves; and it is conspicuous in many cases of progressive muscular atrophy (Fig. 128). In recent cases of injury to the nerve, the muscles present the characteristic reaction of nerve degeneration. In central disease the reaction varies, as it does elsewhere in progressive muscular atrophy.

The distribution of the symptoms differs according to the seat of the disease. When this is in the central gray matter other muscles are always involved, and the distribution of the paralysis in the region of the spinal accessory varies much. As a rule, the highest part of the trapezius suffers later than the rest of the muscle, and for this reason Duchenne called it the *ultimum moriens*. But this rule is not invariable; I have known this part to be the first to suffer. In disease of the trunk of the nerve all parts are involved. Not infrequently the nerve is damaged, by local disease or injury, after it has passed through the sterno-mastoid, and then this muscle escapes, and the trapezius alone suffers. When the lesion involves the intracranial or jugular part of the nerve, it is common for the internal or accessory portion to be likewise affected, and there is then paralysis of the vocal cord on the same side. If the lesion is within the skull the hypoglossal is frequently damaged also, and sometimes the palate (see p. 706).

Treatment.—The treatment of paralysis of the external part of the spinal accessory is, first, that of the morbid process by which it has been damaged, and, secondly, the stimulation of the paralyzed muscles by electricity for the purpose of maintaining or improving their nutrition during the recovery of the nerve. The current must be used to which the muscles most readily respond.

HYPOGLOSSAL NERVE.

The hypoglossal nerve, the motor nerve for the tongue and for most of the muscles connected with the hyoid bone, arises from the medulla oblongata beside the olivary body. Its origin has been already described (p. 482). The fibres within the skull are close to those of the spinal accessory and pneumogastric, a relation that is resumed for a short distance in the upper part of the neck, after the passage of the nerve through a separate foramen in the occipital bone. The nerve, however, soon leaves its deep position to course forward to its distribution. Its most important connection is with the petrous ganglion of the pneumogastric. The function of the nerve is purely motor.

PARALYSIS.

Causes.—(1) Nuclear disease is usually degeneration, rarely acute softening from vascular occlusion. It forms part of bulbar paralysis, acute and chronic, and has occurred in rare cases of locomotor ataxy. The affection is almost always bilateral, the two nuclei being so near together that even an acute lesion scarcely ever affects one only, but unilateral disease has been met with in degeneration of the nucleus in tabes,* and in a case of acute apoplectiform onset.† The lips and throat suffer also in nuclear palsy. (2) Supra-nuclear disease, involving the motor tract anywhere between the medulla and the lowest part of the ascending frontal convolution, and of any nature, may paralyze the tongue on the opposite side. (3) Infra-nuclear disease: (*a*) within the medulla, the root fibres are occasionally damaged by softening or by a tumor, usually in association with the contiguous motor tract to the opposite limbs; (*b*) outside the pons, the fibres of origin are damaged by meningitis, simple or syphilitic,‡ and by growths. In thickening of the bones of the skull the nerve may be compressed within its foramen. In its course outside the skull the nerve is occasionally damaged by deep-seated tumors, by mischief communicated from caries of the highest cervical vertebræ, or is injured by penetrating wounds. It is rarely the seat of rheumatic neuritis.*

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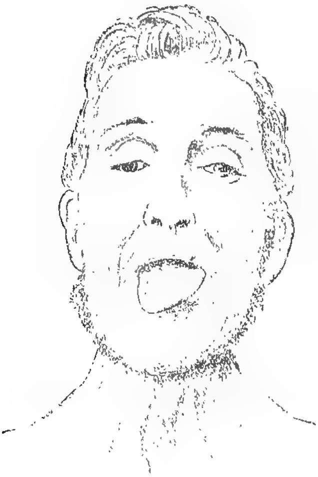
* Raymond and Artaud, *Arch. de Phys.*, No. 3, 1884. The degeneration was proved post-mortem.

† Hirt, *Berl. kl. Wochenschr.*, *1885*, No. 26.

‡ A good example of damage by chronic meningitis is recorded by Hayem and Giraudeau, *Rev. de Méd.*, March, 1883.

FIG. 280.



Paralysis and wasting of the right half of the tongue, due to disease of the hypoglossal nerve.

SYMPTOMS.—The effects of disease of the hypoglossal nerve are motor only; no loss of sensation results. In paralysis of one hypoglossal nerve the tongue at rest is in its normal position in the mouth, but its root is higher on the paralyzed than on the unparalyzed side, in consequence of the loss of the tonic contraction of the posterior fibres of the hyo-glossus. Within the mouth the movement is deficient toward the paralyzed side. When protruded (Fig. 280), however, the tongue deviates toward the affected, and from the unaffected side, because, in protrusion, the tongue is pushed out by the fibres of the genio-glossus, and is pushed toward the weaker side. The point of the tongue is often curved toward the affected half. In complete bilateral paralysis, the tongue lies motionless within the mouth, and cannot be protruded. Articulation is impaired in proportion to the degree of paralysis, but slightly in unilateral palsy, even when this is considerable, and considerably in bilateral palsy even when this is slight. Mastication is hindered, because the tongue fails to keep the food between the teeth, and the loss of the propelling power of the tongue may make it difficult for the patient to get the food into the throat. The nutrition of the tongue varies according to the seat of the disease; when above the nucleus, wasting is absent or insignificant; but when the nucleus or nerve fibres are diseased, the tongue wastes and shrinks. The wasting is of the muscular tissue; the mucous membrane is thrown into conspicuous irregular folds, prominent rugæ with deep furrows between them. Sensation is not impaired, nor is taste except in slight degree in consequence of inability to move sapid substances about in the mouth.

DIAGNOSIS.—The position of the lesion is indicated by the associations of the paralysis. If the disease is in the motor tract above the nucleus (pons, crus, or hemisphere), there is hemiplegic weakness on the side of the paralysis of the tongue. In disease of the nucleus, the paralysis is commonly bilateral, is associated with paralysis of the lips and throat, and there is usually wasting. Disease of the fibres of origin within the medulla is associated with paralysis of the opposite limbs, so that the tongue deviates from the paralyzed side. When the disease is at the surface of the medulla, the paralysis is commonly unilateral, and is associated with paralysis of the corresponding half of the palate and vocal cord. In disease of the fibres of origin within or outside the medulla, there is commonly wasting. The diagnosis of the pathological cause of the paralysis rests on the course of the affection, and on the presence of any causal and associated condition.

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* A case of isolated affection of this nerve in a lad, aged thirteen, possibly a rheumatic neuritis, has been recorded by Erb (*Deut. Arch. f. kl. Med.*, xxxvii, p. 265).

Prognosis.—This is usually unfavorable, on account of the character of the disease, which damages the nerve or centre. Even in syphilitic cases recovery is often incomplete.

Treatment.—The treatment of paralysis of the hypoglossal nerve is that of the causal disease. Tonics, counter-irritation, iodide of potassium and mercury, with occasionally the application of electricity to the tongue, are the most important remedies to be employed, according to the etiological indication. The most convenient method of applying electricity is by means of a tongue depressor in a wooden handle, the blade being insulated by a coating of sealing wax where it comes in contact with the lips.

SPASM OF THE TONGUE.

The tongue participates in certain forms of general spasm, such as those of epilepsy and chorea. The tongue-biting in epileptic attacks is due to the organ being jerked between the teeth by the genio-glossus, when the jaws are brought together by the spasm in the masseters. Spasm occurs also in some forms of stuttering. A peculiar affection, allied to stuttering, in which an attempt to speak brought on spasm in the tongue and muscles attached to the hyoid bone, was described by Fleury* under the name "aphthongia." It is especially excited by emotion, and has been compared to writers' cramp.

The tongue is a not infrequent seat of spasm in hysteria, in which protrusion may occur during paroxysmal convulsive attacks, or rarely without spasm elsewhere. More frequent in this disease is spasmodic deviation of the tongue when it is put out. I have seen this as an isolated symptom in a child, continuing for some weeks, and also in association with some forms of hysterical spasm about the face. Thus, in the case of hysterical ptosis, shown in Fig. 270, the tongue deviated to the left on protrusion, and continued to do so as long as the affection of the eyelids lasted. In another case, a young woman, similar deviation to the left was associated with persistent tonic spasm, by which the left angle of the mouth and the lower lip were drawn downward and outward. It is probable that some other cases of spasm of the tongue, recorded as idiopathic, were due to the same disease. A girl,† aged nine, suffered for some months from attacks, recurring every ten minutes, and sometimes during sleep, in which the tongue was protruded in tonic spasm, and toward the end of the attack the tip was turned upward; if the mouth was shut at the time the tongue was firmly pressed against the teeth. Her own explanation was that she felt an irresistible desire to protrude the tongue. In another instance of a girl suffering from peculiar hallucinations, whenever the tongue was protruded it presented very rapid movements of protrusion and retraction.*

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* Fleury, *Gaz. Hebd., 1865*, No. 16. The case (in a man) was complicated by convulsions, and other indications of cerebral disturbance. It is possible that the symptoms were of functional origin, as similar spasm certainly was in cases (of children) recorded by Panthel (*Deut. Klinik., 1855*) and Vallin (*Gaz. Hebd., 1865*, No. 17).

† Dochmann, *Berl. kl. Wochensch., 1803*, No. 1.

Spasm in the tongue seems to be sometimes caused by irritation of the fifth nerve. In one instance, during each paroxysm of neuralgia in the lingual nerve, there was spasm of the tongue.† In another case, dental caries with ulceration of the gums seemed to be the cause of attacks of spasm, in which the tongue was as hard as a piece of wood, and was curved upward with the tip toward the palate. The spasm afterward spread to the face, neck and arm, and ceased on the removal of the teeth.‡

Paroxysmal clonic spasm in the tongue has been occasionally met with. A man, aged thirty-three, was liable to attacks of protrusion of the tongue, which was pushed out forty or fifty times a minute. Each attack was preceded and accompanied by peculiar sensations in the left side of the tongue and gums. The spasm seemed to be greatest in the left half of the tongue, and it spread to the left side of the face, where there was slight loss of power. Mastication excited spasm; speaking did not. Remak, who recorded the case,§ thought the spasm was probably of cortical origin, and the contiguity of the facial and lingual centres gives support to the opinion. The attacks ceased under iodide and electrical treatment. Somewhat similar paroxysms of clonic spasm were limited to the tongue in a case described by Berger.‖ A sense of tension in the organ preceded each attack, which lasted one or two minutes. The attacks occurred during sleep, as well as by day, and continued during a year and a half. The patient was a woman, aged twenty-eight; she was anæmic, and recovered when treated with iron and change of air. In another case, a man, aged forty, had suffered for two years from attacks in which the tongue was pushed out and drawn back more deliberately. The intervals between the attacks varied from a few hours to several weeks.¶

It seems clear, from these meagre facts, that spasm in the tongue is generally paroxysmal, and dependent on functional states of the nervous system that are removable by tonic treatment. It thus differs essentially from spasm in the face and neck.

PARALYSIS OF THE PALATE.

The movement of the palate that is of chief medical importance is its elevation, which is best seen when the patient utters a long "Ah!"—the base of the palate is then drawn up, so that a depression is formed in the middle line of the upper half of the palate. It is probable that this movement is produced chiefly by the levators, each of which spreads out toward the middle line, and there blends with its fellow. If this movement is lost on one side, when the patient utters the sound the middle of the soft palate is drawn a little toward the unaffected side, and a depression is formed on this side of the middle line instead of in the middle line itself. The difference between the height of the edge of the palate on each side, at rest, is of little significance, since a difference is very common in health ; loss of action of one side of the azygos uvulæ is also seldom to be recognized in cases of unquestionable paralysis of the palate. In bilateral palsy, however, the palate hangs lower than normal, and the uvula is long and flaccid. The effect of palsy of the palate is to permit the regurgitation of liquids, from the throat into the nose, and to interfere with speech by causing a persistent nasal resonance, and by preventing the compression of air necessary for the articulation of the explosive labial consonants. Paralysis of one side of the palate does not usually cause symptoms, and is only discovered by an examination of the throat.

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* Erb, *Krank. der Periph. Nerv.*, 2 Aufl., 1876, p. 296.

† Romberg, *Lehrbuch*, 3 Auf., 1857, p. 388.

‡ Mitchell, *Med.-Chir. Trans.*, vol. iv, p. 75.

§ *Berl. klin. Wochenschr.*, *1883*, No. 34.

‖ *Neur. Centralbl.*, *1882*, p. 49.

¶ Ibid.

The origin of the nerve supply to the palate is one of the most obscure questions connected with the anatomy of the cranial nerves. The levator palati and azygos uvulæ are said to be supplied from the Vidian nerve, and the tensor palati from the otic ganglion of the fifth. The fibres from the Vidian are commonly thought to be derived from the facial nerve, but we have already seen reason to doubt the truth of this opinion. It is certain that the movement of the palate just described, of which the one-sided loss is the most definite and frequent, is innervated from one of the nerves that arise from the medulla oblongata. Whenever disease at the surface of the medulla damages the hypoglossal and spinal accessory nerves so as to paralyze the tongue and vocal cord on the same side, it will almost always be found that this movement of the palate is lost.* It is certain, therefore, that it depends on one of these nerves, or on the glosso-pharyngeal; the intercommunication between the various nerves is so considerable, and the precise origin of the palatine nerves is so uncertain, that a supply from either of these nerve roots is possible.† On the whole, it is perhaps easiest to conceive that the fibres are ultimately derived from the spinal accessory. Fibres from this nerve may pass to the palate by the branch that the pneumogastric gives to the pharyngeal plexus. In some cases of this paralysis the external muscles supplied by the spinal accessory are wasted. The same combination of palsy of tongue, palate and larynx occurs also from disease within the medulla, damaging the bulbar nuclei, and is considered in another section. The causation and treatment of unilateral paralysis of the palate are the same as diseases of the tongue, already described.

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* This fact was first pointed out by Hughlings Jackson, *London Hosp. Rep.*, vol. i, 1864.

† That the spinal accessory is the nerve for this movement of the palate is suggested by S. Mackenzie, *British Med. Journal*, March 3d, 1883.

LOCALIZATION OF CEREBRAL DISEASE.

(RELATION OF LOCALITY TO SYMPTOMS.)

The chief facts regarding the relation of symptoms to locality of lesion have been already incidentally described in the account of the functions of the brain and of the symptoms themselves. It is convenient, however, to recapitulate the various symptoms that are caused by disease in the several parts of the brain, so far as these are known, and are of importance in diagnosis. The variations that depend upon the nature of the lesion will be described in the account of the special diseases. The chief precautions that have to be observed in drawing conclusions from observed facts have been mentioned on p. 457.* It should be remembered that the symptoms, and the way in which they come on, constitute evidence as to the seat of the disease. The mode of onset indicates its nature, and the nature of the lesion is only of localizing significance in so far as some lesions are more common than others in certain parts. But this indication is less frequently of value than is its converse; the seat of the lesion may make one morbid process more probable than another.

CEREBRAL CORTEX.—It is convenient to consider *seriatim* the several regions into which the cortex is commonly divided, beginning with that in which disease most frequently causes conspicuous symptoms.

Central Region, i. e., the ascending frontal and parietal, anterior two-thirds of superior parietal lobule, and paracentral lobule on the inner surface. Destruction causes hemiplegia on the opposite side, permanent, with secondary degeneration of the pyramidal tract, and rigidity of the limbs; a partial lesion affects face, arm, or leg, according to its position (see p. 514).† Partial lesions are very common, first on account of the wide extent of the central region, and secondly, because the region is supplied by different arterial branches, and softening from arterial occlusion is a common lesion. Partial palsy, "monoplegia," is far more common from disease of the cortex than from disease more deeply seated; hence, it always *suggests* a cortical lesion, but does not *prove* it. The leg is probably affected alone only in disease of the medial cortex (paracentral lobule), or when disease of the outer surface is close to the longitudinal fissure. Disease between the longitudinal fissure and the level of the lower frontal sulcus paralyzes the arm,‡ which is affected alone if the lesion is limited to the middle third of the convolutions (Fig. 281). Probably disease at the junction of the ascending frontal and highest frontal paralyzes the foot more than the rest of the leg, and a lesion in the highest part of the arm region affects the shoulder more than the rest of the arm. In drawing conclusions from the preponderant palsy of the extremity of either limb, great care is necessary, because this part is less represented in the other hemisphere than are the muscles of the upper parts of the limbs, and the latter recover some power by compensation (see p. 514). Disease of the ascending frontal, opposite the upper half of the inferior frontal, causes paralysis of the lower part of the face, except the lips, which suffer, with the tongue, in disease of the lowest part of the ascending frontal (Fig. 282). But the bilateral representation of the lips renders the effect of one-sided disease inconspicuous. Disease in the same region sometimes causes transient weakness of the opposite masseter. On the whole, the paralyzing effects of limited lesions of the central region correspond very closely in different cases. Apparent exceptions are sometimes due to the depth to which the disease penetrates. Lesions are seldom confined to the gray substance, and if they extend deeply into the white substance they may interrupt the fibres from another region of the cortex that is not diseased.

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* Ferrier, "Localization of Cerebral Disease," London, 1878.

† The most important writings on the subject of cerebral localization are by Nothnagel, in his admirable and exhaustive "Topische Diagnostik der Gehirnkr.," 1879; Ferrier, "Gulstonian Lectures on Localization of Cerebral Disease," London, 1878; Charcot and Pitres, *Revue de Méd.*, *1877*, *1878*, *1879*, and 1883, and Allen Starr, *Amer. Journ. of Med. Science*, *1884* and 1885.

‡ The arm has been paralyzed by a lesion only a few millimetres from the longitudinal fissure.

Fig. 281.

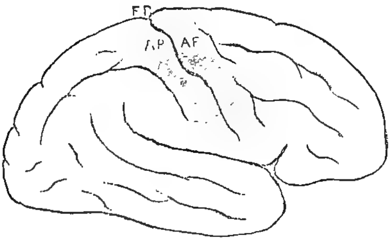


Fig. 282.

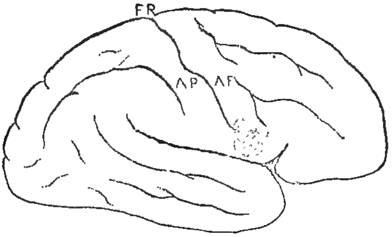


Fig. 281.—Position of a tumor in the middle of the central convolution which caused convulsions beginning in the left arm, and afterward paralysis of the arm without implication of leg or face. (Müller, *Trans. Int. Med. Congress*, London, 1881.)

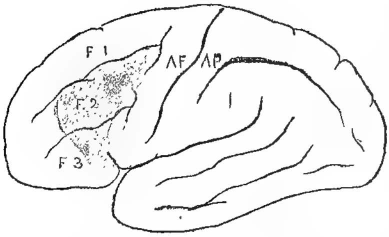
Fig 282.—Position of a small hemorrhage which caused paralysis of the tongue and lower part of the face. (Ballet, *Prog. Med. 1880*, p. 762.)

Irritating disease, and also stationary partial lesions, in the central cortex cause convulsion, which begins locally in the leg, arm, or face, or according as the disease is in or near the region destruction of which causes palsy of the part. Thus, disease of the middle third of the central convolutions often causes convulsions beginning in the hand. According to its degree, the resulting convulsion may be confined to the part whose centre is irritated, or may spread through the whole side, or may be bilateral; the second side being affected after the first (see p. 524). This local commencement is the great characteristic of convulsions from cortical disease. Rarely a lesion elsewhere, either an acute lesion or chronic irritating disease, such as tumor, causes such convulsions. But a stationary lesion of acute onset never causes recurring convulsions unless it is in the cortex. Hence, initial convulsions of this character suggest cortical disease, but recurring convulsions from a stationary lesion prove it. But while these convulsions are thus of great general significance, the indication they afford as to the exact seat of the disease is not always precise; it is less precise than is paralysis; because cortical disease causing local palsy must involve the centre concerned, but such disease causing local convulsion may be only near the centre. The convulsion may begin by a sensation or by spasm, the significant indication is the part in which the discharge commences, irrespective of its nature. Thus, if the fit begins with tingling in the foot, and the sensation seems to pass up the leg and side, and down the arm, and then the hand begins to twitch in spasm, the phenomena indicate disease in or near the leg centre. Further, certain centres seem to be more readily discharged than others, and an influence that exalts irritability in a considerable area may be first manifested in these centres, not because the morbid state is greater in them than elsewhere, but because they respond to it more readily. The centre that influences the movement of the head and eyes to one side is apparently the most sensitive of all the cortical motor centres, because the fits of idiopathic epilepsy commonly begin by this movement; hence, such commencement of a convulsion due to organic disease is of little localizing significance. Persistent tonic or clonic spasm is seldom met with from cortical lesions. Clonic spasm in the face resulted from the small lesion shown in Fig. 275. In a case recorded by Lepine,* a lesion beneath the lower part of the ascending frontal caused trismus during the two days the patient lived. Disease of the central cortex often causes loss of the sense of posture in the part paralyzed, but as this loss also occurs in disease of the motor path in the internal capsule, its diagnostic significance is not great. Cutaneous sensibility is often impaired by disease of the central region, and the sensory paralysis corresponds in seat to the motor palsy, but is always very much slighter in degree, and is chiefly marked on the extremity of the affected limb.† It may be absent, and hemianæsthesia never results from disease limited to this part. Tactile sensibility is more impaired than is sensibility to pain.

There are commonly no definite mental symptoms that can be specially ascribed to disease of this region.

Prefrontal Lobe.—A lesion at the posterior extremityof the upper frontal, at its junction with the ascending frontal, may cause paralysis of the foot, or convulsion beginning in the foot; but with this exception no motor paralysis results from disease of this region, even when it extends up to the ascending frontal. The extensive lesion shown in Fig. 283 produced no palsy. If the movement of the head and eyes to one side is represented in this region, as is suspected from the results obtained by experiments upon animals, the loss in man so quickly passes away by the compensation by the other hemisphere

FIG. 283.



Extensive softening of cortex of left prefrontal lobe which caused no motor or sensory symptoms.

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* *Revue de Méd.*, Oct., 1882.

† References to the evidence of this will be found in the chapter on the functions of the cortex. A series of cases of disease of the central region with loss of sensation is recorded by Petrina, *Prag. Zeitschr. f. Heilk.*, ii, 1887, No. 5.

that the symptom is not of localizing value. Local convulsions are sometimes caused by irritating disease adjacent to the ascending frontal. Disease of the third frontal, on the left side, impairs voluntary speech in the manner already described (p. 546), and a similar effect is produced by disease of the corresponding region on the right side in left-handed persons, and in those who have recovered speech after destruction of the left motor speech region. No sensory symptoms are caused by disease in the prefrontal lobe, but in some cases considerable mental change has been observed, various in character, but sufficiently frequent to be of some significance. It is greatest when both frontal lobes are diseased.

Parietal Lobe.—The symptoms produced by disease of the ascending parietal and the superior parietal lobule have been mentioned already. The posterior extremity of the latter, adjacent to the parieto-occipital fissure, has been found diseased when no motor or sensory symptoms had been observed. Ptosis, on the opposite side, is said to be specially related to lesions of the lower parietal lobule (Landouzy and Grasset), and the opinion is supported by a considerable number of cases. Disease of the posterior part of the inferior lobule probably causes "crossed amblyopia," and on the left side interferes with the visual perception of words, and if bilateral perhaps causes mind blindness. Extensive disease of the whole parietal lobe seems to impair sensibility in the trunk and limbs on the opposite side. Such an isolated effect has not been observed, but it is certain that disease involving the parietal as well as the central cortex causes more complete hemianæsthesia than does a lesion that is confined to the central region.

Occipital Lobe.—Disease of the apical region, and especially of the cuneus, causes hemianopia. Possibly, disease of the anterior part of this lobe causes color hemianopia. No motor or other sensory symptoms are known to be produced by disease elsewhere in this lobe.

Temporo-sphenoidal Lobe.—Only one symptom is caused by disease of the outer aspect: deafness in the opposite ear when the disease involves the hinder half of the highest convolution, and an auditory aura if convulsions are caused by a lesion in or near this part. The deafness is not permanent, and the auditory sensation is not of precise localizing significance, because the disease may be on any side of the auditory centre. Disease of the highest (uncinate) convolution on the medial surface, near the apex of the lobe, may disturb the sense of smell on the same side (see p. 461).

Lastly, extensive lesions of the cortex around the fissure of Sylvius, extending over a considerable area of the central, parietal and temporal lobes (such as is shown in Fig. 185, p. 463) may cause hemiplegia and hemianæsthesia involving all the special senses, the opposite eye being rendered amblyopic.

WHITE SUBSTANCE OF THE HEMISPHERE. CENTRUM OVALE.—Disease of the white substance causes symptoms resembling, on the one hand, those of the cortex, and on the other those of the internal capsule, according as the seat of the lesion approaches one or the other of these structures. Thus, a lesion beneath a given part of the central convolutions produces paralysis similar in distribution to that which is caused by disease of the corresponding region of the cortex. Hemiplegia is produced by lesions that interrupt the fibres which converge from the central region to the motor part of the internal capsule. One important difference, however, exists: the local convulsions, that are so frequent and characteristic a symptom of a cortical lesion, are met with in disease of the white substance only when an irritating lesion is situated just beneath the gray substance, so as to irritate this directly. General convulsions are rare, and occur chiefly in disease that increases intracranial pressure. The loss of speech that results from disease of the fibres passing from the third frontal convolution to the internal capsule is transient, unless the lesion is just beneath the cortex. Its various characters have been described at p. 527. Anæsthesia occurs chiefly from extensive lesions beneath the parietal and central regions, but in most cases in which considerable hemianæsthesia results from disease of the white substance, this extends down to the neighborhood of the posterior part of the internal capsule. Hemianopia may be caused by disease of the white substance of the occipital lobe.

CORPUS CALLOSUM.—The chief lesion that occurs in the corpus callosum is tumor, but this almost always extends into one or both hemispheres. Mental dullness and bilateral weakness in the limbs, greater on one side, have been present in many cases; but it is probable that these symptoms are due to the pressure on or extension into the cerebral hemispheres. We do not yet know of any symptoms that are the result of the damage to the callosal fibres, and it is certain that the symptoms that have been present are indistinguishable from those produced by multiple tumors.*

CENTRAL GANGLIA AND INTERNAL CAPSULE.—*Internal Capsule.*—Disease limited to the anterior part of the capsule, between the caudate nucleus and the anterior extremity of the lenticular nucleus—so as not to involve the "angle" at the junction of the two parts—is very rare, and we do not yet know whether it causes definite symptoms.† Disease of the angle and posterior segment causes hemiplegia of the common type, the lower part of the face, the tongue, the arm and the leg being all involved, and if the disease is on the left side there may be at first defect of speech. The latter is transient; the hemiplegia is permanent if the lesion continues (except only such recovery as may occur by compensation), and the paralyzed limbs are the seat of late rigidity. At the onset there may be deviation of the head and eyes, but there is no permanent and complete palsy of any cranial nerve. Convulsions may attend the onset of an acute lesion, but are infrequent, and, as in other parts except the cortex, they do not recur after the disease has reached a stationary stage. The palsy is thus the typical, common form, the details of which have been described at p. 509. Most cases of simple hemiplegia are due to disease of this part, and we must therefore assume that this region is diseased, unless there are other symptoms to indicate a different seat of the disease. But hemiplegia from disease of the capsule is not always complete; we have seen (p. 469) that the path for the face and tongue seems to pass chiefly at the angle, that for the arm in the anterior, and that for the leg in the middle third of the posterior limb. Small lesions may therefore affect *chiefly* one or the other of these parts, but there is scarcely ever an actual limitation of the palsy. Hemianæsthesia may accompany the hemiplegia, from implication of the separate sensory path in the hinder third; such sensory loss sometimes accompanies hemiplegic weakness in which the leg suffers most, a fact which is explained by the contiguity of the fibres for the leg and the sensory path. The hemianæsthesia produced by a considerable lesion of the path in the capsule may be complete, and involve the special senses as well as the skin and the muscles; the loss of vision is hemianopia, as before explained. Smell may also be lost on the anæsthetic side. This region is the "sensory crossway" of Charcot. But the special senses may escape, even when the cutaneous loss is great. At the deliberate onset of an acute lesion, tingling and other sensations are far more common than they are when the disease is limited to the motor part of the capsule. If the hemianæsthesia is partial, from incomplete disease, pains are often felt in the affected limbs, sometimes during the rest of life.

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* Some interesting cases of tumor of the corpus callosum have been published by Bristowe, *Brain, 1884*, p. 315, but the diagnostic indications formulated by him are no exception to the statement in the text. A remarkable case of extensive hemorrhage into the corpus callosum has been recorded by Erb (*Virch. Arch.*, Bd. 97, 329), but the hemorrhage occurred apparently during the course of cerebro-spinal meningitis, in the symptoms of which any effects of the callosal hemorrhage were lost. The history hardly seems to warrant the conclusion that no symptoms were produced.

† Raymond and Artaud believe that the speech path is in the anterior segment (*Arch. de Neurologie, 1884*), but it is doubtful whether this path is separate from that for the tongue and lips in the angle of the capsule.

Corpus Striatum.—Acute lesions of either caudate or lenticular nucleus generally cause hemiplegia, but this is permanent only if the internal capsule is directly damaged. If the lesion is confined to the gray substance of either nucleus there are usually no persistent symptoms, motor or sensory. If the lesion is small, and is at a distance from the white fibres of the capsule, there may even be no initial hemiplegia. I have seen a narrow vertical band of central softening, extending from the anterior to the posterior extremity of the lenticular nucleus, when no trace of hemiplegia could be detected before death, and a careful history had elicited no account of any previous paralysis. Chronic lesions, such as tumor, often develop in the gray nuclei without causing paralysis. From these facts it is probable that no sensory or motor palsy is produced by disease of the gray matter, and that the hemiplegia which occurs at the outset of acute lesions is due to the interference of the motor fibres in the internal capsule. In a few cases, of which some have been recorded by Demange,* mobile spasm, choreoid movement, has been observed when the lesion involved the posterior part of the lenticular nucleus, but in most cases the internal capsule was also damaged.

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* *Revue de Méd.*, May, 1883.

Optic Thalamus.—Slight hemiplegia has been observed when the disease has been in the middle third of the thalamus, but is absent if the lesion is small or near the ventricular surface; hence, there is a strong presumption that the loss of power is due to interference with the motor part of the internal capsule, which is adjacent to the middle third. In disease of this part of the thalamus another motor symptom has been occasionally observed, mobile spasm and incoördination on the opposite side, chiefly in the hand. The symptom usually succeeds transient hemiplegia. In one such case I found a small cicatrix of old softening, limited to the thalamus,* and several other similar cases are on record. In one there was softening in the lower part of the thalamus, adjacent to the crus.† It seems probable that the initial hemiplegia is due to the interference with the capsular fibres, and that the subsequent spasm is in some way the direct result of the disease of the thalamus. Sensation is not impaired by lesions limited to the thalamus, but hemianæsthesia occurs when the disease extends into the internal capsule outside the pulvinar. In such cases there may also be hemianopia, but this symptom may, as we have seen, be caused by disease limited to the posterior part of the thalamus itself.

It has been thought that muscular sensibility is specially impaired by thalamic lesions (Meynert), but the evidence does not satisfactorily exclude the adjacent capsule. Diminution of reflex action has been thought to be another effect, but it is met with in disease of other parts of the brain. Vasomotor symptoms do not seem to result from thalamic lesions.

CORPORA QUADRIGEMINA.—Lesions whose influence is confined to the corpora quadrigemina are so rare that there is considerable doubt as to the symptoms produced. It has been thought that disease of the anterior pair causes loss of sight and loss of action of the pupils, but in most of the cases on which the opinion is based the disease was a tumor, and optic neuritis coexisted.‡ Ataxy of movement has been present in some cases of disease of the posterior tubercles, but it resembled closely that due to disease of the middle lobe of the cerebellum, implication of which may possibly have been the cause of the symptoms. Loss of the upward movement of the eyes was produced, in one case under my care, by a small growth at the junction of the hinder tubercles with the valve of Vieussens, but the folia of the latter were also involved. In most cases of disease of the tubercula, causing palsy of the ocular movements, the lesion has extended so deeply as to involve the nuclei of the third nerves.

CRUS CEREBRI.—The characteristic symptom is hemiplegia, involving the lower part of the face as well as the limbs, and accompanied by palsy of the third nerve on the opposite side, *i. e.*, on the side of the lesion, the two symptoms coming on at the same time. The affection of the third nerve is usually complete, but now and then is partial; sometimes the fibres for the internal ocular muscles have escaped. Convulsions are seldom met with, but some spasm has been observed in cases of tumor. Loss of sensation accompanies the motor palsy when the lesion extends into the upper region of the crus, and is sometimes accompanied by tingling and other signs of sensory irritation. Vasomotor disturbance is occasionally conspicuous in the paralyzed limbs. Hemianopia is much less common than might be expected from the contiguity of the optic tract, and has been met with chiefly in cases of tumor.

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* *Med.-Chir. Trans.*, *1876*, p. 318.

† Greiff, *Arch. f. Psych.*, Bd. xiv, p. 598. Choreoid movements set in suddenly, with transient hemiplegia and hyperæsthesia.

‡ One case of acute lesion causing these symptoms has been recorded by Dr. Bastian, but at present it stands alone.

Partial lesions in or near the middle line, beneath the corpora quadrigemina, may cause the symptoms of acute nuclear ophthalmoplegia described at p. 616.

Pons.—Unilateral lesions of the pons often cause palsy of the limbs on one side and of the fifth, facial, or sixth nerve on the other, the "alternate hemiplegia" described at p. 515. But a lesion may be so placed, generally in the upper part of the pons, that the cranial nerves and nuclei escape, and the hemiplegia is then indistinguishable from that which results from disease of the internal capsule. An important unilateral symptom is the loss of the conjugate movement of both eyes toward the side of the lesion, described at p. 610. Bilateral lesions are not uncommon; the limbs or face, on both sides, may then be involved. Difficulty in swallowing and in articulation is common, but is less pronounced and prolonged than in lesions of the medulla oblongata. Convulsions are rare in chronic, but common in acute lesions, and have often a peculiar character; both arms or both legs may be chiefly involved, or there may be semi-convulsive paroxysms of coughing. Sometimes there is rigidity of the limbs or of the muscles supplied by the fifth nerve. Choreoid movements have been noted in rare cases; in one instance there was rhythmical spasm on voluntary movement (Bastian). Loss of sensibility in the limbs and trunk is sometimes associated with the motor palsy, in consequence of the implication of the tegmental region of the pons. The region supplied by the fifth nerve usually escapes on the side of the hemiplegia, but is anæsthetic on the side of the lesion, if this is so placed as to involve the fifth nerve. In the latter case, pain in the same region is occasionally a prominent symptom. Vasomotor symptoms are seldom conspicuous. Ophthalmitis is met with when the fibres of the fifth nerve are affected, but much less frequently than when the disease involves the trunk of the nerve. Acute irritating disease, especially hemorrhage, may cause strong contraction of the pupils, and a lesion that extends into the upper part of the pons, beneath the corpora quadrigemina, may paralyze the internal or external ocular muscles. Auditory symptoms are seldom met with; rarely there has been deafness on the side of the lesion, or a subjective sensation of sound has attended the onset of an acute process. Giddiness occurs chiefly when the disease is in the neighborhood of the middle peduncle of the pons. Vomiting is not specially frequent. Disturbance of respiration and of the action of the heart, glycosuria and albuminuria are sometimes observed. Hyperpyrexia is not uncommon immediately after the onset of an acute lesion of the pons, and scarcely ever attends disease elsewhere.

MEDULLA OBLONGATA.—Severe acute lesions of the medulla are quickly fatal by interference with the respiratory or cardiac centres, and, on account of the small size of the medulla, and the close proximity of the structures, a hemorrhage usually has this effect even when of small size. The chief acute lesion that may give rise to a diagnostic problem is softening from vascular obstruction. Motor symptoms in the limbs are often bilateral, and sometimes involve especially the upper or lower limbs; if one-sided, the tongue may be paralyzed on the side opposite to the lesion, if this is in the anterior part of the medulla, so as to involve the pyramid and the root fibres of the hypoglossal nerve. The face (with the exception of the lips) always escapes. The most characteristic symptoms are those that are due to the interference with the bulbar nerves, the hypoglossal, glosso-pharyngeal and spinal accessory. Such symptoms are seldom unilateral, because they usually depend on interference with the nuclei, and these lie for the most part near the middle line, and suffer on both sides. Hence, the resulting palsy commonly involves the orbicularis oris (which is supplied from nerve cells near the hypoglossal nucleus), the tongue, palate, pharynx and sometimes the larynx, with impairment of articulation and of deglutition. The paralysis often amounts to almost complete loss of articulation, but loss of power in the pharynx is seldom absolute, perhaps because the glosso-pharyngeal nucleus is farther from the middle line than are those of the hypoglossal and spinal accessory nerves. The resulting symptoms are described more fully in a separate section on the bulbar paralysis that results from nuclear degeneration, and acute lesions of the medulla.

BILATERAL LESIONS in the motor region cause paralysis on both sides, and if the disease is situated in the centres for the muscles of bilateral use, which recover, by compensation, when one hemisphere is diseased, the paralysis of these muscles is great in degree; in completeness it resembles that caused by disease lower down the motor path where the two paths are near together. Thus, disease of the highest part of the central regions, which sometimes results from meningeal hemorrhage during birth, causes palsy of the legs closely resembling that due to disease of the spinal cord. Disease of the lowest part of each ascending frontal convolution causes bilateral palsy of the lips, tongue and throat, closely resembling labio-glossal paralysis—"pseudo-bulbar paralysis," it is termed. So, too, in the sensory regions in which compensation occurs; complete deafness may be caused by disease of each first temporal convolution. In the few cases on record in which the symptoms commonly due to disease of both hemispheres are produced by disease on one side, it is probable that there is a congenital or old-standing defect on the other side, so that the apparent exceptions do not really contravene the common rule. Bilateral disease of the occipital lobes may cause absolute blindness, a compound of double hemianopia; it has almost always developed in two distinct attacks.

CEREBELLUM.—Disease of the middle lobe of the cerebellum causes unsteadiness of movement—a reeling gait and often a difficulty in standing, a tendency to sway which renders difficult the maintenance of equilibrium. Sometimes, but not often, there is a tendency to fall backward. There is not the irregular movement of the legs that characterizes locomotor ataxy, although in some cases of tabes the unsteadiness closely resembles that of cerebellar disease. In walking, the tendency to oscillation may give a zigzag direction to the walk, and the resemblance to the gait of a drunken person is often close. The arms are usually steady; rarely they present some jerky incoördination. The unsteadiness is due to damage to the middle lobe, either from disease in it, or from compressing disease in one hemisphere. If a lesion in one hemisphere does not compress the middle lobe, this symptom is absent, and such a lesion seems *per se* to cause no symptoms by which it can be recognized. Other symptoms of cerebellar disease occur also in many different intracranial maladies, but are more frequent in disease of the cerebellum than in disease elsewhere, so that they have a slight localizing value; such are giddiness and vomiting, but these occur only in active diseases that irritate or compress, *e. g.*, in tumor and at the onset of acute vascular lesions. The unsteadiness is not necessarily related to vertigo; the two are sometimes associated, but the unsteadiness may be extreme when there is no subjective sense of giddiness. Nystagmus is frequent in cases of tumor, and is perhaps directly due to the disease of the cerebellum.* In the same cases (of tumor) the knee jerk cannot be obtained; its loss is not usually constant; a slight movement can be elicited at times, and at other times none. We are not at present able to explain this curious symptom. Since it is observed only in cases of irritating disease, it is probably the result of some influence exerted on the spinal centres on which the knee jerk immediately depends. We may note that the path from the sensory muscle nerves—on which the myotatic irritability immediately depends—probably goes to the cerebellum, and the centripetal impressions from the muscles influence cerebellar coördination. It is interesting also to compare the loss of the muscle reflex action in cerebellar disease with the loss of superficial reflex action in disease of the cerebral hemisphere; each is apparently the result of an influence exerted on the spinal reflex centres.

Many other symptoms are produced by cerebellar disease that exerts pressure. The pyramidal tracts are often compressed, and hence the limbs are weakened, and the weakness is accompanied by the usual increase of myotatic irritability. Thus, in one case of cerebellar tumor there may be no knee jerk, and in another it may be increased. The cranial nerves often suffer from the pressure, especially the sixth nerves. Occasionally there are other signs of pressure on the medulla oblongata, such as difficulty of articulation, etc. These symptoms, which are almost confined to morbid growths, are described more fully in the chapter on intracranial tumors.

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* It was in a case of tumor of the cerebellum that the curious rhythmical spasm in the pharynx, mentioned on p. 628, was observed.

Convulsions are rare in disease of the cerebellum, but have occurred occasionally, epileptiform in type. Tetanoid rigidity, with bending back of the head, constant or paroxysmal, has been met with, and has been ascribed to the cerebellar disease (Hughlings Jackson). But it occurs only in tumor, and is met with also in cases of infiltrating glioma of the pons, so that perhaps it is an indirect pressure effect. Hemorrhage into the cerebellum sometimes extends into the side of the pons, and the symptoms may be indistinguishable from those of an acute lesion in the latter situation.

Cerebellar Peduncles.—The superior and inferior peduncles are scarcely ever the seat of isolated disease. The middle peduncle is sometimes separately damaged, but the lesion often involves also either the side of the pons or the adjacent cerebellar hemisphere. The chief symptom is vertigo, a sensation of movement and actual movement, but this is present only in irritating lesions. The sensation is often most intense; the movement may be merely an involuntary motion of the head and eyes, or there may be a strong tendency to an actual rotation of the trunk on its long axis, a forced movement sometimes present whether the patient is lying or standing, sometimes chiefly marked in the upright posture, when it may cause the patient to turn round and round. Occasionally the eyes are in an abnormal position, one being on a different level from the other. With these symptoms there is often evidence of damage to the side of the pons, symptoms of paralysis of the fifth nerve, with or without hemiplegic weakness. A stationary lesion may cause no symptoms.

The chief disease of the ventricles that gives rise to localizing symptoms is hemorrhage, in the account of which these symptoms are considered. Of the diseases that affect the base, the local diagnosis in hemorrhage and inflammation is subordinate to that of the morbid process, and the indications of the position of morbid growths are considered in the chapter on intracranial tumors.

DISEASES OF THE MEMBRANES OF THE BRAIN.

CONGESTION.

Active congestion of the membranes of the brain constitutes the first stage of inflammation, and has been supposed to exist as an independent condition in some cases, chiefly in children, in which there are acute cerebral symptoms—headache, delirium, convulsions, coma—ending sometimes in recovery, sometimes in death. In the latter cases indications of meningeal hyperæmia have been found. It is assumed that in all these cases the condition is one of active congestion of the membranes. In fatal cases the symptoms are similar to those of rapid meningitis, and it is probable that the condition is of this nature. In other cases it is possible that the congestion involves the whole brain or part of the brain. The treatment of congestion of the membranes, if it can be recognized, is that of meningitis.

INFLAMMATION, MENINGITIS.

Of the three membranes that enclose the brain, only two are pathologically separable, since the arachnoid and pia mater always suffer together. The separate inflammation of the dura mater is termed "*pachymeningitis,*" and is much less common than the affection of the pia-arachnoid, which is commonly meant when "meningitis" is spoken of. The affection of the soft membranes has been of late termed "*leptomeningitis,*" in more precise antithesis to pachymeningitis.

INFLAMMATION OF THE DURA MATER: PACHYMENINGITIS.

In the dura mater there are two layers, a thin layer with a smooth epitheliated surface, and a thicker, looser, outer layer, which serves as a periosteum for the bones. Inflammation may affect primarily either of these layers, and thus constitute what has been termed external and internal pachymeningitis. The former is the most common.

EXTERNAL PACHYMENINGITIS is almost always secondary to injury or to adjacent disease, and is very rare in children. The injury that causes it is usually a blow on the skull, often one that fractures the bone, and causes an effusion of blood between the bone and the dura mater. The disease is usually caries or necrosis of the bone, sometimes involving only the outer table; caries of the petrous bone is an occasional cause. The disease results, in rare cases, from mischief outside the skull, as erysipelas, probably by the mechanism of the veins of the diploë. Very rarely the meningitis occurs without any traceable cause. The anatomical changes consist at first in redness and œdematous swelling of the tissue, which soon becomes first discolored and then infiltrated with pus, which may accumulate in considerable quantity between the dura mater and the bone. Very rarely pus forms between the two layers of the dura mater. The inflammation may spread to the inner layer, and even from this to the pia mater, so that the two become glued together by lymph. If the inflammation subsides without the formation of pus, there remains a thickening of the outer layer of the dura mater, firmly connecting it with the skull, and into this tissue osteophytic growths may extend from the bone.

The *symptoms* of the secondary inflammation are usually lost in those of its cause. When distinct symptoms exist, they are usually gradual in their onset, and consist in headache, delirium and sometimes convulsions. When there is a collection of pus compressing the motor region of the cortex, there may be paralysis of the opposite limbs. There may be fever, but often there is no elevation of temperature that can be referred to the meningeal inflammation. The condition is serious, but not quite so grave as most other forms of meningitis. The treatment is first and chiefly that of the local disease or injury exciting the intracranial inflammation, and secondly, the same measures as are suitable for other forms of meningitis, in so far as they are compatible with the treatment of the cause of the disease. If there is reason to suspect the formation of pus between the bone and the dura mater, this may be let out by trephining.

INTERNAL PACHYMENINGITIS is met with in two forms, purulent and hemorrhagic, the latter giving rise to what has long been termed "hæmatoma of the dura mater." Both are uncommon; the former, indeed, is extremely rare.

PURULENT INTERNAL PACHYMENINGITIS is usually associated with a similar inflammation either of the outer layer or of the pia mater. In one or two cases it has been met with as an apparently primary condition. The symptoms have been similar to those of purulent inflammation of the pia mater.

"HEMORRHAGIC INTERNAL PACHYMENINGITIS;" HÆMATOMA OF THE DURA MATER; MENINGEAL BLOOD TUMOR.

A peculiar formation that extends over the brain, commonly over both hemispheres, and consists of membranous layers with the remains of blood between them, has long been known by this name. The malady is very rare, and it is, indeed, of interest chiefly as a pathological curiosity and enigma, for it has seldom been even suspected during life, and we do not know whether it can be influenced by treatment. It is commonly described as a result and variety of inflammation of the dura mater, because Virchow, in 1854, brought forward evidence to show that this was its origin. It had previously been ascribed to primary hemorrhage,* and this view, advocated by Prescott Hewett, in 1845,† has been recently revived by Huguenin; the question is still undecided. Provisionally, however, the disease may be kept in its place among the varieties of inflammation. The disease is so uncommon,‡ that a brief notice of it may suffice; Huguenin's exhaustive description of the malady in Ziemssen's "Handbuch"§ is accessible to all readers likely to be interested in the subject.

CAUSES.—The condition is met with chiefly in males, less than one-fourth of the cases having been in females. It is least rare in old age; more than half the cases occur in persons above fifty years of age; forty per cent. are over sixty, and just a quarter are over seventy. Considering the comparatively small proportion of persons who reach the age of seventy, this proportion shows that the tendency to the disease is enormously increased by the tissue changes incidental to age. In early life it is relatively as well as absolutely infrequent, and occurs chiefly in infancy, as frequently in the first year of life as between one and twenty years of age. The affection is commonly secondary; some cases have followed an injury to the head, but the diseases to which it is most frequently consecutive are some forms of chronic insanity (especially general paralysis of the insane) and chronic alcoholism. It is said also to have followed some acute and chronic blood diseases, especially acute rheumatism and smallpox, scurvy and profound anæmia.

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* By Houssard, Baillarger, and others.

† *Med.-Chir. Trans., 1845.*

‡ Its rarity, at any rate outside asylums, may be judged from the fact that during the forty years in which the Pathological Society has received the curiosities of metropolitan necroscopy, not a single specimen has been brought before the Society from any London hospital.

§ Bd. xi, p. 342 (1st ed.); vol. xii, of the American translation.

Pathology.—The condition is bilateral in about half the cases. Within the dura mater, between it and the arachnoid, and commonly adherent to both, are layers of peculiar membranous tissue. This extends over the greater part of one or of both hemispheres. The tissue is soft and red at first, afterward paler and firmer. There may be several layers, even six or seven, adherent at the edges, so as to form a series of sacs, which contain blood, liquid or coagulated, or in various stages of degeneration; and ultimately there may remain only colored serosity, in which crystals of cholesterine are sometimes found. Occasionally the membranes are adherent in places, so as to render the cavities loculated. The red tint of the membranes depends on minute vessels, and on the presence of degenerating blood; the increased consistency and pallor of the later stage is due to the development of a delicate fibrous tissue. The nature of the earliest stage of the disease is a matter on which some doubt exists; Virchow and Huguenin have investigated conditions which they believe to be the initial stage of the disease, and the former finds in it a process of inflammation, the latter a simple extravasation of blood. Virchow believes that a delicate vascular membrane is formed by inflammation, and that into this hemorrhage takes place. Huguenin has found a primary layer of hemorrhage in some cases of general paralysis, and believes that this slowly becomes organized, as blood clots may do elsewhere. It must be admitted that the latter theory agrees well with the etiological conditions, especially with the occurrence of the disease in old age, when hemorrhage is more frequent than inflammation. In the cases of general paralysis Huguenin has traced degeneration in the walls of the veins of the convexity, from which, he believes, the blood comes. In atrophy of the brain, with which, in the old, the condition is often associated, the external support to the veins must be lessened. In many of the general diseases by which it is said to be caused, meningeal hemorrhage has been known to occur. It is possible, however, that both views are correct, and that inflammation, in some cases, takes part in the production of the condition.

Symptoms.—In some cases of hæmatoma in general paralysis, no symptoms have been present other than those that are common in this disease when no unusual condition is found after death. In other cases there have been apoplectiform seizures, recurring, and ascribed to the hemorrhages that occur from time to time in the cavities of the hæmatoma, and there have been more persistent symptoms of depressed cerebral function—somnolence or coma, with contracted pupils and general muscular weakness. Optic neuritis sometimes occurs toward the close. Headache has been a prominent symptom in many cases, and has been sometimes limited to the vertex in the early stage. Convulsions are not common, but have occasionally occurred, generally on one side. In many cases there is hemiplegia, seldom complete, with or without early contracture. The unilateral symptoms occur when the disease is confined to, or greater on, one hemisphere of the brain. In children the disease is said to run an acute course, with convulsions and sometimes fever. It is doubtful whether, in them, the malady can be diagnosed. In other cases, symptoms of impairment of the functions of the brain and headache, following or coinciding with a cause of hæmatoma, may lead to a suspicion of its existence, and this may be strengthened by the occurrence of apoplectic seizures. An injury is the cause that would give most weight to the symptoms, because it is after injury, rather than in cases of chronic degeneration, that the symptoms have most distinctly suggested the condition. But all the symptoms of hæmatoma, including the apoplectic seizures, may be caused by a glioma of the brain substance. The malady has caused death in most instances. One case of recovery is on record; the subject was a drinker, who was murdered six months after the cerebral symptoms had passed away, and in whom the remains of the disease were found.

Treatment.—The part which extravasation of blood manifestly takes in the production of the morbid state suggests treatment calculated to arrest hemorrhage; rest, moderate elevation of the head and shoulders, and the application of cold to the head. Moderate purgation and diuresis may also be employed. The assumption that inflammation constitutes one factor in the pathology of hæmatoma has suggested leeching and counter-irritation, especially in children, but in them treatment is generally powerless to arrest the rapid course of the disease.

INFLAMMATION OF THE PIA MATER (PIA-ARACHNOID)—(MENINGITIS; LEPTOMENINGITIS).

Certain varieties of meningitis are commonly distinguished: (1) According to the seat, whether this is at the convexity, or at the base, or in the ventricles; the seat entails considerable differences in the symptoms, without any difference in the nature of the inflammation. (2) According to the origin of the inflammation, whether primary or secondary to local cause. (3) According to the nature of the inflammation, whether simple, purulent, tubercular or syphilitic. The symptoms of all forms have, however, much in common, and in all cases the problem in diagnosis is first to ascertain the existence of meningitis and then to distinguish its seat and nature. Hence, it is more useful to consider all forms together, noting as we go on the differences between them, than to give a separate account of each variety. Four-fifths of the statements that must be made about each are true of all. Epidemic cerebro-spinal meningitis is separately described.

ACUTE MENINGITIS.

Causes.—Acute meningitis occurs at all ages, but is most common in children between one and ten years of age. Only one form is less frequent in children than in adults—meningitis of the convexity, and this relation is due chiefly to the common traumatic origin of this form. Taking all forms together, the disease is more frequent in males than in females, but the several varieties exhibit some differences in this respect. Hereditary tendency is active chiefly in causing the tubercular form. Station in life, occupation, climate and season are influential only to a small extent as determining some of the causes. The direct causes are very numerous.

Direct Causes. (1) *Traumatic influences* of various kinds, acting directly on the membrane.

(2) *Adjacent disease*, especially that which is attended with suppuration, often causes purulent meningitis. The adjacent disease may be (*a*) outside the dura mater, as injury and disease of the bone, especially caries of the petrous bone, or it may be outside the skull, as erysipelas and other suppurative diseases of the scalp. In several recorded cases, traumatic inflammation of the eye has set up a fatal meningitis; probably the inflammation has passed up the sheath of the nerve, just as inflammation often passes down the sheath from the membranes to the optic disc. (*b*) The adjacent disease may be in the brain, a tumor or an abscess, very rarely hemorrhage or softening. An abscess may cause meningitis without rupture, but rupture always causes intense purulent inflammation. Tumors situated near the surface often cause adjacent inflammation, and adhesions between the dura and pia mater may be thus produced over the growth.

(3) *Acute general diseases* are sometimes attended with meningitis, (*a*) acute specific diseases, measles, scarlet fever, smallpox, typhoid fever. The complication is, however, rare in any of them. It is least rare in smallpox, perhaps on account of the septicæmic influence of the extensive cutaneous suppuration. It is extremely rare in typhoid fever, according to pathological evidence, although its existence is often erroneously inferred from intensity of delirium. (*b*) It occasionally occurs in the course of two diseases excited by cold, pneumonia and acute rheumatism.* It was thought at one time to be frequent in acute rheumatism, on the ground of cerebral symptoms that are now ascribed to hyperpyrexia. In some cases in which meningitis actually exists, it is perhaps produced by the agency of endocarditis. The meningitis that coexists with pneumonia is probably often due to the cause of the inflammation of the lung. It is usually purulent, and is of considerable pathological importance, as an instance of the dependence of meningitis on a morbid blood state, perhaps allied to that which causes the epidemic cerebro-spinal form. (*c*) Septicæmia often causes meningitis, whatever be the source of the blood poisoning, whether external injury, internal abscess, softening of caseated glands, septic puerperal processes, the softening of clots, or ulcerative endocarditis. It may be due in part to the micrococcal embolism of minute vessels, and it is not uncommon in such cases to meet with minute extravasations in the pia mater, with pale centres, characteristic of this mechanism. Sometimes local meningitis exists near the plug in a large vessel, when the source of the plug is a septic form of endocarditis. From all these causes the inflammation is often purulent, almost invariably in the cases of septic origin.

(4) Insolation is an occasional cause, and influences chiefly the convexity. The meningitis that results is usually simple, and not purulent. In cases of rapid death, only indications of congestion may be discoverable.

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* This, and many other forms of meningitis, are often termed "metastatic," perhaps the most inaccurate designation that could be found, implying, as it does, that the morbid process leaves its original seat and goes to the part secondarily affected.

(5) Excessive mental work is usually regarded as an occasional, although rare cause of meningitis. This cause may certainly be followed by acute cerebral symptoms, rapidly fatal, but it is possible that the inflammation affects the whole brain, and not the membranes only (see Hyperæmia of the Brain).

(6) *Specific Processes.*—The most common cause of acute meningitis is the growth of tubercles in the membranes, which is usually attended with inflammation, the degree and intensity of which varies much in different cases. Syphilis is also a cause of meningitis, but the inflammation is usually chronic, not acute.

Lastly, meningitis may occur either at the convexity or the base, for which no cause can be discovered.

Causation in Relation to Place.—Meningitis limited to the *convexity* may result from local adjacent disease, from insolation, or from general disease (acute specifics, pneumonia and septicæmia); but in the last class the inflammation is often general, and involves the membranes at the base as well as over the convexity of the hemispheres, and frequently also the membranes of the spinal cord. Meningitis limited to the base is rare except in association with tubercles, but occasionally results from adjacent disease, basal growths, disease of bone or syphilis, and sometimes simple inflammation occurs precisely like that caused by tubercles. Limited ventricular meningitis occurs especially in young children, sometimes during or after acute diseases, such as the exanthemata. In adults it is extremely rare.

Etiology according to Nature.—*Simple meningitis* may result from any of the above-mentioned causes, except, perhaps, septicæmia; the etiological facts above mentioned are therefore, in general, applicable to this form.

Purulent meningitis is usually the result of adjacent suppuration or of septicæmia. It frequently occurs in cases of caries of the cranial bones, especially of the petrous bone. The communications between the middle and internal ear and the intracranial cavity (described in the chapter on cerebral abscess) facilitate the passage of septic inflammation to the membranes. It may be caused by suppurative inflammation of the ear when there is no external discharge. Suppurating disease of the nose is another occasional cause. It frequently results from distant suppuration, causing septicæmia. The meningitis that occurs in acute general disease is also occasionally purulent. When associated with inflammation of the lungs, there is sometimes reason to regard both as the consequences of a common cause (see Epidemic Cerebro-spinal Meningitis).

Tubercular meningitis occurs at all ages, but is most common in children, especially between the ages of two and ten years. In infancy it is not common, although it has been met with as early as six weeks after birth. It is not rare in early adult life, and is occasionally met with in middle life, but scarcely ever during the second half. In children, general tuberculosis almost always exists, but the indications of this may be slight and the meningeal disease may have the aspect of a primary affection. In later life distinct indications of phthisis usually precede the meningitis, but are sometimes absent in young adults. In some collections of cases males, in others females, have preponderated. It is probable that there is little difference in the proclivity of the two sexes, at any rate after puberty. It is generally thought that in childhood males suffer rather more frequently than girls.* Hereditary tendency is of unquestionable importance. Most cases occur in families in which there is a clear history of phthisis or of infantile tuberculosis, sometimes evidenced chiefly by the early death of many children. The disease is common in all classes of society. Imperfect nourishment and bad air no doubt aid in its development, but the cases are very numerous in which no accessory cause can be traced. A distinct excitant is also rare; although excessive brain work or a blow on the head sometimes precedes the onset, and may help to determine a greater disturbance of the meninges than of other parts that are also affected by the general disease; it is doubtful whether it does more than this. Frequently, however, the disease develops during the impairment of general health due to some other malady. It sometimes follows "scrofulous" diseases of bones and joints, chronic enlargement of glands, or tuberculous tumors of the brain; and it occasionally occurs as a sequel to some other acute disease, especially measles (the influence of which in causing tuberculosis is well known), whooping cough, broncho-pneumonia, diarrhœa, typhoid fever, etc. Very seldom it comes on in the midst of apparent health.

Chronic Meningitis.—Diffuse chronic meningitis is generally the result of alcoholism; occasionally it is due to injury. Focal chronic meningitis, when not traumatic, is syphilitic in most cases; perhaps in all except the rare instances in which local tubercular growths are attended with local chronic inflammation. In children chronic basal meningitis is not uncommon in the posterior fossa.

ANATOMICAL CHARACTERS.—The changes may affect all parts of the membranes or only the convexity or the base. When the cause is adjacent disease, the neighboring region of the membranes may suffer alone. Certain changes are common in all forms of meningitis. The earliest is a diffuse reddening of the pia mater, due to the injection of finer vessels than are distended by the mechanical influence of the mode of death. Soon there occurs opacity of the membranes, recognizable most readily in the arachnoid. It occurs also along the vessels of both arachnoid and pia mater, distinctly in consequence of distention of their lymphatic sheaths. It is well seen over the sulci of the convexity, and the spaces at the base of the brain. Collections of yellowish-white or semi-purulent lymph form at these parts, and around the nerve trunks, the sheaths of which may be distinctly reddened. In the early stage of purulent meningitis, these collections may be punctiform, and may resemble very closely tubercular granulations in the pia mater; by coalescence they constitute irregular semi-purulent areas. In cases of some duration the purulent spots may have undergone caseation or induration, the latter chiefly in simple meningitis. The nerve trunks are often involved, and under the microscope the inflammation may be found either limited to the sheath or infiltrating the substance of a nerve that has been paralyzed. Minute hemorrhages may be seen in the nerve, and its fibres may be in various stages of acute degeneration. Sometimes no change can be found. The inflammation may spread, on the one hand, to the dura mater, and on the other, to the substance of the brain. The inner surface of the dura mater may be reddened, and even covered with a layer of lymph, which may glue it to the pia-arachnoid. The superficial layer of the brain is often reddened, and even spots of softening may be seen within it. Sometimes it contains punctiform hemorrhages. The fluid in the subarachnoid space is increased in quantity, and is often turbid. Indications of inflammation may also be present in the ventricles; the lining membrane may be swollen and opaque, sometimes even covered with a layer of lymph (*ependymitis*); the subjacent brain tissue may be softened. The choroid plexus and velum interpositum may also present signs of inflammation. The lateral ventricles often contain a great excess of fluid, which may be turbid, with flocculi of lymph. It may be so considerable as to distend the ventricles and flatten the convolutions, a circumstance that suggested the name "acute hydrocephalus," formerly applied to all these cases. The ventricular effusion seems to be the result, in some cases, solely of the ventricular inflammation, but often there is also a collection of lymph about the openings by which the fourth ventricle communicates with the space around the brain, and the obstruction to these must increase the ventricular distention, and may often cause it when there is no inflammation of the lining membrane; when there is such obstruction, the third ventricle, aqueduct and fourth ventricle are also conspicuously distended. In the so-called ventricular meningitis effusion into the ventricles and slight traces of inflammation are all that is found; the external membranes are normal. Although the traces of inflammation are slight, the acute and febrile course of the disease is consistent only with its inflammatory nature. In all forms, if the inflammation of the ependyma passes away, permanent thickening of the lining membrane may remain, and there may even be adhesions closing the cavity of the posterior cornu. A permanent excess of liquid may be left in the cavities.

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* The Registrar-General's returns for the twenty-five years ending 1872 give the deaths from hydrocephalus in males under five as 91,681, and females 66,708. Probably the chief cause of death returned as "hydrocephalus" during these years was tubercular meningitis.

In purulent meningitis the membranes are covered with a layer of greenish-yellow, often fœtid pus, sometimes at the convexity, less commonly at the base only, often at both. The purulent inflammation frequently extends down the membranes of the spinal cord, sometimes as low as the cauda equina, and in one recorded case suppurative inflammation in the orbit was secondary to that within the skull.* The surface of the brain is often softened, and small collections of pus may be found within its substance, apparently secondary to the meningeal suppuration. Very rarely primary purulent inflammation has been limited to the ventricles.

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* Eröss, *Cent. f. Nerv.*, *1883*, 221.

In *tubercular meningitis* the inflammation is never actually purulent. The appearances are usually those of the simple inflammation, already described, conjoined with the characteristic tubercles. The amount of jelly-like and semi-opaque lymph is often considerable, but it is found chiefly at the base, and is generally most abundant about the optic chiasma, between this and the pons, and in the commencement of the fissures of Sylvius. The membrane over the convexity has a dry aspect, but to the finger may feel somewhat sticky; and if a scalpel is passed over the surface, it removes a little pyo-lymph. The two opposed surfaces of the hemisphere, in the longitudinal fissure, may be slightly adherent where in contact. There is often an excess of arachnoid fluid at the base of the brain, and then the sheaths of the optic nerves are usually distended, so as to form a pyriform swelling behind each eye. Ventricular effusion is present in the majority of cases (about four out of five); generally the quantity does not exceed a few ounces, but it may be so large as to compress the cortex and to distend all the ventricles. The indications of occlusion of the communication between the fourth ventricle and the surface, already mentioned, are more frequent in tubercular than in simple meningitis. The lining membrane of the ventricles and choroid plexuses may be distinctly inflamed. In addition to the signs of inflammation in the membranes, these present the distinctive characteristic of the variety, miliary tubercle. The granulations are most easily seen where the inflammation is slight or absent, especially on the under surface of the temporal lobe. They are at first very minute, and so transparent that they can often be detected only when their prominence is recognized by looking obliquely at the surface. Although seated in the pia mater, they cause a prominence of the arachnoid where this covers the pia mater closely, resembling the minute air bubbles beneath the arachnoid that are often produced during the process of removing the brain; these disappear if the finger is passed over the surface, while the tubercles remain unchanged. In other places they are of larger size, and semi-opaque, and in some places several are aggregated together. Where the lymph is abundant, only the larger and older tubercles can be recognized. The microscope shows the granulations to consist of lymphoïd cells, situated usually around a vessel, within its perivascular sheath. It is important to remember that meningeal tubercle and tubercular meningitis are not quite identical. Tubercles may be found in the membranes when there is no sign of inflammation, in cases of general tuberculosis, and they may be accompanied by symptoms of cerebral disturbance resembling those caused by inflammation. When inflammation accompanies them, its extent and degree vary. It is usually considerable only at the base, and is scarcely ever seen at the convexity and not at the base; not rarely it is general. Sometimes the inflammation is confined to one small area, as, for instance, to the neighborhood of the central convolutions on one side; such local meningitis is usually associated with tubercles of some size; very rarely inflammation and tubercles of the ordinary character are confined to the convexity of one hemisphere.* Even when the inflammation is apparently confined to the base, tubercles can usually be detected, often in abundance, over the convexity, but they are still more numerous, larger, older and more opaque at the base, especially about the commencement of the Sylvian fissure.

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* Huguenin, Hilton Fagge.

The characteristic bacilli of tubercle have been found in the pia mater in these cases (Cornil and others). Moreover, the organisms have been found when no tubercles could be seen with the naked eye, although commencing granulations were discovered by the microscope,* and even in the gray substance of the cortex beneath a focus of limited inflammation.† Tubercles may sometimes be recognized in the lining membrane of the lateral ventricles, and in the choroid plexuses. A careful examination of the inner surface of the dura mater often shows minute tubercles scattered over it. They are frequently met with in the spinal membranes, dura mater and pia mater, sometimes one, sometimes both, and occasionally in great abundance, especially over the cauda equina, when the membrane may look as if it had been exposed to a shower of fine sand.‡ Signs of inflammation are always slighter in the spinal than in the cerebral membranes, and are often absent, especially when the granulations are upon the dura mater only. The cortex of the brain may be injected and softened at the surface, but if there is much effusion in the ventricles the amount of blood in the vessels of the cerebral substance may be less than normal. Under the microscope the walls of the cortical vessels are often found to be crowded with leucocytes, and sometimes minute aggregations of tubercles may be detected within the cortical tissue.

Massive tubercle (a tubercular tumor) is occasionally found in the brain, sometimes more than one. I have seen a mass of tubercle the size of a filbert growing from the under surface of the dura mater. Extensive miliary tuberculosis of the small vessels and the substance of the brain, causing almost universal softening, has once been observed.§

In young children tubercles are almost invariably found widely scattered through the other organs of the body. They are most constant in the spleen —very frequent, also, in the enlarged mesenteric glands. Exceptions to the general character of the affection are rare, but Gee mentions a case in a girl, aged four, in whom a cheesy mass in each lung was the only indication of tubercle outside the membranes, and similar cases have been recorded by others. The occurrence of a collection of caseating scrofulous material somewhere in the body is indeed almost invariable in these cases, and current theories regard the fact as evidence of an infective process. If the backs of the eyes are removed and examined, tubercles will often be found in the choroid.

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* Dawson and Hebb, *Lancet, 1884*, Ap. 12, p. 660.

† Déjerine, *Revue de Méd.*, March, 1885, p. 174.

‡ Wortmann found no tubercles in the spinal membranes only in four of twenty-seven cases examined. *Jahrb. f. Kinderheilk.*, Bd. xx, 1883, p. 300.

§ Gee, "Reynolds' System of Medicine," vol. ii, 2d ed., p. 408.

Thrombosis in a vein of the convexity is occasionally found in tubercular meningitis, sometimes with the secondary intense congestion of the corresponding region of the brain. Very rarely a sinus is occluded by the extension of a clot to it from the brain, but the special symptoms of this are usually lost in those of the meningitis.

Chronic Meningitis.—The diffuse chronic meningitis of alcoholism affects chiefly the convexity, and is always slight in degree. The pia arachnoid is somewhat opaque, especially over the sulci, and there may be lines of opaque lymph along the sides of the vessels. The focal chronic meningitis of syphilis is often associated with a syphilitic growth or with disease of the walls of the arteries. There is much thickening of the pia arachnoid with opaque lymph, which glues all adjacent structures together, and often unites the pia mater to the dura mater. In children this inflammation occurs chiefly in the posterior fossa, about the pons, medulla and cerebellum, which are often firmly adherent. In another chronic form, which is, perhaps, only a later stage of the syphilitic inflammation, the inflammatory tissue has undergone fibrous transformation, and a thick layer of tissue, tendinous or cartilaginous in aspect, extends over a certain region, more often at the base than at the convexity, surrounding and compressing the nerves, and uniting the various membranes. The thickness of the layer may be as much as a third of an inch; the dura mater is often also thickened. It was such a condition that caused the palsy of the fifth nerve recorded by Erb, and mentioned on p. 641. Whenever chronic inflammation involves the membranes about the medulla oblongata it generally closes the openings of the fourth ventricle, and internal hydrocephalus results.

Symptoms.—Certain symptoms are common in meningitis, irrespective of its seat or form, while others depend on its situation, whether at the base or over the convexity of the brain, and the special pathological forms differ in their course, and in the relative prominence of various symptoms. In every form and position the manifestations of the disease present wide diversity in different cases. Nevertheless, in the majority some of a certain series of symptoms are present, and develop in such a manner as to be fairly characteristic.

The symptoms consist partly in general disturbance, such as attends any local inflammation, partly in cerebral symptoms, some general and others local. In many cases there are also associated symptoms due to the pathological process to which the meningitis is secondary. The chief symptoms common to all forms will be first described, and afterward the peculiarities of course, etc., that characterize the special varieties.

Premonitory symptoms are often present, although never distinctive in character. They consist chiefly of such indications of general indisposition as may precede any variety of malady, languor and malaise, to which is sometimes added mental irritability. In children, vomiting, without cause, or on some slight indiscretion in diet, often precedes other symptoms for some days. It may precede any variety of meningitis, but is especially common before the tubercular form. In the non-tubercular forms prodromata are often absent, and the pronounced symptoms of the disease set in suddenly.

The most important of these are headache, delirium, vomiting, convulsions, rigidity and weakness of the limbs, and paralysis of cranial nerves, coming on acutely or subacutely, usually accompanied by considerable elevation of temperature, and followed by somnolence and coma.

Headache is, of all symptoms, the most common; it is usually one of the earliest, and is prominent throughout, until the patient becomes unconscious. It is most frequently frontal, but sometimes general. Continuous in some degree, severe exacerbations occur, in which the pain is very intense, and may cause shrieks of suffering, which have received the name of the "hydrocephalic cry;" and these may continue even when the patient is almost unconscious. In rare cases, meningitis runs its course with little or no pain. This is especially rare in tubercular inflammation, but not uncommon in the secondary purulent meningitis of septicæmia and in the simple meningitis of some other blood states. Vertigo is occasionally complained of, especially at the onset, but on the whole is not frequent.

Delirium is another important symptom. It may be either slight and quiet, or active and violent. It is often an early symptom, although it rarely occurs at the onset of the disease. It derives its significance from its association with headache. At first there is merely wandering during sleep, and, as the mental disturbance increases, periods of perfect or imperfect consciousness alternate with periods of delirium. The cries of pain are often frequent and severe during the delirium. When it has set in it usually persists, sometimes with intermissions, until the development of coma.

Vomiting consists in the rejection of the contents of the stomach, often without nausea; there is not usually retching when the stomach is empty. It is a frequent and often a very early symptom, whatever be the seat of the inflammation, but is more frequent in meningitis of the base than in that of the convexity. The tongue is at first clean, but it may afterward become thickly furred. The bowels are confined, often obstinately, and the abdomen is retracted.

General convulsions occur irrespective of the seat of the inflammation, but are rather more frequent in children than in adults. They may occur at any time during the course of the disease, from the onset to the end. They are occasionally almost the only symptom of the meningitis that is produced by septic processes. Rigidity of the muscles of the neck, with retraction of the head, is common in meningitis about the posterior part of the base, and may extend to the muscles of the back, if the inflammation passes down the spinal canal. It is a symptom of very great importance, being frequently the first unequivocal indication of the malady.

Optic neuritis is a common symptom of meningitis of the base, but is rare when the inflammation is confined to the convexity. It is not an early symptom, being seldom met with until the end of the first week. I have once seen it (unilateral) as early as the fifth day after the onset of the symptoms of tubercular meningitis. It is due to the extension of inflammation from the membranes to the sheath and substance of the optic nerve within the skull, and the descent of this inflammation to the eye, probably aided by the passage of material down the sheath of the nerve to the neighborhood of the globe. The intraocular changes are seldom intense. There is swelling of the papilla, blurring the edges of the disc, and often (especially in tubercular meningitis) the swelling is paler than in commencing inflammation from other causes. The veins are full, but hemorrhages are rare, and the neuritis is never so intense as in cases of tumor. Choroidal tubercles are sometimes seen in the tubercular variety.

Hyperæsthesia of the skin is occasionally met with, and that of the special senses is very common, so that light and noise distress the patient and increase his suffering. Both are apparently due to the general excitability of the brain. Retention of urine is common in the later stage, when consciousness is obscured. Occasionally there is incontinence of urine and fæces.

Local symptoms occur in the cranial nerves and limbs. In the former they depend chiefly on the affection of the base, in the latter on that of the convexity. These symptoms are partly those of irritation, partly those of paralysis. The most common symptoms are in the ocular nerves, causing alteration in the position or movement of the eyeballs or in the size of the pupils. Strabismus is a very important symptom; it is often at first transient, or present only on movement, and even then inconstant, depending apparently on varying over-action of a muscle. In other cases there is persistent and evidently paralytic weakness of certain muscles. Slight ptosis may occur, but complete paralysis of the third nerve is not common. Indeed, the whole of any nerve is scarcely ever paralyzed unless other nerves suffer in some degree, a point of some importance. The pupils are usually contracted in the early stage, especially when there is intolerance of light; subsequently they are often dilated. Inequality is a very common and most important symptom; it occurs in inflammation of the convexity as well as of the base. It is often transient and variable, present at one time and absent a few hours later; now one pupil, now the other, may be the larger. An undue sensitiveness of the dilator fibres to cutaneous stimulation has been noted by Parrot.

Next in frequency is an affection of the facial nerve, weakness of which is indicated by inequality of the mouth, or distinct defect of movement. When the disease is at the base, all parts of the facial nerve may be affected; when it is over the convexity, the lower part suffers alone in most cases; perhaps there is occasionally brief weakening of the upper part. The muscles of mastication may be rigid, but this is not frequent, and their paralysis is still more rare. The hypoglossal nerve sometimes suffers, and deviation of the tongue results.

The hyperæsthesia of the special senses has been already mentioned. An affection of the olfactory nerve is very rare. So also is considerable impairment of sight, which scarcely ever occurs except in cases in which the inflammation passes into a chronic stage, and damages the optic nerve considerably, partly directly, and partly by the contraction of lymph that forms around it. The auditory nerve suffers more frequently, usually in association with the facial (which lies beside it at the base of the brain), but in greater degree. Both auditory nerves are sometimes supposed to be damaged alone in meningitis associated with bilateral otitis interna, but it is certain that in these cases the deafness is due to the labyrinthine inflammation, and not to the meningitis.

One-sided symptoms are not uncommon, muscular rigidity, unilateral convulsion, or hemiplegia. Complete hemiplegia is rare, and occurs only when the membranes about the central convolutions are involved. Sometimes no difference can be seen in the state of the membranes over the two hemispheres to account for the difference in the limbs; the paralysis is apparently due to arrest of function by the irritation. In rare cases, the hemiplegia is due to the occurrence of thrombosis in a surface vein. Rigidity of the limbs of one side or of both is very frequent. It may be slight or considerable, and often varies from time to time; passive movements then cause pain, and the pain produced is often greater than the amount of rigidity would suggest. Convulsions, beginning locally, or partial in extent, are also common in meningitis of the convexity. Unilateral hyperæsthesia may also occur, and there may be areas of anæsthesia, but complete hemianæsthesia is very rare. Aphasia occurs chiefly in the tubercular variety, perhaps because such inflammation is apt to extend along the fissure of Sylvius. It is occasionally an early symptom.

The pyrexia of meningitis varies much in different cases. Often there is from the first a considerable elevation of temperature, 101°–103°, and it remains high, with irregular variations, throughout the disease. In the most acute purulent form the temperature may rise quickly to 104° or 105°, and remain high till death. In other cases the temperature is at first raised and toward the end falls below the normal, and a rectal temperature of 97° or 96° has been observed. On the other hand, toward the end the temperature may be very high, 106° or 108°. It is said that, in very rare cases, the disease runs its course without fever, and it is certain that for some days the temperature may not be above the normal; apparently this is the result of an influence exerted by the nervous system, which neutralizes the tendency of the inflammatory process.

The pulse presents as wide variations as the temperature, but there may be no correspondence between the variations in the two symptoms. In some cases it is frequent throughout; more often it is slow, 68, 50 or even 40, and it is often not only slow but irregular in rhythm. The premortal rise in temperature is usually attended by extreme frequency of pulse, 160, 180, and sometimes unaccountable rapidity.

The vasomotor system is often disturbed. The well-known *tache cérébrale*, in which cutaneous irritation is followed by unusually vivid and enduring congestion of the skin, has been erroneously supposed to be of special diagnostic significance, but is met with in many other affections. There is often a strong tendency to the formation of sloughs and bedsores, and these vesications are frequently produced by the application to the skin of hot bottles, the heat of which is insufficient to cause the effect in a healthy person; uncovered metallic bottles are especially dangerous. These trophic changes are more frequent in subacute than in acute meningitis.

Respiration is sometimes slightly quickened, but it is rarely much disturbed until the final period, when in tubercular meningitis the Cheyne-Stokes rhythm is often observed, and, in this disease, is always of fatal significance. More frequent are irregular pauses, in which the child ceases to breathe for several seconds. In meningitis of the posterior fossa there is sometimes almost sudden failure of breathing, irregular feeble respiration, with rapid cyanosis, quickly followed by death.

The urine is scanty and febrile in character. Albumen or a trace of sugar sometimes appears.

Course.—The onset is sometimes rapid and violent, marked by sudden and high pyrexia, by rigors, and by special symptoms of intense degree. In such cases the inflammation is usually purulent; the spinal membranes are commonly involved, and the cases resemble the epidemic form. The course of these cases is sometimes so rapid as to resemble that of the slower forms of cerebral hemorrhage. Death may occur within forty-eight hours after the onset of the symptoms. In other cases the onset is gradual and insidious, and this is especially frequent in tubercular meningitis. During the early period, symptoms of excitement of function predominate; delirium accompanies the headache; there are convulsions and muscular contractures. At a later period there is depression of function, somnolence deepens to coma, and various paralyses occur. These are often distinguished as first and second stages of the disease, and the deep coma that precedes death is regarded as a third stage. These stages are not always distinctly recognizable. Coma sometimes develops almost at the outset, and palsy of limb or face may be the earliest symptoms. Often the local symptoms are trifling, and only the general cerebral symptoms are marked.

The duration of meningitis varies from two or three days to as many weeks. The most rapid cases are those due to septic processes and cases of the primary purulent inflammation already mentioned. But the maximum duration mentioned above is occasionally exceeded, and cases are met with that must be regarded as subacute. They are generally examples of simple inflammation, and occur chiefly in children; they constitute a transition to the form of chronic infantile meningitis mentioned on p. 739.

Symptoms in Relation to Locality.—In meningitis of the convexity, delirium is more pronounced, local convulsions and hemiplegic weakness are more common, vomiting is less frequent, palsies of the cranial nerves are for the most part absent, and optic neuritis is rare. In meningitis of the base the cranial nerves suffer early, delirium occurs later, vomiting is frequent, and optic neuritis usually develops before the symptoms have reached a high degree of intensity. When delirium is the chief symptom, the aspect of the case may closely resemble delirium tremens. When meningitis is limited to one part of the base, the cranial nerves suffer that have their course in that part. There is usually retraction of the head when the inflammation affects the membranes about the pons and medulla, a symptom of very great diagnostic importance.

In *ventricular meningitis*, in which abundant effusion and traces of inflammation in the choroid plexus and ependyma are the only morbid changes, the symptoms are, strange to say, the same as in other forms—headache, vomiting, fever, convulsions, rigidity, delirium, irregularity of pulse and breathing, and finally coma, but the functions of the cranial nerves are seldom interfered with. The fontanelle, if open, is distended and pulsates. Death may occur in a few days, or at the end of ten days or a fortnight. Occasionally, after an acute onset, the symptoms lessen, but the remission is followed by their renewal in fatal intensity. It is said that incomplete recovery may occur, with enduring defect in mind or muscular power, contractures, oscillating gait or convulsions. Enlargement of the head may develop, and the case practically becomes one of chronic hydrocephalus. There is nothing in these symptoms that is distinctive. All are common to meningitis that is external as well as internal, and there is considerable room for doubt as to the accuracy of the diagnosis of cases that are not fatal.

Symptoms in Relation to Form.—In simple meningitis the symptoms are those above described, and their precise character depends on the seat of inflammation. The course of the disease is often longer than in other forms, three, four or six weeks, and the symptoms present greater variations in degree and in character. The rise of temperature is often at first great, but it may subside as the disease passes into a subacute stage. Probably in consequence of the longer course, optic neuritis is frequent and considerable in degree. In meningitis secondary to local disease, the seat of the inflammation, and therefore the attendant symptoms, differ according to the position of the disease that excites it. Moreover, the symptoms of the primary malady often mask those of the meningitis which it excites.

Tubercular Meningitis.—The symptoms differ in some extent in children and in adults.

In the child prodromata are frequent. Loss of flesh, and indications of general failure of strength are the most common. They are usually due to the process of tuberculosis, of which the meningitis is one result. Slight evening pyrexia often accompanies these symptoms. Other premonitory symptoms are met with in the nervous system—mental irritability, restlessness at night, and a tendency to frontal headache, the pain being excited by mental work and fatigue.

These symptoms may exist for a few weeks, sometimes for a month or two before the actual onset. When the meningitis is secondary to considerable tubercular mischief elsewhere, as in the lungs, the symptoms of this usually obscure any premonitory symptoms of the meningitis. Occasionally, as the cerebral inflammation develops, the symptoms of the lung affection lessen in a remarkable degree. The headache, which may exist for two or three weeks before the onset of the acute symptoms, is perhaps due to the formation of tubercle in the membranes, if, as is generally believed, this precedes the inflammation. Another symptom that often precedes the definite onset, but usually for a shorter time than headache, is vomiting of food. The vomiting may be apparently causeless, or may follow an inadequate cause, *i. e.*, it occurs after food that is not quite judicious, although not so indigestible as to be alone a sufficient cause of its rejection. This symptom should always excite suspicion if it occurs without other indications of gastric disturbance, and especially if it is repeated and succeeds other premonitory symptoms.

Such vomiting, more severe in degree, is a common symptom of the onset, which is usually attended by a great increase in the headache, or by the development of headache if this was absent before, sometimes by drowsiness, and often by attacks of general convulsion. In the early stage of the disease the prominent symptoms are usually those of general cerebral disturbance, and symptoms outside the nervous system—headache, somnolence, intolerance of disturbance, delirium in other children, sometimes vertigo, vomiting, constipation, elevation of temperature, with a pulse that is relatively, if not absolutely, infrequent; a pulse, for instance, of 80, with a temperature of 101°, or a pulse of 60, 50 or 40. Very frequently there is rigidity of the muscles at the back of the neck, and slight retraction of the head, soon after the onset. Aphasia is sometimes an early symptom; tubercular meningitis is one of the most common causes of aphasia in children. At the end of the first week, sometimes earlier, sometimes later, symptoms in the region of the cranial nerves come on, of which those in the eyes and face are the most common; occasional strabismus, inequality of pupil, slight ptosis, imperfect closure of one eye, or inequality of the mouth at rest or on movement. About this time also changes in the optic disc are often recognizable. In the course of the second week the somnolence deepens to coma, but in older children this may be preceded by delirium, occasionally violent. The cranial nerve disturbance becomes more conspicuous. Retraction of the head, if not present before, often comes on, and there may be rigidity of the limbs. There is often also local convulsion, or local paralysis—hemiplegia, or palsy of the arm or face, seldom of the leg or face. Defect of sensation is sometimes associated with the palsy, and may be more extensive; it may be preceded by hyperæsthesia. The paralysis may be transient or permanent. The pulse often becomes frequent, 140–180, sometimes suddenly, sometimes gradually. Respiration is often sighing or irregular. The temperature may remain at a moderate elevation, or may fall below the normal. Its course is irregular, and of no special type, nor is there any correspondence between the character of the pyrexia and the pathological lesions. Toward the end of this, or in the beginning of the third week, the child lies unconscious and motionless, often with rigidity or flaccidity of the muscles of one side; muco-pus accumulates on the cornea. Sometimes there are convulsions, and even choreiform movements, in older children; the respiration is more irregular, and may present an occasional pause or a distinct Cheyne-Stokes rhythm. The patient may die during the coma, with accumulation of mucus in the chest; bedsores may form, and hasten the fatal issue; or death may immediately succeed an attack of convulsions. Sometimes there is an apparent improvement shortly before death, but as Oxley* has pointed out, the pulse seldom shares in this. The temperature toward the end may sometimes remain at about the same degree of moderate elevation, sometimes it becomes very high, sometimes falls below the normal, and is sometimes very low. Thus in one recorded case on the seventeenth day of the disease, the day before death, the temperature was only 93° (Bokai).

In the rare cases in which tubercular meningitis affects only the convexity, the symptoms in the cranial nerves are absent, vomiting is less obtrusive, and headache and delirium, with convulsions and rigidity of limb, constitute the chief symptoms. The pulse is said to be generally frequent, even from the first. Inequality of pupil is often present when other ocular symptoms (including neuritis) are absent. Partial tubercular meningitis, affecting only a small part of the convexity, causes local symptoms resembling those of a tubercular growth, with which, indeed, it is commonly associated.

The duration from the onset varies from one to three weeks. It is rarely less than a week, but occasionally lasts for four weeks. The average is two weeks. In cases that begin insidiously the duration is determined with difficulty, and if the premonitory period is included the average is more than two weeks. The average duration of tubercular meningitis, limited to the convexity, is said to be less than when the base is affected. In all cases the usual termination is death. Probably all cases which pass into the stage of coma end fatally. But it is probable that cases of tubercular meningitis do sometimes recover. Unquestionably cases recover in which the existence of meningitis admits of no doubt, and in which there is a strong presumption that the inflammation was tubercular, although the recovery removes the possibility of rigid proof. In most cases of children who recover from an illness resembling tubercular meningitis, the cerebral symptoms were general pain, delirium, convulsion and coma; as a rule, there has not been paralysis of any cranial nerve. In a few recorded cases of recovery death has occurred some months later, from a recurrence of meningitis, and good observers have declared themselves satisfied of the existence of pathological evidence of the previous attack of meningitis which ended in recovery.

In the adult the symptoms of tubercular meningitis are essentially the same as in the child, but, if possible, even more varied in their character and combination. Headache, vomiting, and cranial nerve symptoms are the same. General convulsions are less frequent, delirium usually occurs earlier, and its association with persistent headache is often conspicuous. The insidious character of the onset is as common as in childhood, in spite of the ability of the sufferer to describe his symptoms. In young women the early stage of the disease is sometimes attended by symptoms of hysteroid character; the morbid functional tendency incidental to the age and sex determines the form of the general functional disturbance that is at first produced by the disease of the membranes.

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* *Liverpool Med.-Chir. Journal*, July, 1885.

The disease commonly comes on in the course of phthisis, but the indications of lung disease may be so slight as to escape notice, and sometimes the malady has the aspect of a primary affection, although tubercles are found after death elsewhere. Besides the symptoms already described, it should be noted that hemiplegia, coming on in the course of a few days or a week, and often attended by convulsions, may be the chief symptom when the inflammation involves the convexity of one hemisphere. The duration of the disease in the adult is sometimes the same as in the child, but often it is apparently shorter, perhaps because the early symptoms are overlooked in the presence of the more obtrusive pulmonary disease. Occasionally, on the other hand, it is much longer. Headache generally precedes other symptoms, and the sufferers have often been liable to headache, and increasingly so during the ill health that preceded the meningitis in most cases. The habitual headache passes into that of the intracranial disease, and increases the difficulty of fixing the commencement of the latter. The fatality of the disease is great, but cases of recovery from what is apparently tubercular meningitis are observed in young adults more frequently than in children. Doubt always hangs over the exact nature of cases that recover, but the probable evidence as to the tubercular character of some of these cases is very strong. As an instance of this class may be mentioned the case of a girl, aged nineteen, who had lost a brother from caries of the spine, and a cousin from phthisis. For some months she had been growing anæmic and languid, especially since the death of a sister two months previously. Three weeks before I saw her, having previously complained for a week or two of slight headache, this suddenly became intense, chiefly on the left side of the head, keeping her awake at night, making her scream with the pain; it was accompanied by frequent vomiting. After a few days these symptoms lessened, but recurred from time to time, and with especial severity two weeks before the onset, a day or two before I saw her. After this attack she was somnolent, did not speak, and the urine was passed into the bed. The temperature, at first normal, rose to 100° She moved the right arm less than the left, and had several convulsions with tonic spasm, flexion of the arms and deviation of the head to the right. When seen, she could be roused to take notice, but not to speak. There was slight rigidity of the limbs, greater on the right side. The legs and upper arms were moved, but not the hands. The pulse was 88 and irregular. There was slight but distinct optic neuritis, in aspect exactly that seen in tubercular meningitis. Her head was shaved, ice applied to it, and a blister to the back of the neck, and phosphate of iron, iodide of potassium and nitric ether given internally. That evening she had four more convulsions, commencing in the right leg. The day after that on which the blister rose she spoke, and afterward slowly improved; for several days more, however, urine was passed into the bed, and the right arm was distinctly weaker than the left. She ultimately recovered perfectly.

Partial tubercular meningitis is rare. The development of tubercle in the meninges may be local, affect one part of the pia mater only, and give rise only to symptoms of irritation of the underlying cortex. When the disease is over or near the motor region of the cortex it may cause convulsion of local commencement or range, and the symptoms are distinguishable from those of a tumor only by their course.

Purulent Meningitis.—The symptoms depend upon its locality. The course is usually very acute, and the pyrexia considerable and constant, often with an evening rise; the temperature is far more regular in its variations than in tubercular meningitis. The general description given of the symptoms of meningitis is in the main applicable to the purulent form. But no form of inflammation, not even the tubercular, presents greater variations in symptoms and course in proportion to the intensity of the process. Some cases secondary to suppuration elsewhere, present extraordinary latency. I have known, for instance, slight occasional strabismus, slight retraction of the head, moderate headache, irregular fever, and optic neuritis, to be the only symptoms, although after death both cerebral and spinal membranes were bathed in pus, and the meningitis certainly commenced a fortnight before death.

Focal meningitis, involving only a small area of the membranes on one side of either the base or the convexity, is generally chronic—traumatic cases of course excluded. The condition is therefore described in the next section. In the very rare cases in which focal myelitis is acute, the symptoms are, for the most part, similar to those of the chronic form, the chief difference being in their course.

CHRONIC MENINGITIS.

The symptoms of chronic meningitis consist chiefly in local disturbance of function; the symptoms outside the nervous system, so conspicuous in most cases of acute meningitis, are, as a rule, absent in the chronic form. In the adult the symptoms vary much, according to the cause and nature of the inflammation. In the *chronic alcoholic meningitis*, which affects chiefly the convexity over both hemispheres, the chief symptoms are headache, slight delirium, mental failure, and slight optic neuritis. The malady always runs a very chronic course, and its symptoms are often overshadowed by those of some other of the effects of alcoholism, such as liver disease or multiple neuritis. The symptoms may subside if the cause of the morbid state can be arrested. It is probable that they are in part due to the action of the alcohol on the brain substance.

Chronic syphilitic meningitis in the adult is generally local. It usually occurs chiefly in the vicinity of a syphilitic growth, and its chief effect is to extend the symptoms of the latter over a wider area than corresponds to the actual tumor. Thus, in the case of a syphiloma growing into the hemisphere from the outer side of the crus, the effects of this were accompanied by palsy of the fifth nerve on the same side, in consequence of syphilitic meningitis extending from the tumor. Sometimes local meningitis exists apart from any distinct growth. There is always a considerable formation of tissue, thickening the pia arachnoid, sometimes to such an extent as to suggest a diffuse growth. The seat of the disease may be at the base or the convexity; in either situation focal symptoms are produced, scarcely distinguishable in the more chronic cases from those of a syphilitic growth. When the seat of the disease is the neighborhood of the crus, pons, or medulla, a distinction from tumor is afforded by the slighter indication of compression of these parts. The side of the medulla is an occasional seat of this inflammation, which is one of the most common causes of the conjoint palsy of one-half of the tongue, side of the palate, and vocal cord (see p. 707). In very rare cases such chronic meningitis is subacute or even acute. It is highly probable that focal inflammation in adults is always syphilitic in nature, the traumatic form of course excepted. The following case is probably an instance of it. A married lady, a month after her confinement, suffered for a week or two from neuralgic pain about the right temple and eye, and then became feverish; one or two convulsions occurred, of uncertain character, and left hemiplegia developed rapidly, the paralysis of arm and leg becoming almost complete in the course of a few days. There was no optic neuritis. Iodide of potassium was given and mercury rubbed in, etc.; as soon as the influence of the drugs became established the symptoms began to lessen, and they passed away entirely in the course of a few weeks. In this case the evidence of local meningitis afforded by the symptoms and their mode of onset was conclusive, and although the effect of the drugs employed is not certain evidence of the nature of the lesion, it gave some support to the opinion, based on the limited extent of the disease, that it was of syphilitic origin. In such a case it is useless cruelty to suggest to a husband, by questions regarding his past history, that he may be the unconscious cause of his wife's malady. It is unlikely that by such questions syphilis can be absolutely *excluded*, and unless it can be excluded, the anti-syphilitic treatment in such a case is the first duty of the practitioner. Moreover, if syphilis can be excluded, the treatment suitable for syphilis remains the most promising for a simple inflammation.

Chronic infantile meningitis, which is also in some cases associated with syphilis, causes symptoms that depend on the special proclivity of the inflammation to affect the posterior fossa of the base. The most important and characteristic symptom is retraction of the head. In a valuable paper on these cases* Drs. Gee and Barlow point out that the onset of this symptom is sometimes sudden, sometimes gradual; when sudden it is occasionally attended by other symptoms—fever, vomiting, rigidity of the limbs, convulsions. I have known general convulsions to precede for a week the onset of the retraction. The holding back of the head is due to a tonic contraction in the muscles, and becomes greater when the child is made to sit up.

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* "On the Cervical Opisthotonos of Infants," *St. Bartholomew's Hospital Reports*, vol. xiv, 1878, p. 22. The occasional relation to syphilis, noted by them, is confirmed by Money, "Treatment of Disease in Children," 1887, p. 456.

It varies in degree at different times, and is occasionally intermittent. When the condition has become established, it is often attended by rigidity of the limbs, sometimes by epileptiform convulsions. Palsy is rare, but strabismus and nystagmus are sometimes observed.* Occasionally hydrocephalic enlargement of the head follows after a time. The symptoms continue for a period that varies from a month to a year and a half, and may end in recovery or death. After death the signs of chronic meningitis are always found, chiefly in the posterior fossa of the base; generally lymph glues together the medulla and cerebellum, and then there is chronic hydrocephalus, the openings from the fourth ventricle being closed by the lymph. There may also be slight inflammation in the membranes of the spinal cord, and this, as I have seen, without special symptoms of spinal meningitis. In some cases such retraction of the head dates from the time of birth, and it is highly probable that in these cases there is hemorrhage into the membranes about the medulla, a proved lesion depending generally on laceration of the cerebellum (see Meningeal Hemorrhage). The retraction of the head must be distinguished from mere inability to support the head due to weakness of the muscles. I have, however, known such inability to follow the retraction in cases in which the symptom dated from birth.

Pathology.—The pia arachnoid differs from most other membranes that enclose viscera, in its separation into two layers. Nevertheless, it is commonly regarded as a serous membrane, and it presents some analogies to other serous membranes in its pathological liability, but also wide differences from them. Like the pleura, it is prone to spontaneous inflammation, but the most common cause of primary pleurisy, exposure to cold, seems to have little influence in exciting meningitis. It is the seat of specific processes more frequently than any other serous membrane, and this, together with its liability to suffer in states of blood poisoning, must be regarded as its chief pathological characteristic. The process of inflammation also presents some peculiarities in the cerebral membrane. The tendency to the formation of lymph is smaller, and of pus is greater, than in the case of the pleura or the pericardium. Embolic processes may play a part in the generation of some forms of septicæmic inflammation, but it is probable that the circulation of the septic matter in the blood, not necessarily organisms, suffices to excite in the membranes the inflammation to which they are prone. The origin of the miliary tubercles of the meninges is a problem that belongs to general pathology.

The acute purulent meningitis that occurs in children and adults, as an apparently primary affection, seems to be identical with the sporadic form of cerebro-spinal meningitis. Although spinal symptoms, beyond some retraction of the head, may be absent, the purulent inflammation extends, in most cases, down the membranes of the cord. The probable pathology of these cases is considered in the section on the epidemic form.

The relation between the morbid process and the symptoms that reveal its presence, is still, in part, obscure. The affection of the cranial nerves is usually due to their actual inflammation, indications of which may be found in the interstitial tissue, and in the degeneration of the nerve fibres. With regard to the character of the symptoms, we are doubtless safe, in recognizing, with the older writers, the early stage of over-action, as the result of irritation of the nerve elements by the inflammation of the enclosing membranes. In some cases the excitement of function, general so far as the meningitic influence is concerned, seems to be determined in its special form by predisposition, or by coincident influences. Hence it is that in specially disposed subjects the early symptoms may present characters definitely hysterical in their character. But the process of irritation seems sometimes to arrest function instead of exciting its activity. In the early stage of meningitis there is sometimes complete hemiplegia, and I have even known the inflammation to be distinctly slighter over the hemisphere the function of which was thus arrested, than over the other. We can only explain such an arrest of function as the result of irritative inhibition. For the depression of function in the later stage there are two possible mechanisms: first, the ventricular effusion and compression of the brain, and second, by the greater damage of nerve elements that have been previously excited. The fact that the stage of depression may be as marked when there is no effusion as when this is abundant, makes it certain that the effusion cannot be its sole cause. We have no guide as to the degree in which we are to apportion the depression to these two mechanisms. Most authorities follow the older pathologists in attributing them to the ventricular effusion.

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* Money (loc. cit.), who found also persistent slight pyrexia in one case.

It is commonly held that the inflammation, in tubercular meningitis, is secondary in time, as well as in effect, to the formation of miliary tubercles in the membranes. The presence of tubercles before the occurrence of inflammation is generally regarded as the cause of the prodromata.* This is probable, in spite of the fact that tubercles are commonly not found in the membranes in cases of general tuberculosis without meningitis.† But it is certain that the amount of inflammation excited by meningeal tubercle varies very much. Hilton Fagge even suggests that tubercles in the membranes may cause, by their mere presence, symptoms resembling meningitis. They may certainly often be found in parts of the pia mater where no evidence of inflammation can be discovered.

Considerable paralysis of the cranial nerves is no doubt always due to the affection of these nerves at the base of the brain, with the exception of palsy of the face and tongue, which may be considerable in degree when of cortical origin. Thus I have seen paralysis of all parts of the face associated with paralysis of the limbs on the same side, but the palsy of the face involved voluntary and not emotional movements, conclusive evidence that it was not due to an affection of the nerve. Spasmodic strabismus, and inequality of pupil, may also probably be due to the affection of the cortex as well as to that of the nerve trunks. Rigidity of muscles is the result of irritation, either of the cortex, crus, or pons; the retraction of the head is due to inflammation about the pons and medulla, not, as is often said, to the ventricular effusion. Whether any symptoms, in the ocular muscles or elsewhere, are due to inflammatory irritation of the corpora quadrigemina, or of the gray matter lining the third ventricle, is uncertain.

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* Rilliet and Barthez, Huguenin, etc.

† Wilkes and Moxon, "Path. Anat.," 2d ed., p. 210; Hilton Fagge, "Prin. and Prac. of Med.," 1886, vol. i, p. 583.

DIAGNOSIS.—The diagnosis of meningitis rests on the presence of such symptoms as indicate organic disease at the surface of the brain, on the development of these in an acute or subacute, but not sudden manner, and on the presence of the general symptoms of inflammation, especially pyrexia. Of the cerebral symptoms, those of local character are the most conclusive, but often of least actual diagnostic importance, since they occur comparatively late in the course of the affection, and although it is now and then impossible to recognize the disease until they appear, they more often serve to confirm, rather than to establish, the diagnosis which has been already made from the general cerebral and other symptoms. These general cerebral symptoms are significant rather by their degree and combination than by their mere presence, since most of them may be produced by processes commencing outside the nervous system. The significance of the headache depends on its intensity; of the delirium, on its co-existence with headache; of vomiting, on its causeless character and persistence; of general convulsions on their association with the other symptoms; of infrequency of pulse on its combination with the pyrexia that usually accelerates the action of the heart. It is in the early stage, when these general symptoms alone exist, that the chief diagnostic errors occur. Of the local symptoms that are conclusive, the most important are the inequality of pupil; strabismus, even if transient; and retraction of the head. The ophthalmoscopic changes may also decide the diagnosis, chiefly in cases in which they occur early, or local symptoms develop late. In some obscure cases the occurrence of retention or incontinence of urine, or the readiness with which the skin blisters, give sufficient weight to other symptoms to determine the diagnosis. In all cases the Protean character of the disease should be remembered, and above all the fact that there is no symptom of meningitis that is not sometimes absent. Headache is the most constant, rarely wanting, but cases have been known to run their course without any obtrusive pain; its absence, therefore, does not destroy the value of other symptoms that are of significance, and there is no other symptom (not excepting pyrexia) the absence of which is of much negative value. The presence or history of a possible cause of meningitis often assists the diagnosis by directing attention to symptoms that might otherwise be unnoticed. This is especially the case with injuries, ear disease, pyæmia, and phthisis. At the same time adjacent disease sometimes renders the diagnosis more difficult by obscuring, with its own obtrusive symptoms, those of its effect.

The existence of meningitis being recognized, the diagnosis of its seat depends chiefly on the character of the symptoms—whether they are such as to indicate disturbance of the convexity or of the base. The differences that depend upon locality have been already described. Sometimes the cause of the meningitis also helps the diagnosis of locality, since adjacent disease excites inflammation first in its own neighborhood.

In determining the nature of the inflammation we are guided by its cause, its associations, its seat, the age of the patient, and the relative frequency of the several forms. Each of these points must be considered, not only in itself, but in relation to the others. The apparent cause often decides the question—from adjacent caries and in pyæmia the inflammation is probably almost certainly purulent. But if there is distant suppuration of scrofulous origin the inflammation may be either tubercular or purulent, and the former is more probable unless there are other signs of septicæmia. In pneumonia or acute specific diseases the inflammation may be either simple or purulent. If there are signs of phthisis or a tubercular family history, and no other cause can be detected, the meningitis is almost certainly tubercular. The discovery of tubercles of the choroid renders the nature of the inflammation certain. A boy, aged five, was seized with acute pain in the head on the twentieth day of typhoid fever (with characteristic eruption); this was followed by coma, inequality of pupil, deviation of head and eyes to the left, rigidity of the left limbs and half-purposive movements of the right. I saw him on the fifth day after the onset of these symptoms, and found optic neuritis in the right eye only, with tubercles in each choroid—a proof that the inflammation was tubercular, complicating the fever and not due to it. If the inflammation is at the base of the brain, and no cause is discoverable, the probability that it is tubercular is very great. If the inflammation is at the convexity the probability of its tubercular nature is considerable in childhood and youth, but in adult life such inflammation is probably not tubercular. Under twenty years of age there is a presumption, in the absence of other causal indications, that any meningitis is of tubercular origin, but over forty there is a presumption against this, which increases in weight as life advances. The special diagnosis of ventricular meningitis is practically impossible during life.

Differential Diagnosis.—Meningitis may be confounded with general diseases and with other diseases of the nervous system. These errors occur chiefly with tubercular meningitis, because most other forms have an association with some cause which prevents a mistake, or else produce symptoms so acute and characteristic that there is neither time nor room for error. The following remarks on the differential diagnosis apply, however, also to the rare simple basal meningitis, which, during life, cannot be distinguished with certainty from the tubercular form.

The error of mistaking an acute general disease for meningitis is more common than the opposite error. Typhoid fever and catarrhal febricula are the diseases with regard to which a mistake is most common, the former in older children and adults, the latter in young children. The error always arises from ascribing initial headache and subsequent delirium to cerebral disease in the presence of sufficient pyrexia to account for the symptoms, and from disregarding the relation between the two. When they are the result of pyrexia, as Sir William Jenner long ago pointed out, the headache ceases when the delirium begins. In meningitis the headache continues, and coexists with the delirium. In children, convulsions may occur at the onset of the general disease. In all cases, therefore, in which there is first headache and then delirium, and in which there is pyrexia sufficient to account for both, the presumption is strongly against meningitis; this should only be suspected when local cerebral symptoms appear, or if there be general cerebral symptoms, such as optic neuritis, that do not occur during general diseases. The difficulty of the diagnosis between typhoid fever and tubercular meningitis is increased by the fact that the former is sometimes attended by constipation, and the latter by diarrhœa due to tubercular ulceration of the bowels, or by general abdominal tenderness from the formation of tubercles in the peritoneum. It is sometimes necessary to wait and watch the course of the symptoms before a confident opinion can be formed. As a rule, the pyrexia runs a more regular course in typhoid, and the pulse is more frequent and is seldom irregular. The optic neuritis that is occasionally associated with acute specific diseases does not accompany but succeeds these. It should be remembered that convulsions which are partial and begin locally, are local and not general cerebral symptoms. Avoidable error arises in most cases from the judgment being warped by the initial headache, and the practitioner omits to keep a lookout for other symptoms outside the nervous system. In consequence of this, gross mistakes are sometimes made. I have even known simple pneumonia to be mistaken for meningitis.

Retraction of the head, resembling that which occurs in meningitis, is sometimes the result of simple rheumatism of the muscles at the back of the neck. Legroux asserts that in such cases there may be considerable fever,* but distinct cerebral symptoms are, of course, absent, and the muscles are said to be more painful than in meningitis. Such muscular rigidity is said to be also produced, in some cases, by peripheral irritation, and tender cervical glands, and abdominal disturbance.†

Among diseases of the nervous system, one that sometimes gives rise to considerable difficulty in diagnosis is intracranial tumor. A rapidly growing tumor, especially one that at first interferes little with function, may cause symptoms which develop so rapidly as to be easily mistaken for those of meningitis. This is the case sometimes with tubercular tumors, and with glioma of the pons, which may run an almost latent course until they have reached a considerable degree of intensity. The symptoms in the limbs often assist the diagnosis, since the loss of power is more often an early symptom in tumor than in meningitis, and it comes on gradually in the former, whereas early paralysis of the limbs in meningitis usually comes on suddenly, from irritative inhibition. The ophthalmoscopic appearances are of great importance. A slight degree of neuritis may be due to either tumor or meningitis, but an intense neuritis, with considerable swelling and hemorrhages, is practically conclusive of tumor. The neuritis of a rapidly growing tumor is usually intense, and when the appearances are slight at first, if the disc be watched for a few days, the course of the neuritis often decides the diagnosis. It must be remembered that from the absence of neuritis no conclusion can be drawn. In many cases, however, the question can be only decided from the course of the disease. If, after the first two weeks from the commencement, the symptoms continue, slowly increasing, and the patient does not become comatose, the diagnosis of tumor is almost certain. It must not be forgotten that meningitis often co-exists with tumor, by which it is excited, but the symptoms of tumor in these cases have usually preceded those of meningitis, and continue unchanged when the latter have passed away or lessened. The chief difficulty in this respect is met with in the case of tubercular tumors co-existing with tubercular meningitis, but the difficulty is rather in the recognition of the tumor than in that of the meningitis.

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* Legroux, "l'Encéphale," 1885, No. 1.

† Money, "Treatment of Disease in Children," 1887, p. 457.

In some cases of meningeal hemorrhage the symptoms have an acute, not sudden, onset, and may closely resemble those of inflammation over the convexity; headache and delirium are conspicuous; and both diseases may follow an injury. This form of meningeal hemorrhage is, however, very rare. The most important criterion is the absence of fever and the rapidity of course, since traumatic meningitis rarely runs a very acute course.

A considerable difficulty in diagnosis is presented by some cases of disease of the ear, in which symptoms resembling meningitis occur and may prove fatal, although after death only thrombosis in a sinus can be discovered, and sometimes even that is absent. The central symptoms are for the most part general, and may be in part due to pyæmia, but strabismus has been observed, and the difficulty in diagnosis is increased by the fact that optic neuritis may occur and may even reach a considerable degree. It is doubtful whether in these cases a positive diagnosis is always possible.

The same difficulty presents itself in another form in cases of acute double otitis in children, which may be attended with intense pain in the head, vomiting, fever, delirium, giddiness, convulsions, and deafness. Probably in many instances the labyrinth is chiefly affected. An instance of this disease coming on at the age of two and a half is described by Politzer;* the aural lesion was found after death at a later period. Such cases are often thought to be meningitis;† but it is probable that inflammation of the membrane never damages gravely the auditory nerves without the adjacent facial nerves.‡ The general cerebral symptoms alone scarcely warrant the diagnosis of secondary meningitis excited by the inflammation of the ears. It is probable, moreover, that in some of these cases there is optic neuritis. Most practitioners have met with cases in which complete deafness and blindness came on in childhood with such acute cerebral symptoms, and in whom the ophthalmoscope reveals the form of optic nerve atrophy that succeeds inflammation. A series of such cases has been recorded by Mr. Hutchinson.* If optic neuritis may be produced by otitis, it is possible that these cases may be thus explained without the assumption of meningitis. In any case they present a very difficult diagnostic problem, and the difficulty is increased by the fact that the internal ear may be inflamed secondarily to meningitis. Such secondary otitis has been observed in cerebrospinal meningitis, but it is very rare.†

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* Politzer, "Diseases of the Ear," Cassell's translation, 1883, p. 714.

† The facility with which this mistake may be made was pointed out by Votolini (*Monatschr. f. Ohrenheilk.*, *1870*, Nos. 7 and 8) and Reichel (*Berl. kl. Wochenschr.*, *1870*, Nos. 24 and 25).

‡ It has been assumed that the auditory nerves may be so damaged at the base as to cause

The condition of depressed cerebral function that was termed by Marshall Hall *hydrocephaloid*, and is apparently due to anæmia of the brain, may be confounded with tubercular meningitis. In this condition, which is almost confined to young children, there is somnolence and coma, with depressed fontanelle, and local symptoms are absent. It is said, indeed, that rigidity of the neck and strabismus have been observed, but there is some doubt as to the real nature of cases that present such symptoms. The cases are distinguished from meningitis by the depression of the fontanelle, and especially by the occurrence of the symptoms in the profound exhaustion that results from diarrhœa or loss of blood.

Of the general diseases of the nervous system there is only one that is liable to be confounded with meningitis, and especially with tubercular meningitis—hysteria. This error is by no means rare, but occurs only in the cases in later childhood and youth, and especially in the female sex. Meningitis, especially tubercular, is far more frequently mistaken for hysteria than hysteria for meningitis. The former error is very common; it is usually due to the fact that when there exists the state of nervous system that underlies hysteria, pronounced hysterical symptoms are often developed during the early stage of tubercular meningitis. The case is diagnosed as one of hysteria, and when other symptoms develop, they are disregarded, under the influence of the preconceived idea. Even when no hysterical symptoms attend the attack, if such have occurred in the previous history of the individual (as is often the case in tubercular girls), the assumption that the cerebral symptoms are due to hysteria is often made when there is not the slightest justification for it. Indeed, in not a few instances the fact that a girl is the subject of vague general cerebral symptoms is allowed to determine the diagnosis. The only way in which error can be avoided is to

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complete deafness, without any paralysis of the facial. This assumption rests on no evidence, and is most improbable, although, no doubt, as already stated, the auditory nerves may suffer somewhat more readily than the facial in slight inflammation of the membranes.

* *Ophth. Hosp. Rep.*, *1866*.

† Lucae, *Arch. f. Ohrenheilk.*, Bd. 5, 1870, p. 188. It is so rare, and so remarkable in its bilateral character, as to be open to the doubt whether the otitis may not be a coincident effect of the blood state.

search and watch for symptoms of organic origin and allow these the same weight as in a case in which there are no hysterical symptoms, or as in a patient in whom hysteria would not be expected. Pyrexia is of especial diagnostic value in these cases, and so also are convulsions beginning locally, and the ophthalmic symptoms. Strabismus in hysteria is always convergent and attended by spasmodic contraction of the pupils. Divergent strabismus, or inequality of pupil, is certain evidence of organic disease, and as much so if it is transient as if it is permanent. Retention of urine may be due to hysteria, but incontinence never is. The significance of a tendency for the skin to blister has been mentioned; signs of trophic lesions of the skin should be carefully looked for in every doubtful case.*

An illustration of the difficulty that sometimes attends the diagnosis, and of the significance of pyrexia, was afforded by the case of a servant, aged twenty-two, who had had a child three years previously, and had suffered from some headache for six weeks. On account of slight indisposition, her mistress suspected another pregnancy, and, without telling the girl, sent for a medical man to see her. The girl was intensely annoyed, and refused to answer any questions. She went up to her room and fell, bruising her face. All the rest of the day she was dull and lethargic, occasionally, however, throwing her arms about, screaming, and complaining of pain in her head. The symptoms were ascribed to hysteria, but as she was no better next day she was brought to University College Hospital, and admitted. Her temperature was found to be 101°, and the bladder full, so that the catheter was used. For some days she continued dull in aspect and manner, sometimes answering when spoken to, sometimes not. She complained of pain in the head and abdomen. One day she was childish, playing with a doll. The temperature, however, continued raised, varying from 102° to 99°. On the seventh day she became semi-comatose, and passed urine into the bed. On the ninth day, she rather suddenly became dusky, with irregular breathing, and mucus in the chest. Death from respiratory failure being manifestly imminent, artificial respiration was employed, and by this, faradaism to the chest wall from time to time, and food through a long catheter, she was kept alive for twenty-four hours, and died on the eighth day from the onset. Throughout there had been no symptoms in the limbs or cranial nerves. The post-mortem examination revealed general tuberculosis of lungs, peritoneum and intestine, some small masses of yellow tubercle in the cerebral hemispheres, and meningitis of the base, the lymph being specially abundant about the pons and medulla, with opaque tubercular granulations.

The converse error, in which hysterical symptoms are regarded as meningitis, is far less common. The mistake is sometimes made in cases of hysterical sopor with the strong convergent strabismus, but the distinct spasmodic character of the latter is usually distinctive, and there is, as a a rule, no alteration of temperature. The last point is also of diagnostic importance in the curious state of trance-like sleep that sometimes comes on in states of brain exhaustion, usually in hysterical subjects, but in lads as well as girls. When this succeeds severe headache, as it often does, the difficulty of diagnosis may be great. But it should be remembered that it is rare for coma to come on early in the course of the symptoms, and that the absence of associated and conclusive symptoms is also rare in the stage of coma. In the case of meningitis in which contracture involves the muscles of mastication, the disease may be mistaken for commencing tetanus. A short time, however, suffices to dispel the error, since meningitic trismus soon passes away.

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* It should be remembered that these trophic disturbances are often not mentioned, or are even denied, by nurses, who fear that the sores may be ascribed to carelessness.

PROGNOSIS.—In every form of meningitis the prognosis is grave; it is least serious in the traumatic form, and in simple meningitis from adjacent disease, and it is most grave in the purulent form, recovery from which is practically unknown. Nevertheless I have twice known recovery from distinct symptoms of meningitis in post-puerperal septicæmia. In any form, if the stage of coma has been reached, death is all but certain.* In tubercular meningitis there is very little hope, in any stage, that the patient will recover. But the patient has some small chance of recovery in simple meningitis, and perhaps (although still lighter) in tubercular meningitis; and, moreover, the very important fact must be borne in mind that the diagnosis between the two, and between these and meningitis secondary to obscure adjacent disease, is a matter of probability only, however high the probability may be. Hence it is not right, in any case, to assert the *certainty* of a fatal issue.

As an illustration of this I may mention the case of a boy, five and a half years old, with tubercular family history, who, after five days of vague general indisposition, became feverish, with severe headache and double vision. He soon became so weak as to be unable to walk. I saw him after the cerebral symptoms had lasted for five days. The child appeared very ill; the temperature was 101°; the tongue was covered with a thick white fur, both sixth nerves were paralyzed, and there was weakness of the left side of the face. There was no history of injury or discharge from the ear. The case was certainly one of meningitis, and it seemed highly probable that it was tubercular. Two days later, however, there was a sudden and copious discharge of pus from one ear, the cerebral symptoms rapidly disappeared, and the boy recovered perfectly.

In all cases the most material prognostic indication is afforded by the course of the disease. The less acute the attack, the more chance of recovery the patient has. If, at the end of the third week from the onset, the patient has not passed into a state of coma, there is an appreciable diminution in the probability of death. The chance that death may be escaped is least of all when the coma comes on before the first week is over.

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* A case of recovery from an illness resembling tubercular meningitis, in which the stage of coma was reached, and the child seemed for days to be on the point of death, is recorded by West ("Diseases of Infancy and Childhood," 7th ed., p. 96). It is said, however, that the child became blind and afterwards recovered her sight, a feature which does not suggest tubercular meningitis.

Treatment.—If the meningitis is due to adjacent disease, the treatment of this is of the first importance. If none is obtrusive the ears should be carefully examined, since, as the case just mentioned shows, suppuration in the middle ear may excite meningitis even though no discharge has previously been noticed. A free exit should always be made for any collection of pus in the neighborhood of the skull, and if there are any indications of the presence of pus in the tympanic cavity, the membrane should be incised. If no such cause can be discovered, the treatment must be directed to the diminution of the local inflammation and the counteraction of the general state on which it depends. Neither the seat nor the form of inflammation has much influence on the treatment. This has to be conducted in each case on the same general principles; variations being determined by individual differences much more than by pathological nature.

In no disease is perfect tranquillity of greater importance. The patient should be disturbed as little as possible, kept free from all excitement and all mental exertion; the room should be darkened if there be any intolerance of light, and in all cases it should be kept as quiet as possible. But mental depression should be avoided only less carefully than mental excitement. Sleep should be encouraged, and the patient aroused only for the purpose of feeding. Light nutritious food should be given every three or four hours, and if the patient cannot be made to swallow, nutrient enemata should be administered. Stimulants are best withheld unless the state of the pulse urgently calls for them. The head should be high, but the shoulders also raised to avoid flexion of the neck, and mechanical hindrance to the return of the blood from the head.

Abstraction of blood, while undoubtedly in many cases a powerful agent in lessening local inflammation, is rarely desirable in meningitis. Exceptions to this rule are the forms that follow injury, exposure to the sun, or excessive mental excitement, and meningitis for which no probable cause can be traced. In these, if the patient be strong and full-blooded, leeches may be applied behind the ears or to the temples in the early stage of the disease. During the late period abstraction of blood can do only harm. In strong children leeching has also been recommended, but the cases are few in which this is desirable. Most cases occur in weakly, anæmic individuals in whom loss of blood reduces the strength of the inflammation less than it does the strength of the patient. Attempts may be made to keep the blood away from the head by warmth and mustard to the extremities, and by cold to the head. The hair should be cut short or the head shaved. The application of cold should be continuous, either by an ice bag (half filled, and containing no air, so that it may adjust itself to the head), or by a cap made of metal tubing, wound in a spiral, through which water may flow from a vessel above the level of the head to one on the floor.* This is a very convenient mode of applying cold, as effective as ice, and available when ice cannot be obtained.

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* Such caps can be obtained from Krohne and Sesemann, Duke Street, Manchester Square.

Counter-irritation to the occiput is also sometimes distinctly useful. Mustard may be employed, but a blister is more effective. There is a natural reluctance to add the pain of a blister to the sufferings of the patient, but a blister certainly often lessens the intensity of the symptoms, and it is probable that the total amount of pain endured is at least not increased thereby. Counter-irritation over the whole scalp, by blister or irritating ointment, has sometimes been recommended, but it interferes with the application of cold, and it is doubtful whether its influence is such as to counterbalance this disadvantage.

Vomiting is best allayed by ice, and by two mustard plasters applied to the head and given by the mouth; one to the back of the neck, and the other to the epigastrium. The bowels should be opened with a freedom which must depend on the strength of the patient. Constipation is often difficult to overcome, but it is injurious, while purgation is one of the most effective derivatives.

Internal remedies must vary with the state of the patient. In all cases with elevation of temperature a diuretic is safe and often useful. Digitalis in small doses may be given if the pulse is irregular or unduly frequent in the early stage. When the patient was previously anæmic and the temperature is not very high, iron may be given in some non-irritating form, such as dialyzed iron, or the citrate of iron and quinine, but the bowels must be carefully kept open.

Various drugs have been given with a view of influencing directly the process of inflammation, and each has been in turn extolled. It is needless to say that mercury has had the greatest number of advocates, and there is strong evidence that it occasionally does good, at any rate in simple meningitis. Of its value in tubercular meningitis there is more room for doubt. It is easy to deny, and impossible to prove, that cases which run a favorable course are tubercular; but for practical purposes there is the correlative fact that it is also rarely possible to be certain that any case under treatment is not simple. If mercury is employed it should be promptly used. The patient should be brought slightly but distinctly under its influence, so as just to "touch the gums," as the phrase is, as quickly as may be. Inunction is by far the best method of effecting this. A little mercurial ointment should be rubbed into the armpits and groins every four hours, until the effect is produced. Iodide of potassium is another remedy that has been much used, but the evidence of its value is slighter than is that of mercury. It may be, however, conveniently combined with a tonic in cases with much debility, or after the effect of mercury has been produced.

When the temperature is very high, cold baths have been employed with some apparent temporary advantage, but the almost invariably fatal issue has not been retarded by the reduction of temperature. The same may be said of salicin and quinine given for the same purpose.

In cases of septicæmic meningitis, what slender chance of benefit there may be is most likely to be by the free administration of perchloride of iron, a drug which, more than any other, has seemed to me to have the power of saving life in septicæmia. This treatment was adopted in each of the cases of apparently septicæmic meningitis which recovered. In one case there was severe headache and delirium, rigidity of the limbs on one side, and a temperature of 105.5.°

The treatment of acute ventricular meningitis does not differ from that of extra-cerebral meningitis, a fortunate circumstance, since the recognition of its limitation is not practicable during life.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

(CEREBRO-SPINAL FEVER.)

Acute meningitis sometimes occurs in epidemic form, many patients being attacked in a certain district in the course of a few months. The spinal membranes are affected as well as those within the skull, and hence the name by which the disease is known; but, as we have seen, other forms of inflammation may not be limited to the cerebral membranes. There is usually considerable fever, and often an eruption appears in the skin. Sporadic cases occur, closely resembling those of the epidemic form, and probably identical in nature.

The disease has been met with during the present century in various parts of Europe and in the United States. In certain countries, as Sweden and Germany,* it has been especially prevalent. In Sweden alone, 4000 persons are said to have died from the disease between 1854 and 1860. A very severe epidemic occurred in France in 1837. In Great Britain, the chief epidemics have occurred in Ireland, especially in 1846 and 1868; in England only slight outbreaks have been recorded, while in Scotland one small group of cases has been met with.† It has been doubted whether the isolated cases of acute severe and primary meningitis ought to be classed with the epidemic form, but they are not separable by any clinical or pathological features, and they have a distinct tendency to be multiple, which seems to associate them clearly with the epidemic form. Thus in the spring of 1887 four cases were admitted to University College Hospital, and two others came under my notice in other parts of London. Three of the cases occurred in the same street; and two others, though quite separate, developed within a quarter of a mile of each other.

Causes.—No age is exempt, but young persons under twenty suffer much more frequently than others, and in some epidemics children have been almost exclusively affected. Males are said to be attacked more frequently than females.* As it is a disease chiefly of temperate and cold countries, so it has prevailed chiefly in winter and spring, very seldom in autumn and never in summer. No relation has been traced, as a rule, to local endemic influences, although some observers who have watched epidemics have thought that the incidence of the disease suggested some malarial or miasmatic influence. But it has broken out simultaneously in places far apart, and has even been prevalent at the same time in Europe and America. Personal ill health seems to have but little predisposing influence, but insanitary conditions and overcrowding apparently favor its occurrence. The relation to season has been explained by the overcrowding in the houses of the poor during cold weather.† In one epidemic in France the only persons attacked were soldiers crowded together in barracks; another in Ireland fell exclusively on the inmates of workhouses. Nevertheless, contagion seems to play a very small part in the production of the malady. The attendants on the sick scarcely ever suffer. In an epidemic of thirty cases recently in Cologne no two cases came from the same house.‡ Nevertheless, instances have been observed in which an influence exciting the disease seemed to be conveyed by a third person. A series of such facts has been collected by Hirsch, and Frew believed that he traced such a connection between the cases in the small Scotch outbreak. But in the vast majority of instances no evidence of contagion can be traced. Although even very young children are prone to suffer, a woman who had a mild attack, in the course of an epidemic, suckled her child throughout her three weeks' illness and the child remained perfectly well.§ It is said that, in Ireland, each epidemic coincided with an outbreak of a similar malady in pigs and dogs.|| The malady does not seem to confer an immunity against a second attack. A woman died from the disease during one epidemic who had passed through a similar attack five years before.¶

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* It is curious that in 1862 Hirsch wrote, "As far as I know, Germany has been entirely spared, with one small exception," and that, according to Ziemssen, since the first distinct outbreak, in 1863, hardly six months have passed without an epidemic in some part of Germany.

† Frew, *Glasgow Med. Journal, 1884.*

SYMPTOMS.—The manifestations of the disease are in part those of the local inflammation from which it takes its name; in part they are those of a blood disease. In cases of moderate severity, malaise and discomfort in the head may precede the onset for a few hours or for two or three days, seldom for a longer time than one or two weeks.** Sometimes there is vomiting, or slight stiffness at the back of the neck, for a day or two before the acute symptoms come on. In other cases the onset is almost sudden. Vomiting and headache are usually the first pronounced symptoms; their commencement may

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* Of 255 fatal cases in Stockholm, 149 were boys, 106 girls.

† Medin, *Nord. Med. Ark.*, *1880*, and *Deut. med. Wochenschr.*, *1881*, Nos. 41, 42.

‡ Leichtenstern, *Deut. med. Wochenschr.*, *1885*, No. 31.

§ Rzadowski, *Virchow's Jahresb.*, *1879*, ii, 5.

|| Fagge, "Principles and Prac. of Med.," i, 600, on the authority of Ferguson, Vet. Off. to the Privy Council of Ireland. Epidemic cerebro-spinal meningitis has also occurred in horses; an outbreak of 37 cases was observed by Ackermann (*Virchow's Jahresb.*, *1880*, i, 701).

¶ *Virchow's Jahresb.*, *1879*, ii, 5.

** Sabarth, *Breslauer Arzt. Zeitsch.*, *1879*, No. 18.

be attended by a rigor; sometimes there is also a pain in the back. The headache varies in its initial seat, but it often soon becomes general, and is always severe; it is constant, but there are from time to time intense exacerbations. Often the headache is accompanied by giddiness, and, as in other forms of meningitis, by intolerance of light and sound. Delirium in many cases is quickly added to the headache; it may be quiet or violent, but it soon gives place to somnolence and stupor, from which the paroxysms of violent pain at times may rouse the patient. Severe pain in the back is generally soon added to the pain in the head; it is chiefly in the neck and loins, and often referred to the neighborhood of the spine, rather than to the vertebral column itself. Sometimes it is severe in the sacrum. It is increased by movement, and may radiate around the trunk, or into the limbs; it is sometimes severe in the knee joints. Whether there is pain in the back or not, rigidity in the muscles of the neck is almost invariable, and in those of the back is frequent. This gives rise to the characteristic retraction of the head, usually moderate in degree, sometimes so great as to cause the neck to be at right angles to the rest of the spinal column. Often the contraction seems to be chiefly in the deeper muscles. Any attempt to flex the neck gives rise to pain. The retraction of the head is usually an early symptom, although it is not often present at the actual onset; sometimes it is delayed until the later stage of the disease. The rigidity of the rest of the spinal muscles is generally merely enough to prevent bending of the trunk, but it is occasionally so great as to cause opisthotonos. The limbs also are sometimes rigid; the legs are drawn up, and the abdomen is retracted. Rarely there has been slight trismus. General hyperæsthesia of the skin is a frequent symptom and of some diagnostic importance. Convulsions occasionally occur at the onset of the malady or during its course; they are usually general, sometimes unilateral or local.

The face is often pale and has a shrunken aspect. The pulse varies much in frequency; in some cases it is not much above the normal, while in others it rises to 120, 140, or more. It is not often infrequent. The temperature is almost always raised, and it is generally considerably raised, often reaching 104°, 105°, or 106°, the greatest amount of pyrexia being noted toward the termination of the disease. Both the temperature and pulse-frequency present irregular variations, sometimes together, more often without any correspondence. The bowels are generally confined; the spleen is rarely enlarged. The amount of urine has been sometimes increased, and albumen is occasionally present; sugar rarely.

An important feature of the disease, more common in epidemics than in sporadic cases, but varying much in different outbreaks, is the occurrence of eruptions on the skin. Various forms have been met with—erythema, herpes, urticaria, purpura; but the most significant are the herpes and the purpuric spots. The latter are met with in a large proportion of the most severe cases, and are occasionally present in attacks of moderate severity. They may coalesce so as to give rise to dark diffuse extravasation into the skin over a considerable area. Herpes is very common in some epidemics;

in one outbreak of twenty-nine cases herpes was present in twenty-six (Leichtenstern). In another, of thirty-two cases, only six developed herpes (v. Sydow). It usually begins on the lips and may spread to other parts of the face, but is sometimes observed on the limbs. These eruptions frequently present bilateral symmetry, and the several forms are sometimes conjoined. They usually appear after the disease has lasted three or four days. Occasionally there is intense conjunctivitis and even ulceration of the cornea.

Paralysis of cranial nerves, strabismus, inequality of pupil, etc., may be met with in this as in other forms of meningitis. The pupils are dilated, sometimes after initial contraction, and may be unequal. Deviation of the eyes to one side has been occasionally observed. Optic neuritis is common in cases that last for more than four or five days, and may cause permanent loss of sight, if the patient recovers. Palsy of the limbs is not common; when complete hemiplegia occurs early it is often due to irritative inhibition, but sometimes at a later period it is due to a focus of more intense inflammation over the motor region.* Occasionally there is paralysis of part of one side from this cause. In rare instances the symptoms of the spinal meningitis predominate over those of the intracranial inflammation, and complete paraplegia may result.† Myotatic irritability is usually lost in the legs in such cases, and sometimes toward the close in others, in which spinal symptoms are not specially marked.‡

Among the occasional defects of the disease, deafness is of special importance. It seems commonly to depend, not on damage to the auditory nerve at the base of the brain,§ or to the medulla, but on extension of inflammation from the membranes to the labyrinth and middle ear, an extension that occurs with peculiar readiness in the young. The affection of the middle ear is shown, not only by the interference with conduction, but also by injection of the membrana tympani, and sometimes by the discharge of pus through it. The otitis media may be secondary, in time, to that of the labyrinth, or may occur alone. It is a question whether the affection of the internal ear is always secondary to the meningitis. There are many passages by which inflammation may spread from the membranes to the labyrinth. Nevertheless, it has been held by some that the otitis is simultaneous and primary, and the inconstancy, indeed absolute infrequency, of the labyrinthine inflammation gives some support to this view. It is certain that bilateral labyrinthine inflammation in children is sometimes a primary disease; we know little of its causes, but it is possible that they are allied to those which produce meningitis.

The delirium that attends the early stage of the disease soon gives place to a condition of stupor deepening to coma. The period at which this comes on varies according to the severity of the case; the delirium may last a few hours only and the patient may become comatose before the end of the first day, or the coma may only come on at the end of a week. In cases that terminate fatally, the coma is attended with signs of failure of the heart, and by sighing or irregular breathing, and the temperature may fall, or may rise to an extreme degree.

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* An instance is recorded by Charlewood Turner, *Path. Trans.*, *1884*.

† Strümpell, *Deut. Arch. f. kl. Med.*, Bd. xxx.

‡ Carrington, *Path. Trans.*, *1884*, xxxv, pp. 54, 55; Leichtenstern, loc. cit.

§ See note on p. 745. Infiltration of the sheath of the auditory nerve with pus has been found when no deafness was noted, and there has generally been evidence of suppuration in the labyrinth when there has been absolute deafness.

The variations in the severity of the disease are great and are accompanied by corresponding variations in duration. In the most acute cases the patient quickly becomes comatose, and dies at the end of one or two days, sometimes even in five or six hours from the onset. The acute form has been termed "fulminant." On the other hand, slight cases sometimes occur, in which the illness is trifling, and the symptoms consist in headache, pain in the back, and slight rigidity of the neck muscles, a form that has been termed, somewhat inaptly, "abortive." A remarkable epidemic occurred at a village in Lincolnshire a few years ago, in which all the cases were of this form. As in the case of other epidemic diseases, the severe cases are most frequent at the commencement, and the slight cases during the decline of the epidemic. The most acute cases are often attended by extensive cutaneous extravasation, and death seems to be due rather to the blood change than to the meningeal inflammation. In the less severe form, death generally results from asthenia, increased often by bedsores. In cases that recover, the patient begins to mend some time during the second week. The mortality has varied, in different epidemics, from twenty to eighty per cent.; in sporadic cases it is very high.

Besides the varieties that depend on severity, other forms have been occasionally met with. The fever has been observed to distinctly intermit, somewhat after the type of a quotidian or tertian ague, but with much less regular variations in the temperature than are presented by true intermittent fever. Other cases in which the fever and a low asthenic state continue for a considerable time, have been described as a "typhoid" form.

A complication of epidemic meningitis that deserves special mention is pneumonia. This has been met with in many cases, and has been more frequent in some epidemics than in others. It is seen also in sporadic cases, and is of importance from its pathological suggestiveness. Bronchitis is also common.

Among other rare complications may be mentioned tonsillitis, multiple arthritis (sometimes suppurative),* endocarditis, and pericarditis. Combinations of cerebro-spinal meningitis with other maladies, such as scarlet fever, have also been described; it is doubtful whether the combination has been more than a coincidence.

The process of recovery is generally very slow, and is occasionally interrupted by a distinct relapse. Headache persists for a long time, and a lasting liability to headache may succeed the disease. Of the sequelæ of the disease the most important is the deafness from inflammation of the ear. It may be complete, and, occurring as it often does in children, it may cause a loss of any power of speech that has been acquired, and permanent deaf-muteness. In some countries an epidemic of this disease has added enormously to the proportion of the population who were deaf and dumb. With the deafness there is often, for a time, a difficulty in maintaining equilibrium, due, no doubt, to the damage to the semicircular canals. It gradually passes away as compensation for the loss becomes established.* Noises in the ears may also persist for a long time. Another consequence occasionally observed is chronic internal hydrocephalus, which may cause its characteristic symptoms some weeks or months after the primary disease. It probably depends, in some cases at least, on its common cause, closure of the openings of the fourth ventricle, but it may sometimes be due to inflammation of the lining membrane of the ventricles (Merkel). An excess of fluid is sometimes found outside the cord.

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* Medin, loc. cit.

PATHOLOGICAL ANATOMY.—In the most acute fulminant cases, there may be only that transudation of hæmatin into the fluids of the body and rapid decomposition which attend all intense toxæmic states, together with congestion of the membranes; in these the microscope may show collections of lymphoid cells along the vessels, or red corpuscles infiltrating the tissue. In cases of less rapid course there is intense hyperæmia of the pia mater of the brain and cord, with opacity and exudation of lymph, and in most cases that have lasted more than three or four days, there is distinct formation of pus, which may accumulate in the subarachnoid space. It is more abundant over the posterior than over the anterior surface of the cord. The dura mater of the brain is little affected, but there are commonly some signs of inflammation on that of the cord. The ventricles of the brain may contain turbid fluid, sometimes pus, and their lining membrane may present signs of inflammation. Thrombosis in sinuses is rare. The substance of the brain may be pale, or contain small hemorrhages or points of softening, or small collections of pus. The spinal cord may also be inflamed and softened in places. The lesions in other organs of the body are such as result from other acute febrile blood diseases. The spleen and follicular glands of the intestinal canal may be found enlarged, but the enlargement of the spleen is slight and has seldom been detected during life. The kidneys are sometimes in a state of acute parenchymatous inflammation, as in other acute general diseases. The lungs often present signs of congestion, and endocarditis has been met with.

PATHOLOGY.—All the facts of the disease point to the existence of a specific poison acting on the blood, and through the blood exciting the local inflammation. It is, however, to the latter that the symptoms are chiefly due, in all save the most intense cases. The alterations met with in other organs seem to prove the constitutional nature of the affection. But of the nature of the influence which causes the disease, and the way it spreads, little is known. It is clear that children possess a special liability, which cannot be explained, as in so many specific diseases, by the mere absence of the protection afforded by a previous attack. The very slight part played by personal intercourse in the transmission of the disease has been already mentioned. The manner in which disconnected cases appear about the same time, has suggested an analogy to influenza, but it is possible that a still closer analogy exists between this disease and some forms of pneumonia, with which it is not unfrequently associated. The occurrence of herpes of the lips in each disease is a small but noteworthy point of resemblance. Pneumonia has been observed to be especially frequent at the time of epidemics of meningitis.* It is highly probable that one form of pneumonia is due to some specific influence, and a special micrococcus has been observed in connection with it. When meningitis accompanies pneumonia, the same organisms have been found in the cerebral membranes.† The proved dependence of many acute specific diseases on microörganisms has suggested the probability that epidemic meningitis is due to a similar cause.‡ Organisms have been repeatedly searched for, and have been found, although not frequently. In a case recorded by Gauchier,§ the urine, which was albuminous, contained a large number of micrococci, and similar organisms were found in the blood during life, and, in great abundance, in the exudation in the membranes after death. Similar organisms were found by Leyden, in the cerebro-spinal fluid around the cord as well as in the pia mater,‖ and he has pointed out their resemblance to those that are associated with pneumonia and with erysipelas, but notes, as differences, that they are somewhat larger, more distinctly oval, and that some are wider, and present indications of division.

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* Moos, "Meningitis cerebro spinalis epidemica, etc.," Heidelberg, 1881.

Diagnosis.—The symptoms of meningitis, the severe headache and coincident delirium, the retraction of the head, the cutaneous hyperæsthesia, etc., are generally sufficiently distinct to prevent any confusion of the disease with other febrile maladies. The pain in the back, vomiting, and headache may suggest smallpox, but in cases in which the pain in the back is severe, the muscular rigidity usually soon manifests itself. It is said that in some cases of typhoid fever there is hyperæsthesia of the skin and some tenderness of the muscles of the neck (Leyden), but these symptoms quickly lessen, whereas in meningitis they increase. The herpes of the face may help to distinguish the disease from the continued fevers. In tetanus the opisthotonic spasm is generally secondary to trismus, which is scarcely ever an early symptom in meningitis.

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* Medin, loc. cit.

† Eberth, *Deut. Arch. f. kl. Med.*, xiii, p. 1; Nauwerk, *Deut. Arch. f. kl. Med.*, xxix, p. 1; Senger, *Arch. f. Exp. Path.*, xx, 389; Cornil et Babes, "Les Bacteries," Paris, 1886, p. 446. It has generally been assumed that the meningitis complicating pneumonia is due to an infection from the lung. While this theory is supported by the cases in which the cerebral inflammation comes on several days after the onset of the pneumonia, it seems inadmissible in the cases in which the two develop simultaneously, or nearly so, or in which the intracranial disease precedes the other.

‡ See Ziemssen, "Handbuch der Spec. Path., etc.," ii, Th. 2.

§ *Gaz. Méd. de Paris*, March 5, 1881.

‖ *Cent. f. kl. Med.*, No. 10, 1883. See also Leichtenstern, loc. cit. Rod-shaped bacteria were found in the pia arachnoid in a very acute case by Brigidi and Banti, *La Salute, 1883*, No. 23.

Uræmia may cause muscular rigidity, convulsions and coma, thus occasionally giving rise to symptoms somewhat like those of cerebro-spinal meningitis (Murchison), but the temperature is normal, and other symptoms of each malady are usually recognizable. It must be remembered that in children retraction of the head may occur from rheumatic affection of the muscles and other causes (see p. 744).

The greatest difficulty is the distinction of this from other forms of meningitis, and the question whether sporadic cases are to be regarded as examples of the disease. In other forms of cerebral meningitis, spinal symptoms are seldom conspicuous, but it must be remembered that, in the epidemic form, there may not be more than retraction of the head. In tubercular meningitis, the onset is more insidious, and coma is a rather late symptom. The occurrence of the skin eruptions, or the prevalence of the disease in an epidemic form, give material help to the diagnosis. The identification of isolated cases is a subject on which opinions differ. My own belief is that the cases of acute, severe, rapidly-fatal inflammation of the cerebral and spinal membranes are not separable from the epidemic disease, and I think the opinion is supported by the frequency with which pneumonia co-exists. The group of cases referred to on p. 752, of which three came from one street within a few weeks, must, I think, be regarded as belonging to the epidemic form, but these, and the other cases that occurred at the same time elsewhere in London, differed in no respect from the isolated cases that are met with every year. In at least one case of this group there was pneumonia.

The diagnosis, from this disease, of simple meningitis complicating pneumonia is difficult, because the pathological distinction is uncertain. It is very doubtful whether the cases in which the two diseases coincide in time, or are separated in onset by two or three days only, should not be regarded as belonging to the cerebro-spinal form. A considerable diagnostic difficulty is presented by cases in which the meningitis runs an almost latent course. It is sometimes found, after death, in cases of pneumonia in which it was not suspected during life, the headache and delirium having been ascribed to the pulmonary malady. Unequivocal symptoms of cerebral mischief, however slight they may be, should always receive attention in this disease. General hyperæsthesia sometimes first suggests the presence of more than the lung disease.

Lastly, when the disease has been epidemic, some of its symptoms have been produced by the influence of fear, in what has been termed "meningitophobia," and the distinction of this from the "abortive form" has sometimes been difficult. The absence of fever and of definite objective symptoms usually suffices.

PROGNOSIS.—The disease is most serious, except in the slightest form, but even in this a benign course cannot be counted on with certainty. Recovery can scarcely be looked for if coma comes on before the fifth day. The prognosis in different epidemics must be influenced by the prevailing character of the disease; it is, as a rule, decidedly worse in the isolated cases than it is in epidemics. It is said to be least grave between ten and fifteen years. If there is inflammation of the labyrinth, some degree of permanent deafness is probable. In cases in which hearing is lost, any return of the perception of sounds conducted through the bone is of favorable significance.

TREATMENT.—No agent has appeared to exert any specific influence on the course of the disease. Its treatment does not differ from that suitable to other forms of meningitis, but there is perhaps less to be hoped for from mercury. Among the other drugs that have been used, for the most part without distinct result, may be mentioned iodide of potassium, salicylate and benzoate of soda, quinine, digitalis, and chloral. Iodide of potassium and salicylate of soda have, it is true, been credited with a beneficial influence in a few cases. For the prevention of the disease, fresh air and the avoidance of overcrowding are the most important measures.

ORGANIC DISEASES OF THE BRAIN.

ANÆMIA OF THE BRAIN.

The blood within the brain is contained in arteries, capillaries and veins. The functional condition of the brain depends on the quantity and quality of the blood circulating in its capillaries, and it is to this that the special symptoms are related. Deficiency in the quality of the blood supplied to the brain is always of gradual occurrence, and affects the whole brain; deficiency in quantity of the circulating blood may affect the whole brain or part only, and it may be sudden or gradual in its production.

CAUSES.—*General cerebral anæmia* may be due to the following causes: (1) It may be a part of systemic anæmia—defect in quantity or quality of the whole blood, due to causes which are considered elsewhere. This is often seen in cases of hemorrhage, of exhausting discharges, or of defective blood nutrition, as in chlorosis. (2) The supply of blood to the brain may be deficient, the quantity of blood in the body being normal. This may be due to cardiac weakness, or to causes acting through the nervous system on the heart, as in swooning. In systemic anæmia, the lessened cardiac power increases the cerebral deficiency. Whatever lessens the amount of blood discharged from the heart, such as aortic obstruction or mitral disease, may be a cause of cerebral anæmia. Pressure on the vessels conveying the blood to the head, as by an aortic aneurism, has a similar effect. Unequal distribution of the systemic blood is another cause. The intestinal vessels, if dilated, are capable of containing a large part of the blood of the body, and the effect of their engorgement is often seen after paracentesis abdominis. A patient suddenly sitting up, after the abdominal vessels have been thus relieved from pressure, has been known to fall back dead. One theory of shock ascribes its mechanism to vasomotor dilatation of these vessels, and consequent anæmia of the rest of the system. The effect of each cause is increased by the action of gravitation in the erect posture. Some causes act only in that position. (3) Cerebral anæmia has been supposed to occur during the exhaustion after the acute stage of febrile diseases, and to be the cause of certain cerebral symptoms that may attend this stage. The mechanism is, however, uncertain, since an equal amount of prostration and cardiac weakness is more frequently unattended by these symptoms.* (4) The capacity of the cerebral vessels may be diminished by pressure on the brain, exerted by effusions of fluid (hydrocephalus), of blood (in cerebral and meningeal hemorrhage), or by growths within the skull.

Partial cerebral anæmia is due to some obstruction to the passage of the blood through the vessels. To be permanently efficient such obstruction must be situated beyond the circle of Willis. Ligature of one carotid causes immediate symptoms of cerebral anæmia, but permanent symptoms are not frequent. Pressure on or disease of one carotid, for the same reason, rarely gives rise to symptoms. Obstruction in certain arteries of the brain may cause local anæmia, sudden or gradual, temporary or permanent, according to its cause. Such obstruction may be due to narrowing of the calibre of the vessel by atheromatous changes in its wall, or by spasm of its muscular coat, or may be due to actual occlusion by embolism or thrombosis. The pressure effects of an intruding substance within the skull (tumor, or clot) act most intensely in, and may influence only, one region of the brain.

It is obvious that of these causes some act suddenly, others gradually, and the symptoms produced will differ accordingly.

PATHOLOGICAL ANATOMY.—The principal anatomical character of cerebral anæmia is pallor of the brain, observable chiefly in the paler tint of the cortical substance, and the diminished number of red spots in the white centre. The pallor may be partial or general. But pallor of the brain after death does not by any means necessarily show that anæmia existed during life. The amount of blood in the brain depends chiefly on the mode of death. The membranes are usually pale in anæmia, but in some cases of partial anæmia they are hyperæmic. In general anæmia, effusion of serum in the meshes of the pia mater and between the convolutions may be found, and it is even said that the brain substance may be œdematous, and the nerve cells changed in aspect, unduly translucent, or unduly granular. The walls of the minute vessels may also be found degenerated, thickened and homogeneous in appearance, a condition which may aid in the interference with function.†

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* It is quite possible that the symptoms referred to, mental derangement, etc., may be an after effect of the action of the specific poison on the nerve centres.

† Knoll, *Wien. Med. Wochenschrift, 1885*, No. 51.

SYMPTOMS.—The symptoms of this condition vary according as the anæmia is suddenly or slowly produced, and as it is general or partial.

(1) In *sudden* general anæmia of the brain the sufferer feels drowsy; the special senses are dulled; noises in the ears and vertigo are complained of; the pupils are at first contracted; sight may fail; muscular power is weakened; respiration is sighing; the skin is pale, cold, and moist; nausea is common; and headache is rare. If the anæmia is more intense, consciousness is lost, there is universal paralysis; and general convulsions may occur, epileptiform in character, these being especially frequent in sudden extensive loss of blood in strong subjects. Nystagmus is sometimes observed. The pupils dilate,* and the coma may deepen to death. The loss of sight in cases which recover may persist as permanent amaurosis; but this is probably due to an affection of the retina. In syncope the patient may remain blind for some minutes after the mental functions are restored. The somnolence may increase to coma.

(2) When general anæmia of the brain is *slowly* produced, the state of the cerebral functions is usually that of "irritable weakness." Their action is imperfect in degree, and excited with undue facility. There is mental dullness and drowsiness; sometimes, however, insomnia is troublesome. There are often hallucinations of the special senses, and it is said that maniacal attacks or melancholic depression may occur. Delirium is common in severe cases, and in some forms of imperfect blood nutrition, the so-called "inanition delirium." Headache, usually general, is a common symptom. Sensory hyperæsthesiæ, tinnitus aurium, muscæ volitantes, and vertigo are frequent. Sometimes the sight is dim, or there may be deafness, especially in the upright posture. Convulsions are rare, but muscular power is generally deficient. All these phenomena are more marked in the erect than in the recumbent posture, especially when the erect posture is suddenly assumed. It has been remarked that some anæmic persons can think well only when lying down.

In young children, after exhausting discharges, as diarrhœa, symptoms referable to cerebral anæmia sometimes occur—somnolence and pallor, with depressed fontanelle and contracted pupils; occasionally there is strabismus and even rigidity of the neck. The somnolence may deepen to coma, the pupils dilate and lose their sensitiveness to light, and death may occur. Such symptoms have been called *hydrocephaloid* (first by Marshall Hall), from the resemblance to those of acute hydrocephalus.

(3) Partial cerebral anæmia causes, if complete, loss of function in the affected area; and if it is permanent, as in obstruction of a vessel beyond the circle of Willis, necrosis of the cerebral tissue results (see Softening of the Brain). If incomplete and sudden, there is temporary arrest of function. Ligature of one carotid, for instance, causes transient weakness and

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* The early contraction is probably due to irritation of the third nerve iris centre, the subsequent dilatation to its paralysis (see Mayer and Pibram, *Prager Zeitschrift f. Heilkunde*, Bd. v, p. 15).

numbness in the opposite half of the body. There may be at first an over-action of gray matter, causing, in certain regions, unilateral convulsions. If slowly developed, as in narrowing of arteries, pain and vertigo are common, with recurring local symptoms, such as numbness, tingling and weakness.

In all cases of long-continued cerebral anæmia, lasting damage to the nutrition of the brain may result. In the child, the development of the brain may be arrested; in the adult, loss of memory and of general mental power indicate the deterioration of nutrition. In all varieties of chronic anæmia these symptoms are common, and sometimes take the form of chronic insanity. The simple failure usually passes away sooner than the pronounced derangement, but often not till long after the blood state has improved.

Pathology.—The symptoms are, as already stated, dependent mainly on the defective quantity and quality of the blood circulating in the brain. Some influence has been ascribed to the diminution in the blood pressure to which the nerve elements are ordinarily exposed (Burrows); a mechanical influence, of which the possibility cannot be denied and the probability cannot be proved. The precise cerebral disturbance on which the symptoms directly depend is uncertain. Doubtless the cortex is the first to suffer in function, and the mental symptoms are thus produced. The convulsions that are produced by sudden anæmia of the brain are usually ascribed to disturbance of centres in the medulla or pons, but this is rendered improbable by the fact that compression of one carotid, which can affect only the cerebral hemisphere, has caused convulsion in the opposite half of the body. The convulsion is preceded by tingling in the whole of that side. That the nerve cells should "discharge" when the blood supply is arrested, is a fact of very great physiological interest as an indication of the reserve of force that must be stored up in the nerve cells, and of the probability that sudden over-action is due to diminution of resistance to action, and not to an increase in the force-generating function of the cell. Latent energy may be liberated, but new force can scarcely be produced under the influence of sudden anæmia. The disturbance of respiration, its sighing character, etc., are ascribed to the derangement of the respiratory centre in the medulla, but we cannot be sure that this is always the case. Such breathing has been observed to follow obstruction of the carotids. It must be remembered that our consciousness of dyspnœa due to disturbance of the respiratory centre must be produced through related cerebral centres, and a primary disturbance of the latter may act on the respiratory centre, as in the sigh of grief.

Diagnosis.—The diagnosis is not difficult. It rests on the recognition, in a given case, of the causes of cerebral anæmia, and on the exclusion of graver maladies, such as organic cerebral disease. With the latter, it should be remembered, anæmia of the brain, local or general, often co-exists. Some symptoms of hyperæmia of the brain closely resemble those of anæmia. A common pathological state of imperfect blood renewal probably exists in both conditions.

PROGNOSIS.—The severity of the symptoms and the extent to which the cause of the anæmia is amenable to treatment, and is of transient character, must determine the prognosis. As a rule this is favorable when there is no organic disease of heart, vessels or brain. In the so-called "pernicious anæmia" the prognosis is, of course, far more grave. Hydrocephaloid symptoms in infants, if met by prompt and suitable treatment, are usually recovered from.

TREATMENT.—The treatment necessarily varies in the several forms of the affection, but it is in the main causal. The beneficial effect of the recumbent posture in affording immediate relief to the symptoms, and obviating permanent damage to the cerebral nutrition, must be always remembered. In acute anæmia from loss of blood, the head should be kept continuously low, stimulants freely administered, and as a penultimate resort bandages, applied to the limbs from below upward, may increase the proportionate supply of blood to the brain. If this fails transfusion must be had recourse to. In chronic anæmia, sudden change of posture should be carefully avoided, and ferruginous tonics are needed. The form of iron, I believe, matters much less than is usually supposed. When the corpuscles are few, arsenic should be given with it. In severe cases, absolute physical rest is often advantageous, but this depends on the form of the anæmia. When the hæmoglobin is reduced out of proportion to the corpuscles, physical rest is of the greatest importance, greater even than fresh air. Corpuscles are oxygen carriers in proportion to the hæmoglobin they contain. All muscular exertion uses up the oxygen, and the protoplasmic tissues suffer in their nutrition, and are incapacitated for the essential functions of digestion, circulation, etc. The beneficial effect of absolute rest in bed in these cases is often most striking. If, on the other hand, the corpuscles are rich in hæmoglobin (and in pernicious anæmia they may contain fifty per cent. more hæmoglobin than normal) gentle exercise may be permitted, because the oxygen carriers have their full functional capacity, and the nutrition of the tissues does not suffer; if some of the oxygen is used in exercise, the supply to the tissues is practically diminished thereby.

In the cerebral anæmia of syncope, the recumbent posture, stimulants to the skin, cold water, faradization, sinapisms and ammonia to the nasal mucous membrane, assist the recovery of cardiac action and the return of consciousness. In all cases, carefully regulated food and stimulants are needed; beef tea should be given in small quantities and at frequent intervals.

The group of symptoms called hydrocephaloid requires similar treatment. Its diagnosis is of the first importance, because a routine treatment for meningitis would kill the patient. Warmth to the surface of the body and head is of importance. Attempts prematurely to rouse the child to consciousness are unadvisable, but when a distinct improvement has occurred in the general state, and especially in the pulse, some mental activity probably favors the return of a normal state of the cerebral circulation.

In the more pronounced mental symptoms that result from anæmia, opiates are of great service, either opium by the mouth or by morphia under the skin. In states of depression the dose should be small and frequent, $\frac{1}{24}$ gr. of morphia three or four times a day. In excitement a larger dose ($\frac{1}{8}$ gr.) may be given for its soporific effect.

HYPERÆMIA OF THE BRAIN.

Of all regions of cerebral pathology, that of congestion of the brain is perhaps the most obscure. We have very little precise knowledge regarding it, and, as is often the case, theory has flourished in proportion to the deficiency of fact. It was long thought that the state of the vessels of the brain after death corresponds with their condition during life, and post-mortem distention was accepted as proof that any preceding cerebral symptoms were due to congestion. The fact was unobserved or ignored that a similar condition of the brain is equally common when there are no cerebral symptoms during life, and depends chiefly on the mode of death. Hence, an extensive symptomatology was elaborated and built up on an erroneous foundation, and it has to some extent survived its evidence. Moreover, congestion of organs seems to afford so satisfactory an explanation of derangement of their functions, that the temptation to assign the condition as the cause of the symptoms has proved irresistible to unscrupulous practitioners. In this way, also, a symptomatology has grown up, and even statistics have been amassed, the value of which may be estimated from the fact that in one modern text-book the history of cerebral congestion has been manifestly written from cases of pure hypochondriasis. On the other hand, partly by a reaction from this extreme, some have doubted even the possibility of the condition. The truth lies between the two extremes, but its precise position will be long undetermined. Opportunities of ascertaining the exact pathological condition in these cases are very rare, and even if no visible lesion is found, it is not always certain that the symptoms observed during life were the result of congestion. Hence, there is room for wide difference of opinion, even among those who strive to keep their minds unbiased. It is certain, however, that the cases in which symptoms of definite character and considerable degree can be reasonably ascribed to this cause are far from frequent.

The essential pathological condition of hyperæmia of the brain is an increase in the amount of blood within its capillaries. This may occur because there is too much blood in the arteries, or too much in the veins; in the former case more blood enters the capillaries, in the latter too little leaves them; in either case they are over-distended. The two conditions differ in their causes and symptoms; the one is active, the other passive congestion. Neither arterial nor venous distention can exist without a corresponding state of the capillaries; to the capillary hyperæmia we must ascribe the disturbance of function, and hence we are justified in regarding it as the essential element in both forms. The difference between the two arises from the character of the blood circulating in the capillaries. In the one case the supply of arterial blood to these vessels is excessive; in the other it is deficient, because the circulation in them is hindered. Hence the state of passive congestion, in which the brain is under-supplied with arterial blood, approaches that of anæmia; in both anæmia and passive congestion there is anoxæmia, but in passive congestion the capillaries contain blood that has also an undue amount of carbonic acid.

It was at one time thought that the blood in the brain could not vary in amount because the cranio-vertebral cavity is a closed space, and this opinion is still occasionally put forward.* But the mobility of the cerebro-spinal fluid (which occupies not only the inter-membranous space and the ventricles, but also the lymphatic spaces around the vessels) permits the vascular distention to vary. If the cavity were hermetically closed, the variation could be only relative, not absolute. But the numerous foramina of the cranium and vertebral canal are occupied by less resistant structures, which no doubt may yield in some degree. Moreover, the large surface veins of the spinal cord, and still more the enormous plexus outside the spinal dura mater, doubtless constitute an important means of adaptation. Further, the processes of secretion and absorption of the cerebro-spinal fluid, always in constant operation, must be influenced by the degree of pressure, and may quickly vary with it. Although the conditions during life and after death are widely different, yet we may reasonably regard the enormous variations in the total amount of blood within the cranio-vertebral canal after death in different cases, as evidence that considerable variations may occur during life. Some variation is physiological. In the child, before the fontanelles are closed, and in the adult, when a piece of the skull is removed by injury, it is seen that the brain pulsates synchronously with the heart, and that variations also result from the respiratory movements of the thorax. Tracings of these movements have been obtained.†

Etiology.—The causes that produce active and passive congestion are widely different, and require separate consideration. Some act on the brain alone, others on many viscera or even on the whole vascular system; some are transient, others are permanent.

Active Congestion.—(1) *Over-action of the heart* is one important cause. Whatever increases the force of the heart and at the same time interposes no obstacle to the flow of blood into the capillary system, is a cause of active distention of the cerebral vessels. Excited action is more frequently effective than hypertrophy, because the common cause of hypertrophy is an obstruction between the heart and the capillaries. It may occur, however, from the hypertrophy that is due to aortic regurgitation, when obstruction is slight or absent. "Idiopathic" or "primary hypertrophy" without a mechanical cause, is a pathological fiction. (2) A sudden contraction of the arterioles elsewhere, as, for instance, in the skin, from exposure to cold or during a rigor, may cause transient overfilling of the cerebral vessels, together with those of other viscera. (3) Dilatation of the arterioles, and consequently an increase of the blood supply, may be produced by certain toxic agents, especially by nitrite of amyl, nitro-glycerine, and alcohol. The throbbing and headache produced by nitrite of amyl, seems in itself to prove the possibility of active congestion of the brain. Morphia causes first contraction and then dilatation. Some of these agents also quicken the action of the heart, and may thus further increase the blood supply. Emotion may have the same influence, although the evidence of this is inconclusive.* In some diseases a similar mechanism gives rise to congestion. In exophthalmic goitre, for instance, vascular dilatation is associated with cardiac over-action. In other peculiar states of the nervous system, especially in young neurotic persons, singular conditions occur, sometimes periodically, in which there are symptoms of vasomotor paralysis of the cerebral vessels, without other cause than the peculiar neuropathic disposition, and the tendency to such disturbance is sometimes distinctly inherited. (4) Active congestion occurs also as the first stage of inflammation. Acute symptoms are occasionally met with in children, and less frequently in adults, resembling those of the early stage of meningitis or acute cerebritis, and may quickly cause death. Traces of congestion are found, but no evidence of actual inflammation. The immediate cause of the condition is often obscure; excessive brain work,* or exposure to cold, has been supposed to produce it in some cases. There is no justification, however, for referring all sudden cerebral symptoms in children to congestion. Convulsions, for instance, were once thought to be always due to this, but there is reason to believe that they are very rarely thus produced. (5) Insolation is probably a cause of active congestion, which may or may not go on to inflammation. The modern theory, which regards the so-called sunstroke as really heat-stroke, the result of the overheating of the body and not of the action of the sun on the head, is probably too sweeping. The delirium of acute febrile diseases was formerly attributed to congestion of the brain, but it is more probable that the cerebral disturbance is the result of the action of the altered blood on the cells of the brain, and that any congestion is of secondary origin. (6) Active congestion is supposed to be a cause of transient general cerebral symptoms in adults, especially after middle life and in the male sex. It is believed to be frequent in those of a certain build, stout men with thick necks and florid face, and to be favored by alcoholic excess, and by the condition termed "plethora," which, perhaps, consists in an increase in the total quantity of blood in the system. The symptoms come on suddenly, and pass away in a manner that renders it difficult to ascribe them to an organic lesion, and in rare cases the absence of such lesion has been demonstrated. They are said sometimes to follow the suppression of an habitual discharge, especially hemorrhage, as from piles or the catamenia in women.

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* See Moxon's Lectures, *Lancet*, i, 1881.

† In an operation that I recently witnessed (removal of a tumor from the spinal cord), before the dura mater was opened its distention with every movement of respiration was most conspicuous.

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* The pulsation of the brain, observed in persons who have lost a piece of the cranial bone, is said to be increased by both intellectual work and emotion (Mosso, "Reale. Acad. dei Lincei," Roma, 1880).

Partial active congestion occurs in connection with tumors and other organic lesions of the brain, and also when an artery is obstructed, and the adjacent branches of the main vessel receive too much blood. It is possible that partial congestion occurs as a primary condition, especially in certain parts of the brain, in which limited inflammation is occasionally met with; but such inflammation (as of the gray matter of the ocular nerves) is rare, and congestion must be still more rare. Acute congestion of the medulla oblongata has been supposed to be an occasional cause of sudden death in young children, but if this agency is really at work in these cases, its causes are altogether unknown.

Passive congestion is always produced mechanically by some obstruction to the return of blood. It occurs in such heart disease as causes overfilling of the venous system, although the head suffers less than parts that are below the level of the heart. It is produced also by pressure on the superior vena cava, or on the innominate veins, or on the veins of the neck, by tumors, etc. The obstruction to the flow of blood through the lungs occasioned by the act of coughing, playing wind instruments, and by other severe muscular efforts with closed glottis, is a frequent cause. Slight congestion is often produced by wearing tight collars. In death by suffocation it occurs in very intense degree. The horizontal posture probably suffices to produce some degree of congestion, the venous blood losing the aid that gravitation gives to its return, and recumbency may intensify the influence of other causes. Partial passive congestion also results from pressure on, or thrombosis in, one of the cerebral veins or sinuses.

Pathological Anatomy.—In the strict sense of the word, there is scarcely any pathological anatomy of congestion of the brain. Simple active congestion disappears after death in every organ. No trace can be seen, for instance, of the congested areola around a pustule in the skin. Only the vascular distention of actual inflammation persists, lessened in degree. The lesson of general pathology is, therefore, that whenever we find after death distention of the minute vessels, without engorgement of the veins, the condition must be regarded as one of commencing inflammation, and not of mere congestion. Nor is the case otherwise with passive congestion. Intense passive congestion occurs during death from suffocation, and yet the brain may be found anæmic after death (Ackermann, Jolly). But intense venous distention occurs after death in those parts toward which gravitation draws the blood, and hence in the posterior half of the head, when this is not raised after death. The influence of gravitation may keep the blood in the vessels of congested parts, but it is never possible to distinguish the influence of the ante-mortem distention in the presence of the more powerful post-mortem effect. If mechanical congestion has occurred, not merely during the act of death, but for many hours or days previously, and gravitation keeps up the engorgement after death, the distention of the veins and capillaries is very intense; the color of the gray substance is dark, from the more abundant capillaries in it, and even the arteries may contain a good deal of blood. But if the body is so placed after death that the blood can gravitate from the head into the relaxed vessels elsewhere, no amount of mechanical congestion during life can be traced twelve hours after death. It is said that after long-continued mechanical congestion the veins are unduly tortuous, but it is doubtful whether sufficient allowance has been made for the degree of tortuosity that may be present under normal conditions, and for the influence of senile changes in the wall of the vessels. A slight degree of effusion into the ventricles probably results from passive congestion, but it is doubtful whether this is ever sufficient to cause flattening of the convolutions, and still more doubtful is the occurrence of such swelling of the whole brain as to cause flattening, or of any general "hypertrophy" or atrophy which have been said to occur. It is probable, however, that the spaces around the vessels, which arise by a dilatation of the perivascular sheaths, are increased by mechanical congestion, although here again, apart from congestion, the variations met with are so great, and the size of these spaces is often so considerable, that the influence of congestion upon them cannot be regarded as proved. Even in young persons their size is sometimes considerable. Microscopical examination shows the capillaries to be unduly large in passive congestion, and it is probable that systematic measurement would show that, after long-continued congestion during life, their average size is larger than in simple post-mortem engorgement. Bulgings of the capillaries have been occasionally seen. After asphyxial modes of death it is common to find that minute vessels here and there have given way, so that the lymphatic sheath is filled with blood. Blood pigment in the sheaths has been found in cases of slighter long-continued congestion (Bastian). Lastly, minute microscopic hemorrhages into the cerebral substance may be found almost constantly in these cases.

While the pathological anatomy of congestion is thus to a considerable extent negative, it is important to point out that this affords no reason for doubting the occurrence of the condition, since in other parts conspicuous congestion during life may leave no trace.

SYMPTOMS.—The introductory remarks show how great is the difficulty of determining the symptoms of cerebral congestion, and how much caution is desirable in accepting the statements that have been made, founded for the most part on an elaborate symptomatology that was formulated by Andral.

Passive Congestion.—The symptoms produced by passive congestion are much more marked when this is occasional, than when it is constant, uniform, and gradually produced. This is only one instance of the remarkable tolerance of the brain to pressure if slowly developed. A slight degree of mechanical congestion causes dull headache, chiefly frontal, sometimes throbbing, and sometimes accompanied by vague discomfort, described by some patients as a "confused feeling," by others as a "sense of fullness."

This congestive headache is often produced by coughing. I have known a patient suffering from phthisis to seek relief, not on account of the cough, but on account of headache which was solely due to the cough. When intense and sudden, flashes of light may be observed; noises in the ears are much less common. Slight general convulsion may even result, usually consisting only of clonic spasm, and then there may or may not be loss of consciousness. An elderly man, with chronic bronchitis and emphysema, had severe paroxysms of coughing, and the acme of each, when his face was dusky, was attended with a brief attack of general clonic spasm, without any loss of consciousness. Occasionally the convulsion is epileptoid in its character. Sometimes there is loss of consciousness without convulsion. In more persistent mechanical congestion there may be somnolence, besides headache, and the vague cerebral discomfort already described. Sleep may be disturbed by dreams or starts, or by nocturnal delirium. Sleeplessness is rare. Vague giddiness is often complained of, but there is seldom distinct vertigo. The mind is inactive. Tingling in the limbs, chiefly in the extremities, has been ascribed to passive congestion. There may be some general lack of muscular power, but it is doubtful whether unilateral symptoms ever occur.

With these symptoms are commonly associated the signs of general cephalic hyperæmia, a turgid face, and congested conjunctiva, often with viscid secretion. The symptoms are increased by constipation, by stooping, by the recumbent posture with the head low, by tight clothes about the neck, by flexion of the neck, and by all muscular effort.

Active Congestion.—The symptoms of active congestion are much more variable and much less certain. Simple excitement of the heart's action, such as results from emotions, or from nitrite of amyl, will cause throbbing and pain in the head, but in persistent over-action of the heart, as in exophthalmic goitre or aortic regurgitation with hypertrophy, cerebral symptoms are slight or absent.

The cases in which definite and considerable disturbance can be referred, with most probability, to active congestion, are those in which there are recurring paroxysms of headache, delirium, and sometimes fever, preceded by throbbing of the vessels, and sometimes by reddening of the face—symptoms of transient character and uniform recurrence. Thus in a case related by Nothnagel,* a man, aged fifty-seven, had suffered from such attacks at intervals of a few weeks or months since the age of fourteen. At first there was only a sense of fullness, heat, and pain in the head, always relieved by bleeding at the nose. Afterward each attack began with beating of the heart, heat of head, spots before the eyes, and noises in the ears; these symptons were followed by mental excitement and irritability, screams and tears, but no actual delusions. This condition would last a few hours or a few days, but was always at once cut short by venesection. In the intervals he was perfectly well.

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* *Ziemssen's Cyclopædia*, Bd. xi, 1, 2 Aufl., p. 49.

Brief attacks of headache with delirium may be attended with fever and special heat of head, and have been regarded as a febrile form of cerebral congestion. Whatever difficulty there may be in accepting this explanation of such cases, it is at present very difficult to find any other, especially when the attacks are brief and recur. Well-marked instances are occasionally seen in children. Thus a clever, precocious girl of six had been liable, since the age of two, to attacks of the following character, recurring at intervals of three to nine months. There was severe left-sided headache, prostration, and elevation of temperature, reaching sometimes 103°, great heat of head, occasionally delirium, and often nausea, but no vomiting. Each attack commenced suddenly, lasted a few hours, and then the child went to sleep and woke up perfectly well. There is some reason to think that such attacks in childhood may pass into non-febrile migraine at a later period. Whether they are to be ascribed to a primary vasomotor disturbance or to a "nerve storm," the opinion that an active cerebral congestion takes at least a secondary part in the morbid process may reasonably be maintained and cannot be disproved. In some cases of migraine there is evidence of active hyperæmia of the brain during part of the attack, but it is probable that this is of secondary origin. The brief attacks of coma, pyrexia, heat of head, and other cerebral symptoms, which occur during the course of general paralysis of the insane, are commonly regarded as of congestive nature.

Acute symptoms of brain disturbance in children, resembling those of meningitis—headache, delirium or somnolence, vomiting, strabismus, inequality of pupil, various contractions and paralyses, and a prominent fontanelle—are sometimes rapidly fatal without any post-mortem evidence of active inflammation. If congestion of the brain is the cause of these symptoms it is probably that which constitutes the first stage of inflammation.

The most common severe attacks that are ascribed to cerebral congestion are those which occur in the second half of life, and are attended with sudden loss of consciousness (the so-called "congestive apoplexy"), and sometimes by transient hemiplegia, passing away in the course of a few days. The congestive nature of many of these cases is more than doubtful. We know that an actual lesion may occur in many parts of the brain, a small hemorrhage, or a spot of softening, and may cause only general cerebral symptoms, or local symptoms of indirect origin and very brief duration. We know, too, that vascular obstruction may occur and cause no lasting symptoms, because there may be sufficient anastomoses to permit of an adequate collateral circulation. It is in these cases that the facility and reassuring character of the diagnosis of "congestion of the brain" often tempts the physician to an opinion that is, if not erroneous, at least unwarranted. But when the attack is brief, passes away completely, is attended with the signs of what is termed "plethora," and, especially when it recurs more than once, still without enduring symptoms, the diagnosis of cerebral congestion is at least permissible, and it is certain that, in many cases of the kind, no lesion has been found after death to account for the symptoms. It is commonly held, and must be admitted as possible, that cerebral congestion may cause these symptoms, when there are no signs of plethora or of hyperæmia of the external vessels of the head, and even no pallor of the face, but still greater caution should be exercised in making the diagnosis in such patients. As an instance of the condition in which the diagnosis seemed justifiable, I may mention the case of a stout, full-blooded man, about fifty-five, with a large, strongly-acting heart and some arterial degeneration, who, during the preceding year, had had several attacks of left-sided weakness, each lasting only a few hours; the first was attended with loss of consciousness. The day before I saw him, he complained of tingling in both hands, and a few hours later vomited, went to bed, and passed a stool into the bed; then he got up and got into bed again. His temperature was found to be 102°; he did what he was told, and answered questions, but with imperfect articulation. During the next twenty-four hours he did not speak, but was restless, tossing about in bed, sweating profusely, with a fall of temperature to 100.5°. His pulse was 80, full, but not hard, face flushed, pupils rather small. He understood imperfectly what was said to him. There was no evidence of loss of power or sensation. The symptoms all passed away in the course of a few days without the slightest indication of a local cerebral lesion.

There is much difficulty in reconciling hemiplegic symptoms with the idea of congestion as their cause. In the vasomotor inertia of the old, the occurrence of active congestion of any part is not easy to understand (although not, therefore, to be denied), but it is even less easy to understand that one hemisphere of the brain alone should suffer. It is possible, however, that an explanation may be found in partial arterial degeneration, which may determine the greater or less disturbance of one part of the brain.

A "convulsive form" of cerebral congestion has been described, in which severe epileptiform convulsions are the only symptom. But the relation of these to cerebral congestion is very doubtful. Formerly, as already mentioned, almost all convulsions in young children were ascribed to cerebral congestion, but it is certain that simple convulsions from this cause are, to say the least, extremely rare.

It is doubtful whether any form of cerebral congestion is attended by recognizable changes in the circulation within the eye.

DIAGNOSIS.—The most important principles of diagnosis have been already incidentally mentioned. The chief points, disregard of which causes the grossest errors of diagnosis, are two: (1) Persistent focal symptoms, such as hemiplegic weakness, however slight, exclude mere congestion. (2) Cephalic sensations of vague or definite character are alone of no diagnostic value. Such sensations are especially common in hypochondriacal patients, who often suffer much from various feelings of fullness, tightness, heat, burning, and especially from a sensation of pressure on some part of the head, generally the vertex. These sensations are intensified by annoyance and by brain work, and are vastly increased by attention. They are purely nervous sensations, pseudo-neuralgic in nature, and there is not the slightest justification for attributing them to congestion of the brain. And yet such patients, if they consult many doctors, as they usually do, are sure to be told by some that their symptoms are due to congestion of the brain, or even (with a precision that is evidence only of profound ignorance or of actual charlatanry) to "congestion of the base of the brain," a condition that probably never exists but in pathological imagination.

PROGNOSIS.—The danger to life in cerebral congestion is indicated only by the intensity of the symptoms. When these are very intense, and actual coma is present, there may be danger, but in most cases the symptoms pass away completely. They are prone to recur. The future prognosis is hence uncertain, and depends chiefly on the extent to which the cause can be discovered and removed.

TREATMENT.—In *passive congestion* the only effective treatment is the removal of the causes of the mechanical obstruction. These are, unfortunately, for the most part beyond our control. Cough, however, can be moderated; the heart, if necessary, strengthened by digitalis; and in all cases many intensifying influences can be lessened. Effort should be avoided; the clothes should be loose about the neck; the patient should sleep with head well raised and the shoulders also raised, so as to avoid any flexion of the neck.

In *active congestion* a similar attention to posture, etc., is desirable, so as to obviate any hindrance to the escape of the blood from the over-filled capillaries. In other respects the treatment must be varied, according to the individual condition. Its immediate object is to lessen the amount of blood in the cerebral arteries, and there are three modes in which this may be attempted: (1) by lessening the total volume of the blood; (2) by obtaining the dilatation of vessels elsewhere, and so drawing the blood from the head; (3) by causing the dilated cerebral arteries to contract. Some of the measures employed act in more than one way.

The most direct mode of lessening the volume of the blood, venesection, is applicable only to the plethoric form of congestion, in which the face is red and turgid, and the pulse is full. The relief that it affords is immediate and great. If the propriety of venesection is doubtful, leeches may be applied, and in children this method alone is permissible. The leeches should be applied over the mastoid process, because there is a communication between the cutaneous and intracranial vessels through the mastoid veins. Thus we may follow the indication that is given by nose bleeding, which often gives striking relief to cerebral congestion and has a more direct influence on the cerebral circulation than any other hemorrhage. The induction of nose bleeding is impracticable on account of the difficulty of controlling the flow of blood.

Purgation and diuresis both act by diminishing the volume of the blood, and the former at least acts also by increasing the amount of blood in the capacious intestinal vessels, while both alter the constitution of the blood by removing from it effete products. The degree of purgation must depend on the strength of the patient, but in all cases it is important that the bowels should be opened, freely or gently. Constipation causes in some way cephalic discomfort, headache, etc., and intensifies the symptoms of cerebral congestion. A saline purge is the most effective, with or without a preceding mercurial. The saline should not be too much diluted, and thus a maximum osmotic action may be obtained. Both the liquid evacuations, and the thirst which follows the action of the purgative, afford proof of the removal of liquid from the blood. Diuretics are also distinctly useful. A full dose of nitric ether and spirit of juniper may be given every three or four hours. Diaphoretics are of doubtful value, except when the symptoms follow exposure to cold. Except pilocarpine, which has not yet been tried in this affection, a hot-air bath is the only effectual diaphoretic, and this, by raising the temperature of the whole body, may have a prejudicial action. In all morbid states due to cold, early diaphoresis is most useful, but it is doubtful whether cerebral congestion occurs from this cause.

Blood may be drawn to the limbs by immersing them in warm water and keeping them dependent. Even in health, faintness may be produced by the long immersion of the feet in hot water. "Junod's boot," in which the air is partially exhausted around a limb, was formerly employed with this object, but has fallen into almost entire disuse. Gentle constriction of the limbs, so as to compress the surface veins, but not the deeper arteries, has a similar effect.

The third object of treatment is to obtain contraction of the arteries of the brain. It is unfortunately never possible to ascertain to what degree this object is attained, and we can only adopt those measures that seem most likely to secure it. One of these is the application of cold to the head. This should be continuous, by ice or an irrigation-tube cap. A mustard plaster to the neck is also probably of service. The application of mustard plasters to the limbs is of doubtful value; it is possible that, as some think, they may cause a reflex contraction of the cerebral arteries, but their supposed "derivative" influence in drawing blood to the surface must be infinitesimal unless the application is made over a very large area. It is uncertain, also, how far pathological dilatation of the vessels of one part can be influenced by drugs. Ergot, for instance, probably would have less influence on the vessels that are in a morbid state than on those elsewhere, and it might thus increase the congestion. Digitalis may be beneficial, but rather on account of its influence on the heart than on the vessels. It is most important to maintain the circulation as steady and uniform as possible. For this end perfect tranquillity of mind and body should be secured. Alcohol and morphia should be avoided, and insomnia or restlessness treated with bromide.

CEREBRAL HEMORRHAGE.

Hemorrhage may occur into the substance or cavities or membranes of the brain. The term "cerebral hemorrhage" is sometimes used as a general designation for all intracranial extravasation; sometimes it is applied only to hemorrhage into the substance of the brain (including the cerebellum, pons and medulla), and is distinguished from "meningeal hemorrhage," in which the extravasation is into the membranes of the brain.

Etiology.—Hemorrhage is always due to the rupture of a vessel. The rupture may be the result of injury, or may occur "spontaneously," *i.e.*, as a result solely of internal causes. The vessel that bursts is usually an artery, very rarely a vein. Capillaries may also rupture, but only a minute extravasation results.

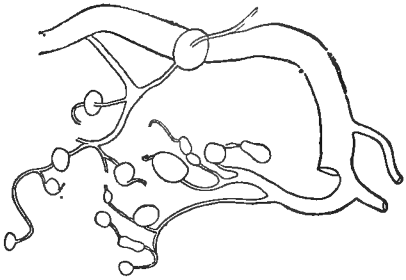
The causation of cerebral hemorrhage includes three subjects: (1) the immediate pathological conditions that lead to the rupture; (2) the influences that bring about this pathological condition; and (3) those that actually burst the vessel. These may be distinguished as the pathological, remote, and exciting causes.

Pathological Causes.—Putting aside traumatic influences, the force that ruptures an artery is the pressure of the blood within it. But as long as the walls of an artery are in a healthy state, they very rarely give way, however great may be the pressure to which they are exposed. Healthy veins may give way under extreme pressure, as in death by suffocation, but arteries do so seldom, perhaps never. When an artery ruptures, it is because the walls are so changed as to be weakened. The weakened wall may give way without any abnormal degree of pressure, but if the pressure is increased the weakened wall gives way the more readily. The two factors, weakening of wall and increase of blood pressure, often coincide, but without doubt the state of the vessel is incomparably the more important of the two, rarely if ever absent, and an effective cause of rupture when the blood pressure is normal.

When the wall of an artery is weakened, it yields before the blood pressure and becomes bulged. By the extension it is thinned, and thus weakened, until it gives way. The bulging constitutes an aneurism. The large arteries at the base or on the surface of the brain are occasionally thus dilated and thus burst. These aneurisms and their consequences are considered in detail at another page. They are, on the whole, rare. The vessels that give way in the common form of cerebral hemorrhage are of much smaller size, and are in the substance of the brain. In these arteries, as in those of larger size, bulging precedes bursting, and the rupture is of the wall of a minute aneurism. Such "miliary aneurisms," as they are termed, are always to be found in cases of cerebral hemorrhage. Their constancy was proved by Charcot and Bouchard, who found them in each of seventy-seven consecutive cases. They are to be discovered by washing away the cerebral substance under water, and carefully picking out the tiny, thread-like vessels that remain. On these little red grains are seen, which a low magnifying power shows to be minute aneurisms (Fig. 284). Their size varies from

$\frac{1}{100}$ to $\frac{1}{25}$ of an inch. As many as a hundred have been found in a single brain. They are met with almost exclusively in the second half of life, and, although once found in a patient only twenty years old, they are as rare under forty as is cerebral hemorrhage. Their frequency after forty increases with age. The order of frequency with which they are found in different parts of the brain is, according to Charcot and Bouchard, central ganglia, cortex, pons, cerebellum, centrum ovale, middle cerebellar peduncle, crus cerebri, medulla oblongata—an order which is not far from that of hemorrhage. In the aneurisms, the muscular coat of the vessel has disappeared, the adventitia and intima are united. The change is said to commence by the production of new tissue elements in the outer coat (Charcot and Bouchard) or in the inner coat (Zenker), and is called an "arteritis" (periarteritis or endarteritis), but the evidence of its inflammatory nature seems insufficient to justify the term. No doubt the effective element in the change is the loss of the contractile and elastic elements in the coat; in consequence of this it yields more and more to the pressure of the blood, any new tissue elements that are formed being extensible and inelastic.

FIG. 284.



Outline of miliary aneurisms from the brain in a case of hemorrhage; magnified. (*After Eichhorst.*)

The common change in the large vessels termed "atheroma" has only an indirect connection with hemorrhage. Atheroma does not affect the small vessels within the brain, and although it renders the wall of the vessel inelastic, it also renders it thicker and less distensible than normal. Both the miliary aneurisms and atheroma are senile changes, and often coexist, but each may be found without the other;* atheroma often exists in high degree without hemorrhage, and the latter may occur when the vessels of the base are perfectly healthy. Nothnagel has suggested that atheroma of the larger vessels may facilitate the production of miliary aneurisms by increasing the force and suddenness of the pulse-wave that reaches the smaller arteries, the larger vessels no longer yielding to the pressure.

Miliary aneurisms are far more frequent in the brain than elsewhere, first, because the external support of the cerebral arteries is slight, in consequence of the wasting of the brain tissue around them; secondly, because the strain to which the vessels are exposed is greater than in other arteries of the same size, in consequence of their origin directly from large trunks; the pressure elsewhere being more gradually lessened by more gradual branching. Both these conditions will facilitate not only the production of aneurisms, but also their rupture.

Remote Causes.—Hereditary predisposition is sometimes very marked, and doubtless consists in a tendency to the occurrence of the vascular change. The influence of heredity is shown not only by the frequency of the occurrence of cerebral hemorrhage in some families, but by the entire freedom of other families through many long-lived generations.

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* Charcot and Bouchard found atheroma absent in one-quarter of the cases of miliary aneurisms.

Sex.—Males suffer more frequently than females, although the difference is less than is commonly asserted. Gintrac's figures of 681 cases* (excluding meningeal hemorrhage) yield a percentage of males 56.6, females 43.4.

Age.—Cerebral hemorrhage is essentially a disease of the degenerative period of life; at least four-fifths of the cases occur after forty years of age, and its frequency increases as life advances. But it is met with occasionally in earlier life, even in childhood and infancy.† The most extensive statistics available on this point are those of Gintrac,‡ although these, consisting of published cases, are less satisfactory than a consecutive series would be, and it is probable that the frequency of the disease in early life is over-represented. The first column of the following table gives Gintrac's figures of the distribution of 653 cases he has collected, and in the second I have reduced these to a percentage. These two columns thus show the relative frequency in each decade of life. But since the number of persons living diminishes in each successive decade, such figures, as Sir George Burrows long ago insisted, convey an erroneous idea of the liability to the disease. The third column of the table is therefore computed by the help of Farr's Life Table, No. 3, and shows the number of cases of cerebral hemorrhage in an equal number of individuals in each decade of life, called 1000 x. It thus shows the relative *liability* in each decade. Of the real value of x we have no means of judging. In the last column I have reduced the series to a percentage, so that the relative liability to the disease at each age may be more conveniently compared with its relative frequency. Thus it is seen that while the disease is actually less frequent between seventy and eighty than between sixty and seventy, the liability to it is greater in the latter than in the earlier decade. From the second column it appears as if the disease were only one-tenth as frequent between eighty and ninety as between fifty and sixty, but the third and fourth columns show that the liability to it is the same in these two decades. It may perhaps be questioned whether the number of cases between eighty and ninety is sufficient to justify the conclusion to which they point, that the liability is much less between eighty and ninety than between seventy and eighty, *i. e.* that in the very old cerebral hemorrhage becomes less prevalent. But I think it probable that the conclusion is correct, and that the condition of tissues which permits the attainment of extreme old age is one in which there is less tendency to the occurrence of the arterial changes that lead to cerebral hemorrhage.

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* "Maladies de l'appareil nerveux," 1869, Tome ii, p. 430.

† Hemorrhages into the substance of the brain, minute and massive, have occurred in young children during the paroxysms of whooping cough. An instance is recorded by Marshall, *Glasgow Med. Jour.*, *1885*, p. 24. It is probable that the rupture is generally of a vein.

‡ Loc. cit., table on p. 431, with the exclusion of the cases of primary meningeal and spinal hemorrhage.

AGE.	Age-distribution of Cases.		Age-distribution of Liability.	
	No. of Cases in each decade.	Percentage of Cases in each decade.	Liability; number of cases in 1000 x persons living in each decade.*	Percentage of liability in each decade.
1—10	15	2.3	1.8	.8
11—20	20	3.1	3	1.3
21—30	38	6.0	6	2.6
31—40	71	10.8	12	5.2
41—50	97	15.0	19	8.3
51—60	129	19.8	31	13.5
61—70	159	24.4	52	22.4
71—80	112	17.2	72	32.4
81—90	22	1.8	32	14

A certain physical conformation is popularly associated with a liability to "apoplexy," a short, thick neck, high shoulders, florid face. It is doubtful whether this condition has any relation to cerebral hemorrhage; most of the subjects are thin and spare, and present almost the reverse of the characteristic "apoplectic build."

Cerebral hemorrhage is said to be more frequent in temperate than in tropical climates, in winter than in summer, and it is probably more common in civilized than in savage races.

Thus the degenerative tendency incidental to advanced life is, in the majority of cases, the most powerful element in the production of cerebral hemorrhage. But this influence is augmented by certain morbid states which increase the tendency to that impairment of nutrition which we call degeneration. The most important of these are chronic alcoholism, gout, and renal disease, especially the common senile form, granular disease of the kidney. Charcot found kidney disease in one-third of his cases of cerebral hemorrhage, and this is probably about the true proportion.* Whatever be the nature of fibroid disease of the kidney, whether the affection is primarily local or general, the minute vessels always suffer, and they suffer likewise, although to a less extent, in other forms of kidney disease, unquestionably primary. The strain on the vessels from the hypertrophy of the heart, whether it helps to produce the degeneration or not, must increase the tendency to dilation and rupture. It cannot be doubted that Bright's disease is a cause of miliary aneurisms in persons after middle life, and sometimes at earlier ages. I have elsewhere described and figured a case of Bright's disease in a woman aged thirty-six, in whose retina several miliary aneurisms could be seen with the ophthalmoscope, and who died a few days later with all the symptoms of a sudden cerebral hemorrhage.†

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* It may be well to explain further the manner in which the figures of the third column are obtained. From Farr's Life Table, No. 3, the population maintained in each decade by 10,000 annual births was first ascertained (the decades being, of course, reckoned according to the first column, 1–10, 11–20, etc.). The figures in the third column for each decade are the numbers that bear the same relation to 1000 as the number of cases in this decade (first column) bears to the number of persons living of that age in a population in which there are annually 10,000 births. The number 1000 is arbitrary. 1 might have been taken instead, but 1000 must be much nearer the actual number than 1 would be. The uncertain number to be added to 1000 is represented by x, which may be more or less than 1. The actual number (*i. e.*, the real value x) could not be known unless there were a post-mortem examination in every person dying in a large and known population. For the sake of simplicity, the actual population at each decade has not been given, but one instance may make the principle of the table clearer. In a population in which there are 10,000 annual births, the number of persons living between 71 and 80 will be (according to the Life Table) 1547. The number of the deaths from cerebral hemorrhage in this period (of the cases collected) is 112. Supposing that number of deaths from this cause to occur among 1547 persons living at that period, the number of cases in 1000 will be 72, and $x = 1$. But as we do not know the exact number of persons, it is termed 1000 x. In any case, the figures represent the relative liability throughout life, so far as the cases collected by Gintrac are representative. They are doubtless not exactly representative; as before stated, the early deaths are probably in undue proportion, because such cases are more likely to be published. But, with this exception, there seems no reason why they should not fairly represent the relation of the disease to age, and they have the advantage over Registrar's reports, that each case is unquestionably one of hemorrhage, since in every case collected there was a post-mortem examination.

It is well established that aneurisms of the larger arteries of the brain are frequently due to syphilitic disease and to embolic arteritis, and hemorrhage results from the rupture of such aneurisms. It is not yet known whether these processes also cause miliary aneurisms. I have once seen extensive hemorrhage into the brain of a boy, aged eight, with inherited syphilis and vascular disease, without any visible aneurism. The hemorrhage had apparently commenced in the right lenticular nucleus, or outside it, and had burst into the ventricles. The syphilitic disease was chiefly in the vertebral and cerebellar arteries. I do not know of any similar case in a subject of acquired syphilis, but it is certain that the small arteries within the cerebral substance are occasionally affected by syphilitic changes, and it is possible that some cases of intra-cerebral hemorrhage in early adult life may have this origin. Aneurism results from embolism, apparently, when a vessel is incompletely plugged, so that the altered wall is still exposed to the pressure of the blood, and plugging of the minute vessels within the cerebral substance must be generally complete.

Lastly, in certain general diseases there is a tendency to bleeding, which may cause intra-cerebral hemorrhage. Thus it may occur in purpura, scurvy, pernicious anæmia, and especially in leucocythæmia, in which it is a common cause of death, and the extravasations are often multiple. Its mechanism in these cases is not yet known. The most probable hypothesis ascribes it to acute degeneration in the walls of the vessels. In leucocythæmia it has been thought to be due to obstruction of the vessels by accumulations of white corpuscles, but the adequacy of this mechanism is not apparent. In these diseases we do not know whether the vessels rupture directly, or whether they first dilate into miliary aneurisms.

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* It has been attempted to depreciate the influence of renal disease in the production of cerebral hemorrhage, by demonstrating the rarity of this in a series of renal cases. But later adult life is essential for the occurrence of the vascular consequences, and nothing is proved by a series of cases taken at all ages.

† "Medical Ophthalmoscopy," 2d ed., plate xii, fig. 1, case 42, p. 326. In albuminuric retinitis, capillary ectases are very common (ibid., pl. xvi, fig. 10); but it is rare to meet with aneurisms on the visible arteries, even when miliary aneurisms are abundant in the brain.

Exciting Causes.—The actual rupture sometimes occurs during some temporary increase in the blood pressure from muscular effort, such as straining at stool, lifting a heavy weight, during coitus or vomiting, violent cough (as whooping-cough), or from excited action of the heart consequent on emotion. But often, also, the vessel gives way when the circulation is tranquil, indeed, during sleep. The cerebral arteries are supposed to be contracted during sleep, but it should be remembered that in the recumbent posture the influence of gravitation on the return of blood from the brain is less than in the upright posture, and the pressure in the arteries must be increased, not only by the increase in the resistance to the flow through the capillaries, but also by the fact that gravitation no longer opposes the influence of the heart on the arteries, as it does in the erect posture. Moreover, if the anæmia of the brain during sleep is due to the contraction of arterioles of smaller size than those which are the seat of miliary aneurisms, the pressure in these must be increased by such contraction.

In extreme mechanical congestion, such as attends all asphyxial modes of death, small extravasations are usually produced, apart from vascular disease. They do not commonly exceed a mustard seed in size, and still more minute hemorrhages may often be found with the microscope scattered through the cerebral substance, and especially numerous in the pons and medulla. Often the hemorrhage is only into the perivascular sheath, which becomes distended with blood.

Meningeal Hemorrhage.—Blood may be extravasated (1) outside the dura mater, separating it from the bone (extra-dural hemorrhage); (2) beneath the dura mater, into what was regarded as the sac of the arachnoid when it was thought that a parietal layer of the arachnoid lined the dura mater (subdural hemorrhage); and (3) beneath the arachnoid, between it and the pia mater (sub-arachnoid hemorrhage). The blood may come from the arteries or veins or sinuses of the dura mater, or from the vessels of the pia mater. The chief causes are as follows: (1) Injury that causes fracture of the skull or laceration of the pia mater. Extensive hemorrhage is usually from the meningeal arteries or sinuses. The blood may be outside or beneath the dura mater. (2) Aneurisms of the larger arteries of the base or surface. (3) Rupture of an intra-cerebral hemorrhage. In hemorrhage into the ventricles the blood often passes through the foramina at the extremity of the fourth ventricle and collects beneath the arachnoid at the base of the brain and about the cerebellum. Sometimes it forces its way out of the descending cornu of the lateral ventricle, through the transverse fissure. (4) Meningeal hemorrhage occurs, apart from visible aneurisms, under the same conditions (age, chronic kidney disease, etc.) as hemorrhage elsewhere in the brain. It is also met with in some chronic diseases with hemorrhagic tendency, as purpura, leucocythæmia, and the malarial cachexia. The mechanism of its production in these cases is not known, but is probably the same as that which causes intra-cerebral hemorrhage in the same conditions. (5) It occurs occasionally in the insane, especially in the subjects of general paralysis. (6) During birth it may result from the compression of the skull, especially in cases in which the head is born last. The blood comes from the vessels of the pia mater or veins of the dura mater or even from the superior longitudinal sinus. These cases are separately described. (7) Spontaneous hemorrhage from a meningeal vein has been observed, but is excessively rare.*

Sub-arachnoid hemorrhage is equally frequent in the two sexes, but the sub-dural form is three times as frequent in males as in females (Gintrac). Taking all forms together, meningeal hemorrhage is far more frequent both in youth and in the middle period of life than intra-cerebral hemorrhage. It is especially frequent during the first two years of life. About one-tenth of the patients do not exceed twenty years of age, and nearly half the cases occur in the first forty years of life.†

Primary ventricular hemorrhage is met with in rare cases; one or more of the ventricles being filled with blood without any, or with only secondary, lesions of their walls. The blood usually comes from the vessels of the choroid plexuses, or of the velum interpositum, rarely from a vein in the wall of the ventricle. Probably the hemorrhage is due in most cases to the rupture of miliary aneurisms, which have been found in the choroid plexus, and it is for the most part related to the same general conditions as intra-cerebral hemorrhage. But it occasionally results also from severe mechanical congestion, as in attempted hanging, or from convulsions. In rare cases it proceeds from a large aneurism that has perforated the ventricle, or from a vascular growth, or occurs in hemorrhagic diatheses, as purpura or leucocythemia. Like meningeal hemorrhage, it is relatively more frequent in early life than is the ordinary form, occurring even in childhood and infancy, both during birth and in the early months of life. Of ninety-four cases collected by Sanders,‡ one-tenth occurred in the first year of life and one-fifth were under twenty, about a quarter under thirty, and one-third under forty. It is rather more frequent in males than in females, at all ages

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* Thus a case is recorded by Andral, in which the veins of the pia mater over the convexity were varicose and their walls soft and friable; one of them had ruptured, and a layer of coagulated blood lay over the hemisphere ("Clinique Médicale," Spillman's trans., p. 95).

† Gintrac's collection of 165 cases (of which only twelve were traumatic) exhibits the following distribution:—

Age.	Cases.	Percentage.
0–10	10	6
11–20	9	5.5
21–30	19	11.5
31–40	37	22.4
41–50	21	12.3
51–60	19	11.5
61–70	26	15.7
71–80	22	13.5
81–90	2	1.2

‡ In a careful study of this form, published in the *American Journal of Med. Science*, vol. lxxxii, 1881, pp. 85, 337.

except 30–40, when females preponderate, in consequence of the influence of child-bearing. In both the pregnant and puerperal states it sometimes occurs, produced usually by the mechanism of convulsions. It sometimes results from injury.

Traumatic hemorrhage, the result of blows and falls on the head, may occur at any age, and is independent of arterial degeneration. It is most common in the meninges, extra-dural as the result of rupture of a meningeal artery or a sinus, or sub-dural or sub-arachnoid in consequence of rupture of a vessel of the pia mater, usually as part of superficial laceration of the brain. Occasionally it occurs within the cerebral substance and is then often multiple. An extravasation in the pons may coexist with one or more extravasations into the hemispheres. Traumatic ventricular hemorrhage is rare, and is sometimes due to the rupture of a vein in the wall of the ventricles.

PATHOLOGICAL ANATOMY.—In the majority of cases there is only one recent hemorrhage in the brain; occasionally there are two or more, of which one is much larger than the others and has given rise to the symptoms. In some constitutional diseases with a tendency to hemorrhage, the brain may contain many extravasations. In leucocythæmia, for instance, more than fifty small hemorrhages have been found in the two hemispheres. The size of the hemorrhage varies from that of a nut to that of the closed fist. It may even have torn up the greater part of one hemisphere, may have distended all the ventricles, and have accumulated at the base. The two hemispheres are affected with equal frequency. Of the several parts of the brain, the central ganglia are the most frequent seat of hemorrhage; about half the total number of extravasations commence in the corpus striatum or its neighborhood. The clot often extends into the optic thalamus, but does not often begin in it. Next in frequency is hemorrhage into the centrum ovale, then successively the cortex, the pons, and the cerebellum. The frequency of hemorrhage in the cerebrum is twenty times greater than into the cerebellum. Hemorrhage into the medulla oblongata and crus cerebri are very rare, and extravasation into the corpus callosum has once been observed (in a case of cerebro-spinal meningitis*).

Within the cerebral substance the blood occupies a cavity formed by laceration of the brain tissue; rarely, when very minute and "capillary," by merely separating the fibres. The blood is clotted, and reddish-black in color; fragments of brain tissue are mingled with it. The containing cavity is often very irregular in shape; its walls are uneven, present projecting shreds of lacerated brain substance, and are blood-stained and softened—at first by imbibition of serum, and later by inflammation. Small extravasations are sometimes seen in the neighborhood of a larger clot. The extravasated blood exerts pressure; the convolutions are flattened; the falx is bulged to the opposite side, and the rest of the hemisphere is anæmic. The effused blood may tear its way into the lateral ventricle; it then speedily distends both lateral ventricles and the third and fourth ventricles, and escapes by the openings at the lower extremity of the fourth ventricle, central and lateral, into the sub-arachnoid space. Or the blood may tear its way to the surface, infiltrate the pia mater, and pass into the sub-arachnoid cavity, often by a very small opening. It is rarely that the artery from which the blood has escaped can be detected. Occasionally the extravasation can be traced to the rupture of an aneurism of some size. The miliary aneurisms, which can often be found, have been already described.

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* Erb, *Virchow's Archiv*, xcvii, 329.

After a time the extravasated blood undergoes changes. The clot shrinks, and gradually becomes, first chocolate, then brown, and ultimately a reddish-yellow; and it then contains chiefly fat globules, pigment and other granules, and hæmatoidin crystals. The rapidity with which it undergoes this change is uncertain, and certainly varies. It is said that the distinctive blood color has disappeared as early as the twentieth day; but it usually persists for a much longer time. Meanwhile the walls of the cavity undergo changes. The inflammation, in rare cases excessive and purulent, is usually conservative, and leads to the formation of connective tissue. A firm wall is thus developed, the inner surface of which becomes smooth by the softening and removal of the loose fragments of brain substance; by this means a cyst is formed. It is said that connective tissue may extend across its cavity, and that in rare cases, the fluid being absorbed, the cyst walls may unite, and a cicatrix result. Such cicatrices are, however, much more frequently due to softening than to hemorrhage. It is asserted that a cyst may be developed in thirty or forty days under favorable conditions.

Traumatic hemorrhage occurs into and from a lacerated portion of brain, and is most frequently found on the surface, occupying mainly the middle of the convex portion of each convolution. It is met with most frequently in regions much exposed to injury, as the surface of the temporo-sphenoidal lobe, and the under surface of the frontal lobe. Traumatic ventricular hemorrhage is said to result occasionally from rupture of a small vein on the surface of the corpus striatum (Prescott Hewett).

Soft tumors (especially gliomata) are sometimes the seat of hemorrhage. The distinctions from simple hemorrhage are the following. The position is often one in which the cerebral hemorrhage is rare; some gelatinous-looking tumor substance may be found, into which hemorrhage has not occurred, and which has characteristic microscopic features. Such tissue is never found in the neighborhood of an ordinary hemorrhage.

Other organs may be healthy, or present the changes which have been mentioned as predisposing causes, of which disease of the kidneys and hypertrophy of the heart are the most frequent. The lungs are usually loaded with serum and mucus. Occasionally intense visceral congestions, and even hemorrhages, are found in the kidneys, stomach and pleuræ. General congestion may be due to the mode of death, but the unilateral character and the intensity of the changes in some cases, render it probable that they are due to derangement of the vasomotor nerves.

The relation of hemorrhages in various situations to the vascular supply has been carefully traced by Duret.* Certain arteries give way more frequently than others, and the course of the vessels enables us to understand the position of the extravasation. The arrangement of the arteries is described at p. 495.

Corpus Striatum.—The hemorrhages into the corpus striatum may be divided into three series, anterior, middle, and posterior. The *anterior* are situated in the head of the caudate nucleus, and are due to the rupture of the branches that come from the anterior cerebral artery. They are usually small, but often break through into the lateral ventricle. The *middle* group comprehends those produced by the rupture of the lenticular and lenticulo-striate branches of the middle cerebral, and are the most frequent of all cerebral hemorrhages. These vessels may rupture anywhere in their course, outside the lenticular nucleus, within it, or in the caudate nucleus. The extravasations outside the nucleus are restrained externally by the gray cortex of the insula and its subjacent layer of white substance; if large, they may displace the central ganglia inward. Extravasations within the ganglia often extend upward and outward in the white matter of the centrum ovale, and may attain a very large size. The *posterior* hemorrhages are due to the rupture of the ventriculo-optic arteries of the middle cerebral, which pass through the lenticular nucleus into the anterior part of the optic thalamus. The extravasation usually commences in the thalamus, or between it and the corpus striatum, and the blood not rarely escapes between the two into the lateral ventricle. They often damage the posterior (sensory) part of the internal capsule. The small arteries to the inner portion of the thalamus, which pass from the posterior cerebral, or posterior communicating, sometimes give way and cause small extravasations near the surface (internal thalamic hemorrhages), which are very prone to rupture into the ventricle. The branches of the posterior cerebral to the hinder part of the thalamus may give rise to extravasations in this situation (posterior thalamic hemorrhage), which may either rupture into the lateral ventricle or may extend down into the crus and even into the pons.

Centrum Ovale.—Large hemorrhages usually spread into the centrum ovale from the corpus striatum. The vessels in the white substance itself are small, and give rise only to small hemorrhages, rarely larger than a walnut, and often oval, with the long axis in the direction of the fibres. Larger extravasations are sometimes found in the white substance of the occipital lobe. These are due to the rupture of branches of the calcarine division of the posterior cerebral artery.

Cortex.—Hemorrhages limited to the cortex may occur in almost any position, but they are rare and are usually small, although they sometimes extend into the white substance, and attain in it a larger size.

Crura Cerebri.—Hemorrhages into either the corpus striatum or optic thalamus may extend down into the crus. Those which commence in the crus are usually small, and oval in form. They may descend into the pons, but do not pass up into the central ganglia. They may be situated in the inner part of the crus, or in the outer part beneath the corpora geniculata, or in the upper part beneath the corpora quadrigemina.

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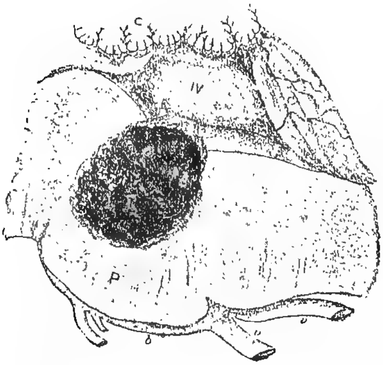
* *Arch. de Physiologie, 1874,* p. 664.

Pons.—Hemorrhages are most frequent near the middle line, from the rupture of the median branches of the basilar; and the raphé usually prevents their extension to the other side. The extravasation often has a spherical shape, and may be kept from the fourth ventricle only by a thin layer of tissue (Fig. 285), which may give way, and the blood may escape into the cavity. Sometimes small hemorrhages extend in a transverse direction, and these are due to the rupture of the small lateral branches of the median arteries. Hemorrhages in the lateral portions of the pons, from rupture of the radicular arteries, are rare. The vessel that most frequently gives way is the branch to the root of the fifth nerve.

FIG. 285.



Hemorrhage into the pons, to the left of the middle line. (*After Carswell*)

Cerebellum.—Large hemorrhages are most frequently due to the rupture of a branch that the superior cerebellar artery gives to the dentate nucleus. A small extravasation may occupy the interior of the dentate nucleus, arising from a branch of the same artery. Hemorrhage into the inner and hinder part of the hemisphere may occur from rupture of a branch of the posterior cerebral. Extravasations near the fourth ventricle readily burst into it. The cortex of the cerebellum offers much less resistance to hemorrhage than does the gray matter of the cerebral convolutions. The superior and inferior cerebellar peduncles are rarely the seat of hemorrhage, but an extravasation sometimes occurs in the middle cerebellar peduncle, and, more frequently, a hemorrhage in one-half of the pons may extend a short distance into the peduncle, passing in the direction of its fibres.

In *ventricular hemorrhage*, whether primary or secondary (*i. e.* by rupture of an extravasation in the substance of the brain), the blood fills all the ventricles in a third of the cases. If slowly effused it may occupy only one lateral ventricle, and such limitation is met with in about one-quarter of the cases of each form. It occupies both lateral ventricles alone (not extending to the third and fourth), more frequently in primary than in secondary hemorrhage. It is rarely confined to the fourth, still more rarely to the third. The blood is usually coagulated, and if small in amount the clot may correspond in form to the ventricular cavity. If the blood is slowly effused, the distention of the lateral ventricles may be less than that of the third and fourth; in the latter the accumulation of blood is aided by the influence of gravitation in the recumbent posture. In primary hemorrhage the walls of the ventricles may be intact, the choroid plexuses are anæmic from pressure, and it may be difficult to discover the source of the blood. Superficial lacerations of the wall of the distended ventricle may be of secondary origin, as is shown by the fact that there are many of similar aspect. Sometimes it is difficult to say whether laceration is the source of the hemorrhage or is produced by it. Occasionally there are separate extravasations into the substance of the brain; or a meningeal hemorrhage may coexist, even when the ventricular extravasation was small.

The appearances in *meningeal hemorrhage* differ according to its seat and amount. There is a layer of blood either upon the arachnoid or in the subarachnoid space, sometimes in both. The blood accumulates especially in the sulci and depressions, and is generally most abundant at, and sometimes confined to, the base. If considerable in quantity over the convexity, the convolutions may be distinctly flattened. Just as, in ventricular hemorrhage, blood may escape from the fourth ventricle into the meninges, so in extensive meningeal hemorrhage blood may pass into the fourth ventricle, and a little may even find its way into the lateral ventricles. The blood may also pass down into the spinal canal, whether the meningeal hemorrhage is primary, or is due to the escape of blood from the ventricles. It often distends the sheaths of the optic nerves.

SYMPTOMS.—The occurrence of cerebral hemorrhage is indicated by symptoms of two classes, the one general and transient, the other local and more or less permanent. Both sets of symptoms are usually present, but either may be so slight as to be inconspicuous. In addition, the onset may be preceded by what are termed premonitory symptoms, and followed by symptoms outside the cerebral functions, alterations of pulse, temperature, urine, etc.

The premonitory symptoms, so called, are, on the whole, rare. They consist of headache, slight vertigo, weakness or tingling in the limbs, slight mental changes, or slight affection of speech. These symptoms may be continuous or paroxysmal, coming on suddenly, and passing away after a few hours or days. It is doubtful how far it is correct to speak of them as prodromata of hemorrhage. The miliary aneurisms, however numerous, cause no symptoms until they burst. The severe forms of these antecedent disturbances are sometimes due to the occurrence of actual small hemorrhages, or to the general commencement of a hemorrhage that is at first slight and afterward suddenly becomes considerable.* Slighter forms are probably due to the atheroma that often coexists with miliary aneurisms, and are the result of interference with the blood supply to certain parts of the brain. They are thus indicators, not of the cause of the cerebral hemorrhage, but of an associated condition. As might therefore be expected, they are far less frequent before attacks of hemorrhage than before attacks of thrombotic softening, and they are similar in the two cases.

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* Thus, a young man who had suffered from lead poisoning, was attacked with severe pain in the right side of the head, and after this had continued for ten days, left hemiplegia suddenly came on, without loss of consciousness. He subsequently died, and a hemorrhage was found in the right hemisphere, which had damaged the whole thickness of the internal capsule, but had apparently occurred in two stages (Hardy, *Gaz. Méd. de Paris,* June 11, 1880).

Of the general cerebral symptoms the most common is loss of consciousness; of the local symptoms, hemiplegia. The loss of consciousness usually comes on suddenly, and constitutes the most common form of "apoplexy." The patients may fall senseless, without any subjective symptom. More commonly giddiness, pain in the head, weakness in one side or difficulty in speaking, is the first indication of the attack; if the patient is standing, the weakness is noticed chiefly in the leg, which gives way. He sits or lies down, and in the course of a few minutes, or longer, becomes unconscious, seems to go to sleep, but it is a sleep from which he cannot be roused, the sleep of "coma." Occasionally the obscuration of consciousness is incomplete; the patient can be partially roused, or may only seem confused or "dull." Rarely, there is not even this interference with the cerebral functions, and only the sudden onset of the local symptoms announces the occurrence of the hemorrhage.

In the most severe cases, the symptoms of apoplexy, as described in a previous page, are present in the most intense degree. The muscles are relaxed and flaccid, urine and fæces escape, reflex action is abolished, not only in the limbs, but in the conjunctiva and iris. Only the beating of the heart and the movements of breathing indicate the continuance of life, while the irregularity of the former, and the labored character and stertor of the latter, show how frail is the tenure by which life is held. In this state the patient may die a few hours after the onset, but death occurs so rapidly only when the hemorrhage is into the pons or medulla, so as to damage directly the vital centres, cardiac or respiratory. In very rare cases death has occurred within an hour, once in as short a time as five minutes (Abercrombie). Often the coma, although complete, is less deep; the iris still acts to light, liquid placed in the mouth may still be swallowed, the muscles of one side only are flaccid, and on the other muscular tone remains and the raised arm falls less suddenly.

The aspect of the face presents great variations. It may be flushed and turgid, or pale and pinched. The surface of the body is usually wet with perspiration. The pulse is generally at first slow, often small, and incompressible, sometimes quick. Respiration, besides its labor and stertor, may have the Cheyne-Stokes rhythm, always of evil omen. The urine is usually at first abundant, of low specific gravity, and acid in reaction. Occasionally albumen is present for a few hours after the onset when there is no kidney disease.

A convulsion may usher in the attack, but is not common unless the hemorrhage is in the cortex, although hemorrhage into the corpus striatum or elsewhere now and then causes a general convulsion. Vomiting occasionally occurs, more frequently when the hemorrhage is into the cerebellum than when it is in other parts of the brain. Its occurrence is probably influenced in part by the condition of the stomach; if a meal has been taken shortly before the onset, the process of digestion may be arrested, and the contents of the stomach may be vomited.

The temperature usually falls within an hour after the onset, and may be only 97° or 96°; it may even reach 94.4° in the rectum.* In cases fatal during the first twelve hours, the fall may continue until death. In other cases the temperature may become normal at the end of the first day, and remain so in slight cases, or rise, in more severe forms, to two, three, or four degrees above the normal (Bourneville). But there is one apparent exception to the rule of initial depression. In cases of hemorrhage into the pons or medulla, the temperature may rise above the normal within an hour of the onset, and during the next hour may attain a height of 104° or 106°, and the rise may go on until death.

The duration of the apoplectic state varies much in cases that recover from it. At the end of from half an hour to six hours reflex action returns in the limbs, the patient gives some signs of returning consciousness, and moves those parts that are not paralyzed. Difficulty of swallowing and impairment of articulation may exist for a day or two. Dull general headache is usually felt as consciousness returns, and there is occasionally some delirium.

Even during the state of apoplexy some indications of a unilateral lesion may be present; reflex action, if lost, returns on one side only; on the other it remains absent in the foot, cremaster, or abdomen; the muscular tone is different in the two arms, as already mentioned, and sometimes the affected side becomes rigid. The head and eyes may be directed to one side in "conjugate deviation," usually from the side on which the limbs are affected, sometimes toward it. As the general cerebral symptoms lessen, the local symptoms, persisting, become more prominent. The return of movement in the unaffected limbs defines the paralysis, which is usually hemiplegic. When the loss of consciousness is slight or absent, the loss of power is conspicuous from the first, and the subjective symptoms, already described as occasionally attending the onset of apoplexy, rise into greater prominence.

The onset often occurs during sleep. The patient is either found unconscious in the morning, or wakes unaware of what has happened, and only discovers his paralysis when he attempts to stand. The state of sleep has been thought to favor the occurrence of hemorrhage, but it is not proved that the number of cases occurring during sleep is larger than corresponds to the proportion of existence spent in that condition.

In all except the slightest cases, there occurs, two or three days after the onset, slight general febrile disturbance, due to inflammatory changes about the cerebral lesion. There may be merely headache, loss of appetite, quickening (sometimes slowing) of the pulse, and a rise of temperature of one or two degrees, lasting two or three days and then passing away. In other cases the rise of temperature is greater, consciousness may be dulled or even again lost, or there may be some delirium. If consciousness has not been regained, the coma deepens, there is a tendency to sloughing and vesication of the skin, and the patient usually dies. During this stage of inflammatory reaction, rigidity ("early rigidity") often develops in the paralyzed limbs. The urine becomes less abundant, and may lose its acidity.

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* In a case of extensive ventricular hemorrhage, due to the rupture of an aneurism, recorded by Bastian, *Trans. Clin. Soc.*, *1883*, p. 18.

Before the initial coma has passed away it may be suddenly deepened, or consciousness, after being regained, may be again suddenly lost, in consequence of the extravasation bursting into the ventricles. The symptoms of this occurrence are described further on.

As the general cerebral symptoms subside, the local symptoms become prominent. Ultimately only those remain that are due to the local effect of the lesion, and consist in the loss of those functions that are subserved by the structures destroyed. Since the hemorrhage is usually in one side of the brain, the persisting symptoms are usually unilateral in distribution. But the unilateral symptoms that can be at first recognized are much wider in extent than those that ultimately remain. There may be at first an apparent loss of function of almost all parts of one hemisphere, and even of the whole brain. There is not only loss of motor power, but there is often loss of sensibility, and this in cases in which the ultimate loss is purely motor. Indications of hemianopia may often be found in the early stage; if the finger is brought suddenly before the eye, first from one side and then from the other, it will be found that the eyelids blink when the finger comes from the unparalyzed side, and not when it comes from the affected side. The early conjugate deviation of the head and eyes, already mentioned, is sometimes a symptom of the same character. These wide initial symptoms show that the interference with the function of the hemisphere extends at first far beyond the limits of the destruction wrought by the hemorrhage. The effect is probably partly due to the influence of the pressure on the adjacent nerve elements, partly to anæmia produced by that pressure, partly to inhibition by the irritative influence of the acute lesion. These symptoms gradually lessen; some, such as the hemianopia, usually pass away in a few days, others in a few weeks, until there remain only those that are the direct effect of the destruction; these persist for months, and, unless the loss is of such a character that it can be supplemented by the action of the other hemisphere, they continue for the rest of life. The pressure is, of course, greatest on the structures nearest to the hemorrhage, and the symptoms thus produced last longer than do those that result from the slighter pressure on distant parts. The symptoms produced by these two mechanisms have been distinguished as "direct" and "indirect" symptoms.* But the two are the same in character, and can only be distinguished in practice by the graduaal disappearance of the one, and the persistence of the other.

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* A not very happy distinction, since the pressure effects are as much the direct effects of the hemorrhage as is the laceration. Only the inhibitory symptoms are, strictly speaking, indirect, but these cannot be separated practically from those that are due to pressure.

In some cases there is a slight increase of the local symptoms during the period of "inflammatory reaction," due, no douht, to the damage to adjacent structures by the inflammation around the lesion.

Chronic Stage.—The enduring symptoms, which persist after the initial stage is over, are due to the local interference with the functions of the damaged part of the brain, and are determined by the situation of the lesion. Persistent general cerebral symptoms, such, for instance, as are so conspicuous in cases of tumor, are for the most part absent in hemorrhage. Headache is trifling; optic neuritis is practically unknown. Convulsions are very rare. Some mental change is often present, sometimes slight, sometimes considerable, and evidenced chiefly by defective memory, irritability, and emotional mobility. This may be in part due to a general impairment of the functions of the cortex, under the influence of the shock of the lesion, but it more probably depends on damage to the connecting fibres between different parts of the cortex, and between this and the cerebellum, the integrity of which is essential for perfect mental action.

Local symptoms, persistent in most cases, are in a few absent. The lesion is so placed as to cause no recognizable loss of function. When symptoms exist they may be motor, sensory, or trophic, and may affect the limbs or the regions supplied by the cranial nerves. As there is no region of the brain in which hemorrhage may not occur, so there is hardly any observed combination of focal symptoms that hemorrhage may not cause.

By far the most frequent symptom is motor hemiplegia. The reason for this is that the most frequent seat of hemorrhage is the region of the corpus striatum and internal capsule, and the middle, motor part of this rarely escapes laceration or compression. Permanent sensory symptoms, in the form of hemianæsthesia, are less common, because hemorrhage is less common in the neighborhood of the posterior part of the capsule, in which the sensory tract runs. But it is not uncommon, when no absolute loss remains, and the slightest touch can be perceived, for this to be felt somewhat differently on the two sides. Increased sensitiveness to pain is sometimes present, and may coexist with tactile anæsthesia, and be accompanied by spontaneous pains, but these are less common than in cerebral softening. So too are considerable chronic trophic disturbances in the hemiplegic side, persistent elevation of temperature and the joint inflammation, described in the chapter on hemiplegia.

The symptoms that attend hemorrhage in other parts of the brain present some variations according to its seat. The symptoms produced in each locality are, for the most part, those that have been described at a previous page (p. 708). A few additional facts, relating especially to hemorrhage, may be here considered.

Cortex.—Hemorrhage into the substance of the cortex is very rare, but occurs occasionally from aneurisms about which there is sufficient inflammatory thickening to prevent external rupture. The onset is usually attended by convulsions, which are not general, as are those that occasionally result from cerebral hemorrhage elsewhere, but begin locally in the manner characteristic of cortical disease. Subsequent paralysis may affect only part of one side. Hemorrhage into the centrum ovale causes symptoms similar to those due to a cortical lesion in the corresponding situation, but without the symptoms of irritation.

Crus Cerebri.—A limited extravasation may cause the characteristic crossed palsy of third nerves and limbs, but often the hemorrhage passes beyond the limits of the crus, either upward into the foot of the internal capsule (and then causes well-marked hemianæsthesia, as well as hemiplegia) or downward into the pons, and may then paralyze both third nerves, and the limbs on both sides. The corpora quadrigemina are never the seat of limited hemorrhage.

Pons Varolii.—Initial loss of consciousness may be present or absent, just as in hemorrhage elsewhere. Initial convulsions are especially frequent, usually general, rarely unilateral, sometimes affecting the legs only, a symptom almost unknown from disease in other parts. The convulsion is often irregular in type, sometimes tonic or toxic, varied with occasional clonic jerkings. The paralysis is often bilateral, but a small hemorrhage may cause hemiplegia from the first. The paralysis sometimes affects mainly the legs or the arms. Anæsthesia often accompanies, and may preponderate over, the motor palsy. The pupils are often strongly contracted, so as to suggest opium poisoning, or they may be dilated and motionless, the difference depending on the irritation or paralysis of the nuclei of the third nerve. The deviation of the head and eyes from the side of the lesion, described at p. 512, is occasionally observed. Respiration often suffers early, and may present irregularities, sometimes almost convulsive in their character. Vomiting is frequent. The temperature often rises rapidly to a hyperpyrexial degree. Death is usually more speedy than in hemorrhage into the cerebrum; it has been known to occur in seven minutes from the onset.*

Medulla Oblongata.—A considerable hemorrhage causes death very quickly, and even instantaneously. It is doubtful whether convulsions occur when the extravasation is confined to the medulla. The patient rarely survives the actual onset. The symptoms that may persist resemble those of bulbar paralysis, but this is extremely rare, only one conclusive case being on record. Apoplectiform bulbar paralysis is due, in the vast majority of cases, to softening from vascular occlusion, not to hemorrhage.

Cerebellum.—Loss of consciousness is as common as in hemorrhage elsewhere, and presents the same variations in its degree. There is often no initial paralysis; sometimes there is hemiplegia, due to pressure, and it may be on the same side as the hemorrhage, or on the opposite side, according as the pressure is exerted on the pons or on the medulla. The absence of hemiplegia is more significant than its presence. The pressure may also cause various paralyses in the parts supplied by the cranial nerves that arise from the pons and medulla. The state of the pupils varies; vision is unaffected. Vomiting is more frequent than in hemorrhage elsewhere, being met with in half the cases, and it is also more often persistent. It may occur without, as well as with, loss of consciousness. If recovery takes place, the pressure effects, including the hemiplegia, pass away, and the only lasting symptom is cerebellar unsteadiness, and this persists in some but not in all the cases. Hemorrhage into the cerebellum often bursts into the fourth ventricle, causing the rapid depression of the functions of the medulla described at a subsequent page. When the middle cerebellar peduncle is the seat of hemorrhage, the characteristic symptoms, forced lateral position of the head, difference in height of the eyes, and a tendency to lie on one side, and even to rotate, are usually distinct at the onset, sometimes during the stage of apoplexy, and if the patient is conscious there is usually intense vertigo.

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* Mickle, *British Med. Journal, 1881*, ii, 151.

Ventricular Hemorrhage.—The effusion of blood into the ventricles of the brain is indicated by severe apopleptic symptoms, the origin of which is easy to understand. When, as in the majority of cases, the rupture is into one lateral ventricle, the blood rapidly distends the other also, and the whole cortex is compressed. The blood soon passes into the fourth ventricle, and compresses the important structures that lie in its floor. In the rare cases in which the rupture is into the fourth ventricle, the pons and medulla bear the full force of its pressure, and the grave symptoms of interference with this part of the brain are conspicuous from the onset, and resemble closely those produced by hemorrhage into the substance of the pons. This is also the case when the rupture is into the lateral ventricles, and occurs slowly; the fourth ventricle may then be much more distended than the lateral ventricles (see p. 784).

Hemorrhage into the ventricles is sometimes primary, but much more frequently secondary to hemorrhage into the cerebral substance, from which the blood tears its way into the cavities. In the former case the severe apoplexy is primary; in the latter it supervenes on the symptoms of the cerebral hemorrhage already described. The primary apoplexy may have passed away or have lessened, or may be still at its height, when it suddenly returns or becomes deeper. The initial deviation of the head and eyes ceases, and is often replaced by a deviation in the opposite direction. The pulse is again slowed, and may fall to 50 or 40 beats per minute. The temperature may fall to 97° or 96°. The respiration becomes more labored and stertorous. The reflex action is again lost in the limbs and eye; the pupils are sometimes dilated, sometimes contracted; rigidity often appears in the limbs on the hemiplegic side, sometimes in those on the other side; there may be convulsions, sometimes general, sometimes affecting only the unparalyzed side. Paroxysms of general and clonic spasm, with slight opisthotonos, have been observed.* After a few hours the pulse often becomes more frequent; the temperature may remain low or may rise, sometimes to 104° or higher. The chest becomes filled with râles, occasionally with extreme rapidity, even within a couple of hours of the onset; the face becomes livid, respiration is increasingly difficult, and the patient dies from the interference with breathing. In many cases the blood escapes from the fourth ventricle into the sub-arachnoid space at the base of the brain, and the symptoms may be in part due to this secondary (or rather tertiary) meningeal hemorrhage.

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* Bastian, *Trans. Clin. Soc.*, *1884*, p. 22.

Primary ventricular hemorrhage causes symptoms which may, from the first, closely resemble those of the secondary form, but more frequently the onset resembles that of hemorrhage into the substance of the brain, in the presence at first of unilateral symptoms. Prodromata are rare, but headache is occasionally met with, very variable in seat, character and duration. The onset may be (1) By sudden apoplexy, deepening rapidly; death may occur in a few hours. (2) By apoplexy with hemiplegic symptoms, or with convulsions. (3) In the very rare slow hemorrhage, hemiplegia first occurs alone, loss of consciousness only supervening after a few hours. Hemiplegia occurs because the blood is effused first into one lateral ventricle, and causes paralysis on the opposite side by the compression of the motor path or centres. When the effusion is rapid and both lateral ventricles quickly become distended, the unilateral symptoms quickly give place to general relaxation of the muscles and loss of all reflex action. Rigidity is often met with, but less frequently than in the secondary form; it is usually bilateral, sometimes one-sided, and occasionally involves only the muscles of mastication; it is often intermittent. Convulsions are also frequent, occurring in at least a third of the cases, sometimes general, sometimes affecting only the paralyzed side, or only a part of it. In cases of slow onset, speech is often lost before consciousness. The power of swallowing usually persists until the apoplexy becomes profound. The temperature resembles that of other forms of cerebral hemorrhage. The malady is usually fatal, but recovery has occurred, as is proved by old and altered clot being sometimes found in the lateral ventricles, but it is possible only when the hemorrhage is small in quantity and the symptoms are then slight and equivocal.

Meningeal Hemorrhage.—The symptoms of meningeal hemorrhage vary much according to its cause. The rupture of a large aneurism at the base causes severe apoplexy, rapidly deepening to lethal intensity. But since slight hemorrhage sometimes occurs before the final rupture, the loss of consciousness may be preceded by definite prodromata, and is so preceded more frequently than is intra-cerebral hemorrhage. These prodromata are severe headache (sometimes occipital), giddiness, and occasionally vomiting. The attack itself is attended by paralysis and atony of the limbs on both sides, and sometimes by palsy of cranial nerves or by convulsions.

When the hemorrhage is of traumatic origin the effect of the injury obscures the initial symptoms. In these and other cases in which the escape of blood is gradual, the patient may recover consciousness and continue his occupation for some hours or even for a day or two, complaining only of headache, and then gradually become somnolent and pass into a state of coma.

In some cases of meningeal hemorrhage, convulsions are the most prominent symptom, and they may be either general or unilateral, and, in the latter case, may commence locally in the face or arm, or by deviation of the head. Rigidity of limb is comparatively rare, far more so than in meningitis. In some cases there is mental excitement or delirium, in others there is mental dullness. The state of the pupils is very variable; they may be contracted or dilated or unequal. Sometimes headache and giddiness are accompanied by tingling in the limbs and weakness on one or both sides. Initial apoplexy may be absent, and these symptoms, commencing suddenly, may increase until consciousness suffers. The variation presented by these cases is thus very great.

In the majority of cases the patients die in the state of coma, and sometimes apparently from the violence of the convulsions. Coma sometimes passes away and recurs. In slight cases recovery is possible, as subsequent post-mortem examination has proved. The symptoms of the meningeal hemorrhage of newly-born children are separately described.

PATHOLOGY.—While the rupture of the vessel is always the result of weakening of its wall, and of the pressure of blood within it, the actual conditions of rupture vary considerably. The variations relate to the degree of blood pressure—the size of the artery, of the aneurism, and of the opening in it—the freedom of exit of the blood, or the hindrance to its escape by clot within the dilated part, and the resistance in the tissue into which the blood passes, which is less in the gray than in the white substance. The precise conditions in any individual case can rarely be traced, but on them must depend the size of the hemorrhage, and the rapidity with which the blood is effused. These two elements chiefly determine the symptoms that attend the onset. No doubt the occurrence of both aneurism and hemorrhage is due largely to the slightness of the support that the cerebral tissue affords to its vessels. This is less in the old than in the young, on account of the larger size of the perivascular spaces, occupied only by mobile liquid.

It is a well-known law of hydrostatics that if a liquid passes into a closed chamber by a small opening, the total pressure within the chamber is that in the opening multiplied by the number of times the area of the wall of the chamber exceeds the opening in size. Thus the total pressure within the aneurism must be much greater than in the artery from which it springs, and, at the same time, the wall is weakened. Hence, we can understand the tendency to rupture. Moreover, the same law will obtain after rupture, since the blood in the cavity formed by the extravasation is influenced by the pressure exerted through the opening into the aneurism. No doubt the actual condition is very different from that in a closed chamber, and the result is correspondingly different, but the law must hold good in some degree. We are thus able to understand how so small a jet of blood may produce a cavity in the brain of so large a size. Doubtless the process is facilitated in some cases by the softening and disintegration of the tissue around the blood by imbibition. On the other hand, the transmission of the pressure must be modified and retarded when the blood begins to coagulate.

The most common seat of hemorrhage is the region of the corpus striatum, because the arteries that most frequently rupture are the branches that come off at right angles from the middle cerebral, in the fissure of Sylvius, and pass upward through the lenticular nucleus and the internal capsule to the caudate nucleus and optic thalamus (see Fig. 214, p. 497). A large artery which passes between the outer part of the lenticular nucleus and the external capsule, then through the former to the internal capsule, is so frequently the source of the extravasation that it has been termed by Charcot *par excellence* "the artery of cerebral hemorrhage." A small hemorrhage from it simply separates the external capsule from the lenticular nucleus, occupying a narrow fissure thus formed, which may correspond to almost the whole outer aspect of the lenticular nucleus, but a large hemorrhage displaces inward and erodes all the central ganglia. All these branches of the middle cerebral pass to the internal capsule, and as this lies over the inner-upper aspect of the lenticular nucleus, it rarely escapes damage—by pressure if the hemorrhage is small, by laceration if it is large. Hence hemiplegia, transient or permanent, is so common a symptom. The branches that have a posterior course, to the hinder part of the capsule, less frequently give way, and hence lasting hemianæsthesia is comparatively rare.

The mechanism by which the loss of consciousness and the other symptoms of apoplexy are produced has been the subject of much discussion and speculation. Of this, some account has been given on a previous page (534). It is only necessary here to repeat that we must recognize a double mechanism, the mechanical effect of the pressure on the cortex, and the inhibition of its cells by the mechanical irritation of the torn nerve fibres. Both of these effects are the greater the more rapidly the blood is poured out, and the larger its amount, while the inhibitory effect is doubtless influenced also by the position of the lesion; injury to structures that have an extensive connection with the cortex being the most effective.

Thus, consciousness is preserved at the onset, only when the hemorrhage is small, or the blood escapes very slowly, or when the extravasation is so placed that the irritation has but a slight influence on the cortex. Hemorrhage into the pons usually affects consciousness, even when small, probably because the fibres that pass through the pons have a very wide connection with the cortex. When the effusion occurs slowly but the conditions are unfavorable to the cessation of the flow of blood, consciousness may be lost, not suddenly at the onset, but gradually as the hemorrhage attains a considerable size, giving rise to the "ingravescent apoplexy." As Broadbent has pointed out, this form is usually due to rupture of the artery that passes outside the corpus striatum. It must not be thought that ingravescent apoplexy is the common result of rupture of this vessel. Much more frequently there is sudden initial apoplexy, and the occurrence or absence of this initial loss probably depends on the character of the rupture in the wall, whether it is large or small. Ingravescent apoplexy may also occur from rupture of a vein; the pressure in the vein is low, and the blood escapes slowly. Hence it is that in traumatic hemorrhage (which is often from a vein) many hours may pass, during which the patient's intellect is unaffected, before consciousness is lost.

Diagnosis.—The diagnosis of the nature of the cerebral lesion has to be made under two conditions, first during the initial apoplexy, and secondly, when this has passed, and only the enduring effects of the lesion remain. The first is incomparably the most important, since by it the treatment has to be determined. Unfortunately, it is often, of all the diagnostic problems presented to the physician, at once the most difficult and the most urgent.

The first question in diagnosis is, whether an attack of apoplexy is of cerebral origin. The chief points in the differential diagnosis have been considered in the account of this condition. If cerebral symptoms, such as unilateral numbness or weakness, have preceded the loss of consciousness, or are to be traced in it, as by one-sided relaxation of limb or deviation of the head, the cerebral nature of the attack is clear. If these are absent the question must be determined by the considerations mentioned at p. 534. An actually sudden onset limits the diagnosis to the distinction from syncope, and this should be easy. If the onset was gradual, or, as is often the case, its character cannot be ascertained, the distinction has to be made from poisoning (especially by chloral, opium and alcohol), and from uræmia, by the indications already described in the section on apoplexy.

The distinction from the apoplectiform attacks of cerebral congestion, of general paralysis of the insane, and from the so-called "simple apoplexy," in which there is no sign of congestion, and after death no lesion of the brain, is more difficult. In all three, local symptoms are absent. In congestion the coma is rarely profound; the loss of consciousness is often imperfect, and is generally brief. The only symptoms are general; there is no local loss of power. There have usually been previous attacks of the same character, transient, leaving no after symptoms. In a first attack, or without a history, the diagnosis may be impossible. In general paralysis of the insane, the preceding symptoms are almost always sufficiently pronounced to indicate the nature of the apoplectiform seizure. The attack lasts only a few hours, and the patient rapidly recovers his ordinary state. They are more readily confused with attacks of simple congestion than with hemorrhage.

The attacks of "simple apoplexy" that occur in the old, mysterious in their nature, may perfectly resemble the apoplexy of cerebral hemorrhage, and it is doubtful whether a distinction between the two is possible in practice. Lastly, it may seem strange that so grave and sudden a lesion can be regarded as hysterical, but the mistake has been made by permitting the sex and age of the patient to warp the judgment of the observer, and prevent the recognition of the significance of the symptoms.

If the presence or history of local symptoms makes it certain, or the intensity of the general symptoms renders it in high degree probable, that the attack is due to an organic cerebral lesion, the chief diagnostic question is whether the lesion is hemorrhage, or softening from vascular occlusion. If the patient is under forty, the presence of heart disease, or, in its absence, a suspicion of syphilis, renders vascular occlusion far more probable than hemorrhage, provided the apoplectic symptoms are of moderate severity. But these causal indications do not absolutely exclude hemorrhage, even in early adult life or in childhood, since both these conditions are undoubted causes of aneurism of the larger cerebral arteries. The rupture of an aneurism is probable, if, under such circumstances, the apoplexy is intense in degree and the coma rapidly deepens. In a case of heart disease, preceding cerebral symptoms, headache, etc., increase the probability of aneurism, but in syphilis they only have this significance if they are such as to indicate the existence of a compressing tumor, since slight cerebral symptoms may be due to ordinary syphilitic disease of the vessels.

In the second half of life, especially after forty-five, when miliary aneurisms become common, neither the presence of heart disease nor a history of syphilis affords the same strong presumption against hemorrhage that it does in early life, and their significance is subordinate. Thrombotic softening from atheroma of the arteries becomes common, *pari passu* with hemorrhage, and it is only when the symptoms make it probable that the lesion is vascular occlusion, and not vascular rupture, that the heart disease or syphilis raises a question as to the cause of the occlusion. The older the patient the less weight do these points deserve; because atheroma increases in frequency with advancing years, and syphilitic disease lessens in frequency, while the degenerative valvular disease of the heart of the old causes embolism far less frequently than do the endocarditic lesions of the young.

Between thrombotic softening and hemorrhage the diagnosis is often difficult and sometimes impossible. Some recent writers go so far as to say that even a probable diagnosis can be made in only a third of the cases; but this is, I think, an under-statement. The distinction is of great importance, because the treatment suitable to the two conditions is very different. Between the two there is never, in any case, an unfailing criterion. A probable diagnosis can be made only by comparing the several symptoms, cerebral and general, and balancing their indications. Neither age nor sex gives help, except that, in extreme old age, over eighty, there is a probability in favor of softening rather than hemorrhage. The conformation and nutrition of the patient are not of much significance. A turgid face, and strongly beating arteries of the neck, is in favor of hemorrhage, but a thin, worn, aged aspect does not render hemorrhage less probable. Considerable degeneration of the arteries of the limbs is somewhat in favor of softening, as Nothnagel has pointed out. A high arterial tension is decidedly in favor of hemorrhage, and this irrespective of the size of the pulse. The state of the heart is of great importance; a hypertrophied, strongly acting heart renders hemorrhage probable; if the heart is feeble, dilated, and irregular in consequence of its disease, softening is far more likely than hemorrhage. But in deep coma, it must be remembered, irregularity may be due to the cerebral lesion ; the pulse is then usually infrequent. In Bright's disease, especially the granular kidney, there is a slight, but only a slight, probability in favor of hemorrhage, for atheroma and softening are also frequent consequences. Hemorrhages in the retina in connection with albuminuric retinitis do not render cerebral hemorrhage more probable, unless they are large. Visible aneurisms in the retinal arteries probably do constitute strong evidence of cerebral hemorrhage, but they are very rare. If the attack was apparently induced immediately by strong mental excitement, the fact is in favor of hemorrhage ; if by prolonged grief, it is in favor of softening.

Prodromata of some duration in the form of numbness, tingling, or weakness in the side afterward paralyzed are in favor of softening, and so also are previous headache or slight attacks of weakness in other parts. Local convulsions at the onset of the attack are also in favor of softening, while a general convulsion is in favor of hemorrhage. The significance of the occurrence of loss of consciousness at the onset depends on other associated conditions. As a general rule loss of consciousness is more frequent, greater in degree, longer in duration, and occurs with a smaller lesion, in hemorrhage than in softening. Its significance depends, therefore, on the probable size of the lesion. If there is reason to think that this is small, loss of consciousness is in favor of hemorrhage ; if it is probably large, the absence of apoplexy is strong evidence of softening. But it is unfortunately very difficult to judge of the extent of the lesion in the early stage. Partial hemiplegia is, however, almost always due to a small lesion, and the occurrence of initial apoplexy in such a case is in favor of hemorrhage. Deep coma is always in favor of hemorrhage when there are unilateral symptoms; with bilateral symptoms it is of rather less significance, since bilateral thrombosis may cause apoplexy as deep as that produced by ventricular hemorrhage, but as bilateral thrombosis is much rarer than ventricular hemorrhage, the fact does not altogether destroy the value of the indication. A considerable initial fall of temperature, exceeding 1°, or a considerable rise within a few hours, is in favor of hemorrhage, provided the symptoms do not indicate obstruction of the basilar, in which there may be an initial fall similar to that of hemorrhage. The greater the secondary inflammatory disturbance, the more probable is softening, especially if there are also secondary convulsions. In the subsequent chronic stage of hemiplegia, the most important indications are that mobile spasm (athetosis, post-hemiplegic chorea, etc.) and recurring convulsions beginning in the paralyzed limbs, are strong evidence that the lesion was softening, and not hemorrhage.

The diagnosis of secondary ventricular hemorrhage rests on the occurrence of a second apoplectic seizure, or on the distinct intensification of primary coma, with the extension to the second side of the muscular relaxation that was at first unilateral. But the significance of this depends on the initial attack having the character of hemorrhage. When there is thrombosis in an artery of one hemisphere, the formation of another clot in a large artery on the opposite side may be attended with exactly the same symptoms as the rupture of a hemorrhage into the ventricles ; hence the occurrence of such symptoms should not be allowed to influence a diagnosis of thrombosis if the evidence of this was distinct.

The diagnosis of a primary ventricular hemorrhage is rarely possible. There is no distinctive symptom, and the combination of symptoms that attend it may be produced by hemorrhage into the substance of the brain or into the meninges. It may, however, be suspected if severe apoplexy comes on in early life, without preceding symptoms to suggest an aneurism.

In meningeal hemorrhage the diagnostic problem differs according as there is or is not apoplexy. If there is, the distinction is chiefly from intra-cerebral hemorrhage, and this is often impossible from the symptoms alone. If there is no initial loss of consciousness, the symptoms are chiefly headache, delirium and convulsions, and, developing gradually, may closely resemble those of meningitis. In all cases an important element in diagnosis is the condition under which the disease occurs. In the young, in whom alone the diagnostic difficulty is likely to occur, meningeal hemorrhage very seldom occurs except after injury. In the old, in whom spontaneous hemorrhage may be met with, primary meningitis is very unlikely.

Prognosis.—Two questions always present themselves: first, the risk of death; secondly, the prospect of recovery from the resulting paralysis. The initial danger is proportioned to the intensity and duration of the coma. If it has not begun to lessen at the end of twenty-four hours the probability is against recovery. Most cases die in which there is marked interference with respiration, indicated by its sighing character, Cheyne-Stokes' rhythm, or by the accumulation of mucus in the lungs. Bilateral symptoms in the limbs are also of very grave significance, indicating, as they usually do, either hemorrhage into the ventricles or into the pons. The lower the initial temperature the graver is the immediate prognosis. A considerable rise of temperature within a few hours of the onset is also very serious, and so is the early appearance of albumen or sugar in the urine. If the initial symptoms were severe, or the patient is old or feeble, the period of secondary inflammation is also attended with danger, the indications of which are considerable fever, delirium, or a tendency to the formation of sloughs and bed sores. Most cases die in which a slough appears on the buttock before the end of the first week. In ventricular hemorrhage the prognosis is almost certainly fatal. In the few cases of the primary form that recover, the hemorrhage is small in amount, and the diagnosis of the condition, as a rule, impossible. In meningeal hemorrhage with coma the prognosis is exceedingly grave; it is probable that many slight traumatic cases get well, but in such cases the diagnosis is doubtful.

The prognosis as regards the paralysis depends on whether it is due to direct destruction of the nerve elements by a lesion involving the conducting path, or whether it is due to indirect damage by a lesion adjacent to it. It is usually necessary to wait for a diminution in the palsy before an opinion can be formed. The parts in which there is some return of movement before the end of a month will probably recover useful power. Paralysis that is complete at the end of three months will probably remain considerable in degree for the rest of life.

TREATMENT.—Little can be done to prevent the occurrence of cerebral hemorrhage, because miliary aneurisms, its main cause, are almost entirely beyond control. The degenerative chances incidental to age, or the result of an inherited tendency, cannot be arrested, and perhaps not even retarded. A tranquil life, as free as may be from severe exertion of body or anxiety of mind, with nutritious but light food, probably has some tendency to lessen the rapidity of the deterioration of the tissues, but the cases are few in which these conditions can be secured at will. The other factor in the production and rupture of aneurisms, the intra-arterial pressure, can be to some extent influenced by occasional saline aperients and diuretics. In persons who are developing the conditions favorable to hemorrhage, and especially in those whose relations have suffered from apoplexy, these measures are of especial importance, as is also the avoidance of severe muscular exertion, which entails a sudden increase of the strain upon the arteries. It is almost superfluous to add that a preceding cerebral attack renders these measures doubly important.

In the attack itself the aim of treatment must be to secure the conditions that favor the arrest of the bleeding. Physical rest is of the utmost importance. The patient should be laid down, but with the shoulders and head well raised. The clothes about the neck should be loosened, and flexion of the neck avoided, so as to prevent the hindrance to the return of blood from the head that compression of the veins would cause. All muscular effort should be forbidden. It cannot be doubted that, in many cases, the amount of hemorrhage has been greatly increased by the patient walking about after the onset of the attack. Even passive movement should be as little as possible.

Venesection was for long regarded as the most important element in treatment; and many authorities are still of the same opinion, although the disrepute into which the lancet has fallen leads to hesitation in its use. Formerly the surgeon did not scruple, in any case of apoplexy, to cut across the temporal artery to obtain a free flow of blood. No other agent reduces so quickly and so considerably the tension of the blood, and patients occasionally regain consciousness while the blood is flowing. On the other hand, it has been objected that external hemorrhage only ceases when the loss of blood weakens the heart's action to a degree that would increase the peril of an extensive hemorrhage into the brain. It is also said that, in most cases in which venesection is employed, no distinct beneficial influence can be traced. Both these statements are true. The conditions of cerebral hemorrhage are, however, in many respects unlike those of surface bleeding. Some of the influences tending to arrest are more powerful, and these may be aided by a slighter flow of blood than would be necessary to stop a surface bleeding. Nor is the absence of immediate evidence of its utility a consideration of much weight. Indeed, it is somewhat strange that such evidence should ever be forthcoming, when it is remembered that the arrest of the bleeding does not at the moment lessen any of the conditions on which the loss of consciousness depends.

But venesection should not be employed indiscriminately. The diagnosis of hemorrhage should be reasonably certain, since in thrombosis loss of blood will do only harm, by weakening the heart and favoring the extension of the clot. .This effect, to be desired in hemorrhage, is to be dreaded in softening. The indications for venesection are a regular, strongly-acting heart, and an incompressible pulse. They are strengthened by distinct hypertrophy of the heart, by full arteries, strongly pulsating carotids and a turgid face. The contra-indications are softness of the pulse, irregularity and dilation of the heart. If the apoplexy is so deep that the respiration and heart are suffering, it is very doubtful whether venesection can do any good. When it is indicated, the sooner the blood is drawn the better. A large opening should be made into the vein, and ten or twelve ounces allowed to flow as quickly as possible. The application of leeches has probably very little influence.

The next important element is free purgation. This has a powerful influence on the cerebral circulation, by filling the capacious vessels of the intestinal canal. It should always be employed, but its slowness renders it an inefficient substitute for bleeding if the symptoms are urgent and venesection is distinctly indicated. Croton oil or calomel is the most convenient agent. It is unfortunate that at present we have no purgative that can be used hypodermically. With the same object a diuretic should be given.

To promote the contraction of the cerebral vesssls, ice may be applied to the head, provided the patient is not collapsed, and a grain of ergotine may be injected under the skin. Mustard plasters to the nape of the neck may be employed for the same purpose, to induce reflex contraction of the arteries, if the patient's condition is one of pallor and collapse. The old practice of applying them to the soles and calves rests on an intelligible basis, since even from these distant parts an effect may be produced on the cerebral vessels, and the dilatation of the vessels of the surface must aid in attracting the blood from the brain.

Alcohol should be avoided unless the patient's collapse and cardiac depression are extreme. If a mild stimulant is indicated, ammonia may be given. It is very desirable to arrest convulsions and vomiting, because these tend to increase the bleeding. In one case, an injection of thirty grains of chloral into the rectum appeared to stop the convulsions.*

In all cases, but especially if the coma is prolonged, or the secondary pyrexia considerable, the danger of the occurrence of bed sores must be borne in mind, and should be obviated as far as possible by extreme cleanliness, by a water bed, and by changing the position of the limbs from time to time, so as to prevent long-continued pressure on the same spot. Hot-water bottles should also be employed with great care, since a degree of warmth that can be borne with impunity in health will cause a blister and slough in early hemiplegia.

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* Bastian, *Trans. Clin. Soc.*, xvii, 1884, 22.

In the after-treatment of these cases, when the disturbance of the onset has passed away, and slowly lessening palsy remains, it is important, with the view of preventing a recurrence, to observe the rules of life described at the commencement of this section. The treatment of the consequences of the lesion, such as hemiplegia, is the same as when they are due to softening, in the account of which they are described.

The treatment of ventricular and meningeal hemorrhage does not differ from that of the intra-cerebral form, although little can be hoped for from any measures that can be adopted.

INFANTILE MENINGEAL HEMORRHAGE (CEREBRAL BIRTH PALSY).

Meningeal hemorrhage is occasionally produced during birth ; the accompanying damage to the brain is the cause of permanent symptoms, weakness of the limbs and of the trunk, incoördination, spontaneous movements, convulsions, and mental defect. The cases vary much in degree, and it is only in the more severe forms that all these symptoms are present. In some cases the legs suffer chiefly, and the condition is then termed "congenital spastic paraplegia," a malady that has already been described (p. 328). When the spontaneous movements constitute the most conspicuous symptom, the state has been described as "congenital chorea."

The relation of these symptoms to injury to the brain during birth was suggested by Dr. Little,* and has been clearly established by the researches of an American physician, Dr. Sarah McNutt.† The following account of the symptoms is based on thirty-six cases that have come under my notice.

The hemorrhage that injures the brain is almost always due to special conditions of parturition. In some cases the presentation is unnatural, and the head is born last ; in such cases intense mechanical congestion accompanies the compression, and the occurrence of hemorrhage is easy to understand. This was the cause of one-fifth of the cases that have come under my notice (seven out of thirty-six). In the cases of head presentation, there has almost always been a special difficulty in the birth ; the labor was prolonged, and often there was great difficulty in eliciting signs of life in the child. The majority of the children are first-born ; of twenty-four cases of head presentation in which this point was noted, the labor was the first in seventeen instances. In most of the multiparous cases the previous labors had also been difficult. In one case, for instance, in which it was the third confinement, each of the two preceding labors had been so prolonged, in consequence of a very narrow pelvis, that the children were dead. In some of the cases the forceps was used, but it is probable that the lesion was the result, not of the use of the instruments, but of the conditions that made them necessary. In one case, a fifth labor, the forceps was not used, although it had been employed in each of the preceding labors, and the children had escaped injury. The condition seems to be as frequent in one sex as in the other; of thirty-four cases in which the sex was noted seventeen were girls and seventeen boys.

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* "Nature and Treatment of Deformities," 1853 ; *Obstetrical Trans., 1862.*

† *American Journal of Obstetrics,* January, 1885, and the *Am. Journ. of Med. Science,* January, 1885.

In the majority of cases nothing particular is noticed in the condition of the child during the first few days or weeks of life. In a few, however, conspicuous symptoms attract attention. One arm and leg are not moved; sometimes none of the limbs are moved, but the latter receive less notice than does the one-sided immobility. Another frequent symptom is convulsion, general or one-sided, and often accompanied by persistent rigidity of the limbs and with inversion of the thumbs. Rarely the head is bent back. The convulsions usually cease at the end of a week or two. In many cases it is not until the child is four or five months old that a deficiency of movement attracts attention; in others, again, suspicion is only excited when the child fails to stand and walk at the age at which it should begin to do so. Sometimes the first thing that is noted is strong adductor spasm in the legs; in other cases it is the inability to affect ordered movements with the hands. In some instances, convulsions are the most prominent symptoms, and the difficulty in the use of the limbs is often ascribed to them.

The symptoms, which usually increase during the first two years of life, consist of weakness, spasm, incoördination and spontaneous movements. Their distribution varies, but all four limbs are affected in about two-thirds of the cases (twenty-two out of thirty-six), and the trunk and neck muscles are also conspicuously involved in about one-third (eleven cases). In a small proportion the limbs of one side only are affected (seven cases), and in rather fewer (six cases) the legs alone suffer in considerable degree. Very rarely the symptoms are confined to a single limb.

In the arms there is weakness and some spasm, which varies in character, especially toward the extremity of the limb, and interferes with voluntary movement. The elbow is generally flexed, sometimes extended. The wrist is at times bent at a right angle to the forearm, at other times it is over-extended. The persistent strong flexion may even lead to subluxation. The thumbs are inverted or extended; sometimes the metacarpal bone is extended and the phalanges flexed. The fingers are flexed at the metacarpo-phalangeal joints, and in many cases they are over-extended at the middle joint, which may be subluxated. Often, however, the fingers and wrist are in constant movement, now flexed and now irregularly extended. The movement is generally slow, like the post-hemiplegic athetoid spasm; sometimes it is quick, and may bear considerable resemblance to the movements of chorea. In slight cases the amount of spasm is trifling, and its chief effect is the interference with voluntary movement, which always exists in some degree. The fingers are separated and moved irregularly in an attempt to take hold of an object. It is often impossible for the child to pick up an object, although it can grasp firmly but slowly. In a severe case, when the hand has at last closed, it may be very difficult for the child to relax the grasp. The condition of the legs is that described and figured at p. 329. Any attempt to move them, and any peripheral impression, excites extensor spasm, which renders the limbs rigid, the heel being drawn up and the toes pointed down. There is commonly some persistent contracture in the calf muscles, but this can almost always be completely overcome by steady pressure on the sole. Often the feet are slightly inverted, and I have once seen such extreme talipes varus that each foot was turned in and fixed at a right angle to the leg. Occasionally there is some persistent contraction of the flexors of the knee. There is often also spasm in the adductors of the thighs, which may be sufficient to carry one leg in front of the other on an attempt to stand or walk. The knee jerk is excessive, but foot clonus can seldom be obtained. Spontaneous movements of the toes and feet may occasionally be observed. In very rare cases the arms suffer more than the legs. The limbs that are affected are generally thin, but there is never extreme muscular wasting. Diminished length of limb may be perceptible when the affection is one-sided, but if bot sides are involvehd on difference can be found.

The muscles of the trunk are sometimes weakened, so that the child has a difficulty in sitting up. Still more frequently there is a difficulty in supporting the head. There is never distinct paralysis of the face, but sometimes traces of spasm may be observed about the mouth. In rare cases there is some unsteadiness of the tongue, and occasionally difficulty in swallowing and defective articulation exist, but the latter is usually due to another frequent symptom, mental defect. This exists in at least two-thirds of the cases, from a doubtful amount to complete idiocy with permanent inability to speak. In most of the cases in which mental power is low, the affection of the limbs is considerable, but the opposite rule does not hold good ; the muscular affection may be severe and general, although the mind is nearly or quite normal. Defect of sensation can never be demonstrated. I have once seen considerable defect of sight (associated with great mental weakness) ; the ophthalmoscopic appearances and action of the pupils were normal.

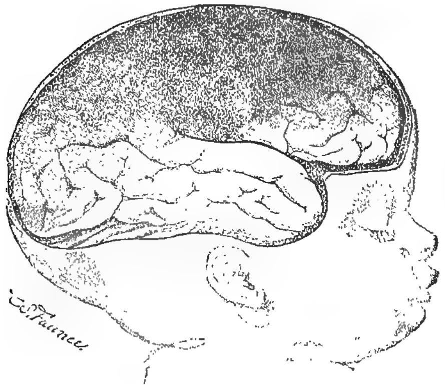
The affection of the two sides is seldom equal, and the two limbs most affected are always on the same side. Occasionally, as already stated, one side escapes, and if the right limbs are those involved the child is left-handed. When the legs seem to be alone affected, slight incoördination, or at least awkwardness of movement, may generally be observed in one arm or in both.

Convulsive attacks occur only in a small proportion of cases, and they are rather more frequent when the affection is one-sided than when it is general. They seldom date from birth; initial convulsions rarely persist. Subsequent fits begin generally during the second half of the first year; they may cease, after lasting for a short time, or may recur. The occurrence of fits bears no relation to the severity of the limb symptoms ; and, indeed, the latter may be scarcely recognizable when the former are severe. If the limb symptoms are one-sided, so also, as a rule, are the convulsions.

The subsequent course of these cases is one of slow improvement, but it is only when symptoms are slight that they approximately disappear. There is a peculiar oscillation in walking, and for a long time the patient may walk on the ball of the foot, the toes not touching the ground. Some peculiarity of gait may persist through life.

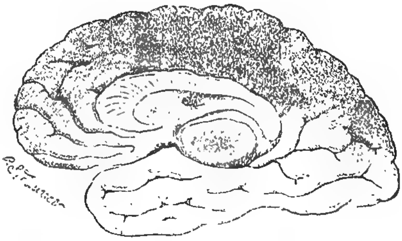
Pathology.—In many of these cases, as we have seen, convulsions, rigidity and paralysis are observed immediately after birth. If children die, who present such symptoms, meningeal hemorrhage is invariably found. The extravasation is sometimes over the convexity of the brain, sometimes at the base. In the former situation it is generally bilateral, and is most considerable over the central region and toward the middle line (Fig. 286); it is found also on the medial aspect of the hemisphere (Fig. 287); sometimes it exists only over the posterior part of the convexity. Where the layer of blood is thicker the convolutions are much compressed, and are sometimes much injured, the cerebral tissue being broken up and infiltrated with blood. The hemorrhage at the base is chiefly in the posterior fossa beneath the tentorium, surrounding the pons, medulla and cerebellum, and it generally proceeds from a laceration in the cerebellar hemisphere. Dr.

Fig. 286.



Meningeal hemorrhage during birth. The shading represents the extravasation.

Fig. 287.



Medial aspect of the hemisphere; same case. (*After McNutt.*)

Fig. 288. Fig. 289.

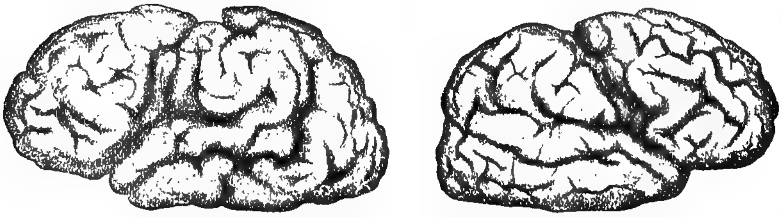


Fig. 288 left, and Fig. 289 right, hemispheres of the brain of a child, the subject of bilateral birth palsy. (*After McNutt.*)

McNutt found that the basal hemorrhage occurred in cases of head presentation, while the most intense extravasation over the cerebral hemispheres were found in cases of foot presentation. It is probable, however, that the cortical hemorrhage also occurs in cases in which the head is born first.

In older children, who present the symptoms that have been described, the lesion found is atrophy of the convolution in a certain region of the brain, generally the central convolutions, which may be small and indurated, and lie at the bottom of a considerable depression. This appearance agrees perfectly with the condition found in early cases, since the blood will be slowly removed, and only the damage to the cortex of the brain will remain.

These conditions explain, to a large extent, the symptoms observed. The facts that the thickest part of the extravasation is over the motor region, and that it is over both hemispheres, explain the distribution of the symptoms in the limbs, and their bilateral character. The frequency with which the legs are involved in considerable degree is, no doubt, due to the position of the leg centres near and in the longitudinal fissure. Experiment refers to the same region the centres for the trunk muscles, which are also often weakened. The persistence of some damaged nerve cells may explain the occurrence of the convulsions, and perhaps also of the spontaneous movements. The spasm may be connected in part with the secondary degeneration (or arrested development) of the pyramidal fibres. That in the legs resembles, in its main features, the spasm that follows a lesion of the pyramidal tracts in the spinal cord, and the differences are no doubt due to the occurrence of the disease during the stage of development. It is possible that a more complex mechanism may be concerned in producing the incoordination, such as the deficient control and balance of other cerebral centres. The extent of the damage to the cortex of the brain is a sufficient explanation of the mental defect that so often coexists.

We are not at present able to say what symptoms are due to hemorrhage about the pons and medulla. It seems reasonable to ascribe to this the weakness of the neck and the symptoms in the region of the bulbar nerves. Nevertheless, it is possible that these may be due to symmetrical lesions in the cerebral hemispheres. It is unlikely that any limb symptoms are due to injury to the anterior pyramids of the medulla, because the symptoms are similar in all cases, and are certainly generally due to the cortical lesion. That they are the same, whether or not there is weakness of the neck, is a fact that suggests the cortical origin of the latter symptom also.

DIAGNOSIS.—In a well-marked case, in which the symptoms are general, the recognition of the disease is easy if its occurrence is known. Some difficulty is presented by cases in which the symptoms are one-sided, or in which the legs only are affected. The most important distinctions from other cerebral diseases are, first, that the condition is stationary and not progressive, and secondly, that there is no history of a distinct onset of the symptoms at some period subsequent to birth. Symptoms of similar character are occasionally caused by a tumor of the brain, but then gradual development and steady increase show the existence of a progressive lesion. Similar stationary symptoms may succeed an acute lesion; there is then a clear onset to be traced, often with severe symptoms, and preceded by perfect freedom from similar symptoms. In the case of birth palsy, if the derangement of movement was first noticed at a certain period, it is generally evident that attention had not been specially given to the state of the limbs. A distinct difficulty in birth, or an unnatural presentation, can generally be ascertained; and in many cases there is a history of convulsions, etc., immediately after birth, to confirm the diagnosis. When the legs suffer chiefly, the case is often thought to be disease of the spinal cord. As already mentioned, however, slight derangement of movement in the hands can generally be detected, and it should be remembered that chronic disease of the cord in young children is almost unknown. In any case of spastic paraplegia in a child in whom there is no bone disease, a cerebral cause is always probable.

Prognosis.—As the damage to the brain is necessarily permanent, so are its effects. But when there is no considerable mental defect, the disorder of movement lessens in later childhood, because the will gradually acquires more power over the movement of the limbs, and is able, to some extent, to counteract the influence of the spasm. In all except the most severe cases the child ultimately acquires some ability to stand and walk, although it may be only at the age of six or eight. As already stated, the gait is often permanently peculiar. The mental condition also slowly improves. In every case, however, the prognosis must depend on the severity of the symptoms; what state the improvement will ultimately leave must depend upon the amount of defect, motor and mental, and upon the signs of improvement that can be discerned. It must be remembered also that, as a rule, the greater the defect, the later does improvement occur.

Treatment.—Drugs have no influence on the morbid state, and but little on the symptoms. Even in the early stage, soon after birth, it is doubtful whether any treatment can influence the process by which the brain is damaged. The meningeal hemorrhage probably occurs during birth only, and there is no evidence of any subsequent increase in the extravasation. No agents can be expected to facilitate the removal of the compressing blood, which is only slowly absorbed after permanent tissue changes have occurred in the cortex. On these the persistent symptoms depend, and their diminution is due rather to the functional education of the least damaged and undamaged structures, than on any alterations in the disease itself. Treatment, as far as it is possible, must consist in promoting this education by practiced movements of the nature of gymnastics. As soon as the child becomes able to stand, the attempt to walk is, in itself, a valuable training. Instruments sometimes enable it to stand some time before this would otherwise be possible, and they may thus distinctly accelerate improvement. It is useless, however, to employ instruments until there is enough voluntary power and control to enable the child to stand alone with their aid. Electricity in all forms is useless; its prolonged use in cases under my care has shown that has no influence over either the weakness, the spasm, or the incoördination. The treatment of convulsive attacks is similar to that of other forms of epilepsy, but the attacks are less readily influenced by drugs. Operative interference with the brain is not to be thought of; it could do good only soon after birth, and it is not likely that any child could possibly survive such a procedure as would be necessary for the removal of the clot.

SOFTENING OF THE BRAIN.

Softening of the brain* is the common result of many morbid processes. The diminution of consistence is due to the breaking up of the nerve elements; the resulting particles become separated by serum, so that a soft pulp replaces the original firm tissue. It is the characteristic change in inflammation of the brain—and not long since all forms of softening were thought to be inflammatory. We now know that the old view is erroneous, and that most cases of acute softening are due to a very different cause, to the arrest of the blood supply by occlusion of an artery.† In the part supplied by the occluded vessel, the nerve elements, deprived of nutrition, at once lose their functional powers, and quickly undergo degeneration. In many parts of the brain there is not sufficient communication between the terminal arterial twigs to permit of the reëstablishment of the circulation, and structural degeneration occurs in the delicate nerve elements with great rapidity. A similar result may follow occlusion of a vein: if the venous communications are insufficient to allow the blood to escape by some other channel, the circulation in the part is at an end, and the nerve elements break up; but the venous anastomoses are generally sufficient to prevent complete necrosis of the part. In rare instances there is softening that cannot be referred to either of these processes, but it is then always chronic, never acute. Acute Softening from (1) inflammation, (2) arterial occlusion, (3) venous occlusion, and (4) Chronic Softening. The first of these varieties will be described in the chapter on inflammation of the brain, and the last among the degenerations.

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* It should be remembered that there is a considerable difference between the popular and the medical uses of the term "softening of the brain." The general meaning attached to the term is chronic mental failure, and the disease most frequently thus designated is general paralysis of the insane. In medical use the term is restricted to the condition in which the pathological change actually exists. Some care must therefore be exercised in using the term lest it convey an erroneous impression of the future course of the disease.

† The relation of senile softening to degeneration of the arteries was first clearly recognized by Rostan in 1823, and soon afterward by Abercrombie, Carswell, and others. That embolism is a frequent cause of softening was first pointed out by Virchow (in the first volume of his "Archives"), and in this country by Kirkes, who in an admirable paper (*Medico-Chir. Trans.*, *1852*, p. 281) brought our knowledge of the subject within a measurable distance of its present position, and he did so apparently without being acquainted with the earlier paper of Virchow.

SOFTENING FROM ARTERIAL OCCLUSION.

(NECROTIC SOFTENING—ENCEPHALOMALACIA.)

Softening from arterial occlusion is not only far more common than all the other forms of softening put together, but it is one of the most frequent diseases of the brain, perhaps exceeding in frequency cerebral hemorrhage.*

General Etiology and Pathology.—Two pathological processes may cause the occlusion of an artery; a plug coming from a distance may be carried into it by the blood (embolism), or a plug may be formed in it by coagulation (thrombosis). There is an important difference in the pathological relations of these two processes. Embolism is the result of a morbid process elsewhere in the circulation, commonly in the heart. Thrombosis is usually the result of local disease of an artery of the brain, by which either its calibre is narrowed, or its inner surface is changed, so as to cause coagulation; often both these conditions coincide. The chief pathological processes that cause these changes are atheroma and syphilitic disease. A change in the constitution of the blood, rendering it prone to coagulate, and slower movement of the blood, from feebleness of the heart, often aid the effect of the arterial disease in producing thrombosis, and sometimes cause it when the arteries are free from disease.

But although the primary processes of thrombosis and embolism are thus distinct, and usually occur under very different conditions, they are often conjoined in a secondary manner. If an artery is obstructed by embolism, the stagnant blood in it may clot, extending the occlusion. If a clot forms in an artery it may be detached (as Laborde has shown) and may obstruct the vessel further on by a process which is actually embolism. But such a condition, in which the plug is formed in the vessel occluded, although not at the place of occlusion, is regarded as a form of thrombosis. Moreover, increased coagulability may cause a clot to form in the heart, and thus be a cause of embolism as well as of thrombosis *in situ*. But in spite of this occasional combination of the morbid processes, the general distinction between them is of the first importance.

EMBOLISM.

The source of the plug must be somewhere between the lungs and the brain—in the pulmonary veins, the left side of the heart, the commencement of the aorta, or the large arteries of the neck. It is possible that septic material may pass through the lungs, and lodge in the brain, but only capillaries can be obstructed by particles that can pass through the capillaries of the lungs, and any local softening of the brain thus produced is usually purulent, a cerebral abscess or minute points of suppuration. When an artery has been plugged in consequence of septicæmic processes in the general system (as uterine phlebitis), there has usually been either a pulmonary abscess or endocarditis, to furnish the embolus. In most cases the plug comes from the heart, and its source is either diseased valves, vegetations from which are separated and carried along with the blood, or a coagulum in the left auricle, a fragment of the clot being detached. It is rather more common in cases of recent endocarditis, rheumatic or "ulcerative," than in chronic disease. A fresh attack of endocarditis, in valves previously diseased, is especially likely to give rise to embolism. The plug may come from either the aortic or mitral valves, from the latter much more often than from the former. The most frequent valvular disease that causes embolism is mitral constriction, in which several conditions probably favor the occurrence. The slow flow through the narrow orifice during diastole must favor the aggregation of white corpuscles on the valve, and the quick flow during the auricular systole favors the detachment of the masses thus formed. Moreover, in this disease, there is often great dilatation of the auricle, and clot forms in the auricular appendix; fragments of this clot which are apt to be detached, or softening occurs within it, and the softened particles pass into the blood. The softened clot often contains bacterial organisms, and in ulcerative endocarditis, also, the particles detached (usually small) may carry micrococci into the cerebral arteries. Hence in both these conditions there is often plugging of minute vessels associated with signs of septic poisoning.

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* This is the common opinion, and is very likely correct, although the evidence is not conclusive. In the post-mortem room hemorrhage is the more frequent lesion, but hemorrhage is more often quickly fatal than softening is. It is probable that post-mortems in workhouses would show the cases of softening to be in considerable excess.

Outside the heart the plug comes, in rare cases, from the lungs, and may arise there from many morbid processes, growths, etc. Less rarely it comes from the aorta; when its wall is the seat of atheroma; fragments of degenerated tissue or of clot may be carried away by the blood. I have once known the plug to come from the interior of an aneurism.

Embolism occurs with equal frequency in both sexes. It is met with at all periods of life, but it is most frequent between later childhood and middle life. In the old it is less frequent, both absolutely and in comparison with thrombosis. The subjects of it have usually suffered from those diseases that are attended with endocarditis—rheumatic fever, chorea, scarlet fever, in this order of frequency. It may occur in the course of these diseases, or succeed them at any interval of time. At the time of the embolism, or soon afterward, the signs of organic heart disease are almost always to be recognized. It occurs also, although very rarely, as a result of other acute specific diseases, any one of which is occasionally attended with endocarditis.

An immediate exciting cause is rarely to be traced. I have met with one instance in which the obstruction followed immediately a severe fright, which, by exciting the action of the heart, may have effected the detachment of the plug. Increased coagulability of the blood sometimes coöperates by causing an increase of deposit on diseased valves, or the formation of a separate clot in a feeble heart. Hence embolism often occurs a week or two after childbirth. This is a point of some practical importance; conditions favoring thrombosis do not necessarily render embolism less probable.

THROMBOSIS.—As we have seen, thrombosis may be due to atheroma, to syphilitic disease of the arteries, or to a change in the blood.

Atheroma.—The larger arteries at the base of the brain are very common seats of the thickening of the inner coat, called by Virchow "Endarteritis deformans," which, when fattily degenerated, constitutes "atheroma." Opaque yellow thickenings, sometimes calcified, are the result. Only one or two of these may be present; but the change usually involves many of the larger vessels at the base, and extends for a considerable distance along the chief cerebral branches. The distribution of the degeneration may be symmetrical. It may coexist with a similar change in arteries elsewhere, or may be isolated. The exciting cause of this disease is probably the strain to which these arteries are exposed in consequence of their proximity to the heart and their deficient external support. It is not easy to explain their occasional freedom from atheroma when this is abundant elsewhere. The result of these nodular degenerations is to change the calibre of the vessel, sometimes to lessen it or to close it altogether, sometimes to enlarge it. The alteration in the lining membrane leads to the formation of clot upon it, as on a foreign body. Where the calibre of the vessel is increased the current is retarded, and this also facilitates coagulation. The smaller arteries in the cerebral substance do not suffer in the same degree, although their coats are sometimes thickened by similar changes which may even lead to the obliteration of the vessel.* The small arteries coming off from an atheromatous vessel, are often occluded by the disease at their place of origin, although the trunk is pervious.

Atheroma is common after middle life, and increases in frequency with age. It occasionally occurs before forty, chiefly in association with chronic Bright's disease, in which the arterial tension increases the strain on the vessels. Softening from atheroma shows a corresponding relation to age. It increases in frequency as life advances, and the increase continues, if due correction is made for the diminished population, to extreme senility, exhibiting in this respect a contrast to hemorrhage (see p. 776). The occurrence of atheroma, and therefore of softening, seems facilitated by chronic alcoholism. A cachectic state of the system, or some cause (such as prostrating illness or grief) that alters the constitution of the blood, and weakens the action of the heart, often concurs in producing the thrombosis.

Syphilitic disease is a common cause of softening. It is chiefly a consequence of acquired syphilis, and so occurs in adult life. About half the sufferers are between thirty and forty at the time of the attack, one-third between twenty and thirty, and most of the remainder between forty and fifty; under twenty, and over fifty it is very rare. The period after infection at which it is developed varies much, but in the majority of cases is from one to twelve years. I have met with it as early as six months and as late as nineteen years after the primary sore. Twenty-six cases in which the diagnosis was practically certain, and the period after infection could be definitely ascertained, were distributed as follows: 1 year, 3 (in none less than six months); 1–3 years, 9; –6 years, 4; –9 years, 4; –12 years, 2; –15 years, 2; –18 years, 1; –20 years, 1 (at 19 years after infection). Thus 16 cases out of 26 occurred during the first six years, 6 occurred during the second six years, and 5 during the third six years. No cases in married women are available for comparison because the date of infection cannot be ascertained; many other cases are not available because the individual has had more than one sore. The above twenty-six cases are all that could be employed for this purpose out of about a hundred of which I have notes, in which the diagnosis was reasonably certain. It has been met with in characteristic form as a result of inherited syphilis, between birth and the tenth year.

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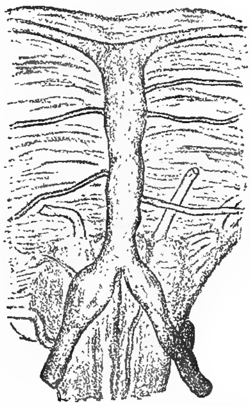
* Fatty degeneration of the cells lining the perivascular sheaths of the minute arteries is very common even in the young, but is without pathological significance.

As an isolated change (apart from massive syphilitic growths) it is almost confined to the larger arteries of the base. The vessels most commonly affected are the internal carotid, middle cerebral, vertebral, basilar and posterior cerebral. Usually several vessels are affected. The distribution of the change is commonly irregular, but sometimes it is symmetrical. The change may proceed to the obliteration of the vessel, but more frequently when the lumen is considerably narrowed a clot forms within it and suddenly arrests the circulation. The wall is thickened at circumscribed areas by a fibro-nuclear growth, which causes a nodular projection on the exterior (Fig. 290), and diminishes also the calibre of the vessel. The structure of the growth resembles that of syphilomata elsewhere. It generally begins by a nuclear proliferation between the inner coat and the elastic lamina; and in some cases it attains its chief development in this situation, the elastic lamina being pushed outward and the lumen of the vessel obliterated. The middle coat may ultimately disappear. Vessels may form in the substance of the growth, and its centre may undergo fatty degeneration.

Fig. 290.



Syphilitic disease of the basilar and vertebral arteries; thrombosis of left vertebral.

Other forms of arterial disease causing thrombosis are very rare. Occasionally pressure on an artery so narrows it that coagulation occurs. Secondary arteritis may result from inflammation of the membranes, especially from traumatic meningitis. The outer coat of the vessels becomes thickened, and degenerative changes may be set up in the middle and inner coats, and may lead to thrombosis months or years after the primary mischief. In a case I have recorded elsewhere* the patient died from softening so produced some years after the original injury, from which he had apparently quite recovered. In the course of tubercular meningitis, a tubercular infiltration of the walls of the arteries has been met with, and has produced thrombosis (Schuh). Lastly, it may be mentioned that ligature of the carotid occasionally, although rarely, causes necrotic softening of the brain. Usually the collateral circulation is sufficiently free to prevent this.

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* "Medical Ophthalmoscopy," Case 4, 2d ed., p. 270.

Blood States; Simple Thrombosis.—In certain conditions of the blood coagulation occurs with undue readiness. These states accompany childbirth, acute diseases, cancer, gout, tuberculosis, and general malnutrition at all periods of life, but especially in young children and old persons. Coinciding with disease of the arteries, it exerts a powerful influence on the occurrence of thrombosis in them. In the old this influence is often seen; the accident follows, for instance, some general illness or some depressing emotion. Arterial thrombosis may occur also from this cause when there is no arterial disease, or the clot may form in a vein or sinus. This occurs occasionally in adults, as, for instance, in the course of phthisis. It has even been known to occur in chlorosis.* Sudden hemiplegia sometimes occurs under conditions of impaired general health in young adults, in whom syphilis and heart disease can both be excluded; spontaneous thrombosis seems the most probable cause. A sudden cerebral lesion frequently occurs in children during the course of an acute specific disease, or in young children without any exciting cause; it is probably due in most cases to thrombosis in a vein or artery, but it is occasionally the result of endocarditis and embolism. On account of the uncertainty as to their exact nature, and certain clinical peculiarities, these cases are separately described.

Cardiac Weakness.—The slower the blood current the more readily does coagulation occur. Hence feebleness of the heart's action is a powerful factor in the production of thrombosis. The weakness may be due to cardiac disease, especially dilatation of the left ventricle, or it may be due to general causes, prostration from acute or chronic illness, and the like. Many of the general causes of thrombosis probably act as much or more by weakening the heart as by altering the blood.

PATHOLOGICAL ANATOMY.—*Embolism.*—The plug may consist of fibrin, usually decolorized; of vegetation, soft or firm, from a cardiac valve; sometimes of a calcareous mass from a valve of the heart or the wall of an artery; and sometimes of minute fragments resulting from the breaking down of clot or atheromatous material. In the latter case minute vessels, even capillaries, may be plugged; in the former case a large vessel is usually obstructed. Occasionally a plug, after obstructing a large vessel, breaks up, and the fragments pass on into the smaller branches. The embolus is usually arrested where the artery is narrowed by giving off a large branch, very often at a bifurcation, Fig. 291. If firm, it may retain its original shape, but if soft it may be moulded to the form of the vessel by the pressure of the blood. A coagulum usually forms beyond the plug, and extends into the distal branches, and another often forms on the cardiac side of the plug as far as the origin of a large branch. The secondary clot is red, and from it the paler embolus can usually be distinguished without difficulty (Fig. 291). Sometimes the distal part of the vessel is empty and contracted. The obstructing plug may break up and pass on into the minute branches before coagulation occurs. Hence we cannot always find an obstructed artery even when extensive softening has occurred.

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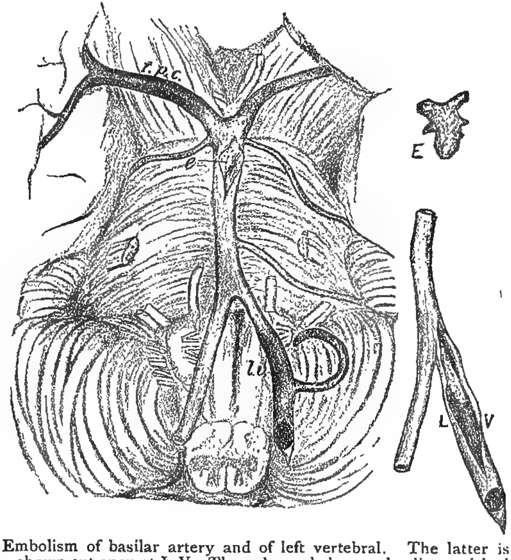
* Skerritt, Clin. Soc., March 13th, 1885.

The middle cerebral arteries and their branches are the most frequent seat of embolism, because they are the direct continuation of the carotid, and from the mode of origin of the left carotid the left middle cerebral is rather more frequently plugged than the right. But the difference between the two is less than is often stated, the proportion being as six to five.* Both middle cerebral arteries have been obstructed in rare instances.† Very seldom the internal carotid is obstructed, but the circulation in its branches is usually maintained by the circle of Willis. Next in frequency is the posterior cerebral, and then the vertebral. The left vertebral is more often plugged than the right, for the reason mentioned on p. 495, but the two posterior cerebrals are affected with equal frequency, the plug having to pass through the common basilar. The anterior cerebrals, the cerebellar arteries, and the basilar are rarely the seat of embolism. Embolism of the basilar has, indeed, been thought to be impossible,‡ but it certainly occurs, as Fig. 291 shows. A plug too large to be arrested in the vertebral, and too small to enter the posterior cerebral, must of necessity be arrested in the anterior extremity of the basilar, and I have recorded an example of this.§ Rarest of all is embolism of the cerebellar arteries, doubtless in consequence of the angle at which they arise from the larger trunks. More than one artery may be plugged, but usually at different times, rarely at the same time. When the plug comes from a septic source, as in ulcerative endocarditis, a secondary arteritis may occur at the ob-

FIG. 291.



Embolism of basilar artery and of left vertebral. The latter is shown cut open at L V. The paler embolus can be distinguished from the secondary clot behind it. E is the plug *e* from the extremity of the basilar, moulded by pressure into the shape of the vessel, with projections corresponding to the branches. There is a secondary thrombus in the left post. cerebral, *l. p. c.*

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* Gelpke, left 64 (49 per cent.), right 54 (41 per cent.), *Arch. der Heilkunde, 1875.*

† As in a case of ulcerative endocarditis recorded by Carrington, *Path. Trans.*, vol. xxxv, 1884, p. 108.

‡ Nothnagel, *Topische Diagnose, etc., 1879*, p. 105. Leyden, *Zeitsch. f. kl. Med.*, Bd. v, 1882, p. 175.

§ *Brain*, vol. i, 1882.

structed part, and sometimes the inflammation may extend to the neighboring structure. Occasionally the artery behind the plug, or when the obstruction is imperfect, is dilated into an aneurism ; the walls, changed by the inflammation and rendered inelastic, yield before the pressure of the blood.

Thrombosis from Atheroma.—Many arteries of the base are usually affected by atheroma, and thrombosis may occur in any of them, but is most common in the internal carotid, middle cerebral, basilar, vertebral and posterior cerebrals. It may occur in the branches that come off from the diseased and thickened part of the vessel, although the trunk remains pervious. The branches given off by the basilar to the pons may be thus obliterated, and softening of the pons may occur. In the same way the branches of the middle cerebral to the central ganglia may become closed. When a clot forms and obstructs the vessel, it usually extends on into its distal branches. This is true of the internal carotid, thrombosis in which usually extends into the middle and anterior cerebrals, and even to the ophthalmic artery. In this respect thrombosis of the internal carotid presents a contrast to embolism, and has much more serious consequences. A clot in the vertebral may extend into the basilar, or the circulation in this may be maintained by the other vertebral.

A clot usually obstructs the vessel where it is formed, but occasionally, as already stated, a clot that has formed on the side of a vessel may become detached and may be arrested at a narrower part of the vessel a little further on (Laborde). This mechanism, although probable, can rarely be proved. When the clot is in the place at which it was originally formed, it is adherent to the wall, pale and often laminated. In a recent case the secondary coagulum that forms in the distal part of the artery is usually red and adherent. After a time it undergoes changes, may become organized, or degenerate, and even calcify. The artery may become transformed into a fibrous cord. Very rarely a thrombus breaks down, or is pushed on before distal clotting occurs, and the circulation is thus partially or entirely reëstablished.

Syphilitic Disease.—The disease narrows the cavity of the vessel to a greater extent than does atheroma, and hence thrombosis plays a smaller but still effective part in producing the final occlusion. The branches coming off from the vessel are obstructed, without occlusion of the trunk, even more frequently than in atheroma. Otherwise, the two conditions produce very similar pathological effects.

Anatomical Consequences.—The first effect of arterial obstruction is to cause the arrest of the arterial blood supply to the region supplied. There is generally arterial anæmia ; sometimes the capillaries may become distended with blood from the veins, and may give way in places, so that the area may be infiltrated with hemorrhagic points. For the first twenty-four hours there is little change in the consistence of the parts, and if the vessels are empty, it is difficult to distinguish the affected area from the rest of the brain, the only difference being its paler tint. After the first twenty-four or thirty-six hours the consistence of the tissue rapidly lessens ; the nerve elements break down, and their fragments are separated by effused serum, so that an area of softening results. The serum comes, no doubt, partly from the blood, and partly from the lymphatic sheaths, into which the lymphatic fluid must collect if the vessels collapse. The tint of the softened area depends on the amount of blood within it, and may be white, yellow, or red, from the beginning. The blood corpuscles escape into the softening tissue, partly, perhaps, by diapedesis, and partly by the rupture of the capillaries, the walls of which undergo degeneration. The red color changes, in the course of a few weeks, to yellow, by transformation of the blood pigment. Thus, three forms of softening are distinguished: white, red, and yellow, but these do not correspond to pathological varieties. In all forms the process of breaking of the nerve elements proceeds to the formation of fine fatty granules, in part aggregated into "granule corpuscles." These constitute a distinction from post-mortem softening, in which the segmentation of the nerve tubes does not form such small fragments, and granule corpuscles are absent.

Red softening is found chiefly in the gray substance, where the vessels are numerous, especially in the cortex and central ganglia. The tint varies; the red color is usually punctiform, or mingled with yellow and white. If the extravasations are large and numerous, "capillary apoplexy" results. The diminution of consistence is usually moderate. According to the amount of effusion of serum and blood there is swelling, and the diseased area may project above the cut surface. Inflammatory changes result from the vascular distention, and in proportion to these, increase in the nuclei of the neuroglia is found. From this cause, and from the migration of white corpuscles, pus-like cells appear. The vessels are dilated, and may present a moniliform appearance. Their perivascular sheaths are often distended with blood. Commencing degeneration of the effused blood may cause a brown tint.

Yellow softening results from red softening, by degenerative changes in the blood effused. It has a similar seat, being frequently met with in the convolutions, where it constitutes the *plaques jaunes* of the French. Its consistence is usually slight, its aspect granular. The color depends on the presence of minute pigment granules, diffused coloring matter and hæmatoidin crystals.

White softening has the tint of the normal cerebral substance. In consistence it varies; it may be only a little below that of the brain tissue, or it may be diffluent. Its aspect is uniform, or white flakes are scattered through it. The limits are usually gradual. Under the microscope it presents the detritus of nerve elements, a few nuclei from the connective tissue, granule corpuscles, and, ultimately, corpora amylacea. White softening is chiefly found in the white substance of the hemispheres. It occasionally has a gangrenous odor, and then may be found in the white or in the gray substance, probably resulting from the obstruction of capillaries by septic material.

The process of softening is attended with inflammatory changes around the affected area, as in other cases of necrosis. This does not usually leave any visible alterations, but it is probable that the nerve elements often suffer permanently in their nutrition and function from this cause, and it is important, in regard to the symptoms, to remember that the destroyed area is surrounded by a zone of slighter damage.

Ultimate Changes.—White and yellow softening may remain for years unchanged. Sometimes the alterations in the elements of the neuroglia and the extravasated white corpuscles result in the formation of a considerable quantity of connective tissue, consisting of fine fibre cells and fibres, most abundant in the margins of the softened area, which become firm and dense, while trabeculæ of connective tissue cross the cavity. After a time the fluid may be absorbed, the fat removed, and a sort of cicatrix results. In other cases the walls alone are thus altered, the solid particles are removed from the softened tissue, and a cyst is formed. The outer portion of the cyst or cicatrix may be limited by a zone of dilated blood vessels.

Seats of Softening.—There is no part of the brain in which softening has not been found, but its most frequent seats are the cortex, the corpus striatum, and the optic thalamus. It is also frequently found in the pons, and occasionally in the medulla and cerebellum. An example of softening of the pons is shown in Fig. 229, p. 506. Its occurrence, position and characters depend on the distribution of the vessels. The small arteries of the corpus striatum and optic thalamus are "terminal arteries," having only capillary communications with other vessels. The arteries to the surface of the brain are also sometimes terminal, but sometimes possess arteriole anastomoses with other branches. Hence obstruction in the central arteries leads invariably to softening. Obstruction in the superficial arteries also often causes softening, which involves the gray substance of the convolutions and some of the subjacent white centre to which the vessels penetrate; but frequently the anastomoses of the superficial vessels are so free that softening does not result.* Hence obstruction of a main trunk (as the middle cerebral) may lead to softening of the central region (corpus striatum), while the convolutions escape, or both may suffer. Some parts of the cortex escape more frequently than others; the convolutions about the fissure of Sylvius are often destroyed by obstruction of the middle cerebral when the higher regions of the cortex are intact (see, for instance, the softening figured on p. 463).

The changes thus described are met with in all forms of vascular obstruction. The causes that determine the amount of blood in the softened part are not all understood, but the amount seems to be larger in the young than in the old, and, perhaps, in embolism than in thrombosis. Cohnheim and Litten have proved that embolism in any organ is usually attended with distention of the veins and capillaries in the infarcted area, and with abundant effusion of serum. It is surprising that such distention is not more constant in the brain.†

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* As an illustration of the free arterial communication that often exists in the cortex, it may be mentioned that Prof. Thane has informed me that attempts to inject separately the arteries supplying the cortex fail more often than they succeed, on account of the readiness with which the injection passes from one arterial region to another.

† An extraordinary case has been recorded by Toterick, in which, two days after the

Symptoms.—The symptoms produced by the occlusion of an artery, and consequent necrotic softening of the brain, resemble those of cerebral hemorrhage in being of two classes, one transient, the result of the sudden interference with the functions of the brain, the other more or less permanent, the result of the destruction of the nerve elements in the affected area. The permanent symptoms depend on the position of the lesion; the symptoms that attend the onset vary according not only to the seat, but also to the nature of the vascular occlusion.

Premonitory symptoms are rare in embolism. When present they are not true prodromata, but consist in slighter attacks of the same character, due to the obstruction of small vessels by small emboli, or else they are really the first symptoms of the onset when the obstruction is incomplete and the occlusion is gradual. There is no persistent preceding headache or other indication of encephalic disease. Until the plug obstructs the vessel, the brain is in a normal state. In thrombosis from atheroma, on the other hand, premonitory symptoms are frequent, being present in the majority of cases. They depend on the interference with the supply of blood, due to the disease of the blood vessels. They may exist for months before the onset or only for a few hours. The most common are dull general headache, giddiness, tingling, "numbness," slight weakness in one-half of the body, sometimes limited to a single limb, and often, but not always, corresponding in seat to the subsequent paralysis; less commonly there is defective articulation, or some mental change, failure of memory, or irritability due to the general malnutrition of the brain, that is produced by widespread arterial disease. In syphilitic disease premonitory symptoms are also frequent. In some cases they resemble those just described, but more severe general cerebral symptoms are both common and characteristic. Headache is the most frequent, and is often severe, usually general, and may be worse at night. It may exist for weeks or only for a day or two before the onset. Another occasional premonitory symptom is giddiness. Sometimes there is considerable mental dullness or a somnolent condition, lasting for weeks.

The onset of cerebral softening is attended by loss of consciousness in a considerable number of cases, although this is decidedly less frequent than in hemorrhage. The obstruction of a large vessel, however produced, frequently entails a distinct apoplectic attack. The obstruction of a small branch, causing only a small spot of softening, often causes no loss of consciousness. For lesions of equal size, the more sudden the obstruction, the more pronounced is the apoplectic seizure. This is well seen in cases of the same nature. In embolism a gradual onset, occupying many hours, is rare, but when it occurs, there is no unconsciousness; but with a sudden onset

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occurrence of embolism of the right middle cerebral artery at the place of division, hemorrhage occurred outside the right corpus striatum, which must have come from a branch beyond the obstruction. It was fatal, a week later, by rupture into the ventricles, perhaps facilitated by softening consequent on the embolism. (*Lancet*, *1883*, i, 1042.) An aneurism may have existed before the embolism.

loss of consciousness is very frequent. In syphilitic disease, on the other hand, consciousness is more frequently preserved than lost. The contrast between these two conditions is more marked than between embolism and senile softening, because the age of the patients renders loss of consciousness more readily produced in the latter. When there is well-marked apoplexy, the loss of consciousness may be the first symptom; but, rather more frequently than in hemorrhage, focal symptoms, especially hemiplegia, come on before the coma. This is especially the case in atheromatous thrombosis. The apoplectic condition itself presents no characteristic feature; it resembles closely that produced by hemorrhage. The turgid face and strongly pulsating carotids are, as a rule, absent, but these are not very common in hemorrhage. As a rule, the degree of coma is slighter, and its duration shorter, than in hemorrhage, but when a large vessel is plugged, the coma may be as intense and as prolonged, the resolution of the limbs as perfect, and the interference with breathing as marked, as in a severe cerebral hemorrhage. It may last for several days, and end in death or in recovery. Coma lasting for five or six days may pass away when it is due to softening, but usually ends in death, if due to hemorrhage.

The mechanism by which apoplexy is produced by vascular obstruction can scarcely be the same as in hemorrhage, since the important element of pressure is, if not absent, at least trifling in degree and late in time, due only to the œdematous swelling which comes on after the onset. Attempts have been made to explain the unconsciousness by the disturbance of the blood pressure. It has been ascribed, for instance, to the increase of blood pressure in the other branches of the same vessel (Heubner), or to the pressure being suddenly diminished in the area of the infarction by the arrest of the blood tension within it, and a consequent abnormal pressure in the vicinity (Wernicke). The latter mechanism is hypothetical and doubtful, and neither seems adequate. Moreover, the fact that extensive softening is sometimes unattended with apoplexy, even when quite sudden in onset, is difficult to reconcile with either explanation. On the whole, it seems most probable that sudden cessation of nutrition in the affected area is equivalent to a traumatic irritation, and that the loss of consciousness is chiefly due to an inhibition of the highest centres, similar to that which, in the spinal centres, causes relaxation of the limbs.

Convulsion frequently accompanies the onset in embolism. It may be general, and irrespective of the artery plugged; sometimes it begins locally in the part afterward most paralyzed, and is then generally due to embolism of the middle cerebral, affecting the motor cortex. In atheromatous softening, and in syphilitic disease, initial convulsions are much more rare.

Vomiting is not more common in any form of softening than in cerebral hemorrhage. It is most frequent in obstruction of the basilar.

Delirium may take the place of an apoplectic seizure, in either embolism or thrombosis. In atheromatous softening, brief initial loss of consciousness is often succeeded by quiet delirium lasting for several days or even weeks.

When there is no loss of consciousness, the onset is usually marked by sudden headache, giddiness, or incoherence, to which are quickly added the paralytic or other symptoms due to the destructive lesion. The loss of power is often accompanied by tingling in the affected side.

Focal Symptoms.—In rare instances focal symptoms are absent, the lesion being so placed as to cause none. In most cases, focal symptoms may be recognized during the period of unconsciousness, similar to those produced by hemorrhage, unilateral atony of muscles, loss of reflex action, inequality of face, and conjugate deviation of head and eyes. Hemiplegia is the most common symptom, because the middle cerebral is the artery most often plugged, and aphasia accompanies it when the left artery is affected. Bilateral palsy may be due to bilateral obstruction, or to thrombosis of the basilar, causing softening of the pons.

The fall of temperature that attends the onset of hemorrhage is less marked in softening, rarely exceeding half a degree, and is very often absent. A rise usually occurs in ten or twelve hours, or may be deferred, even to the third or fourth day. The period of inflammatory reaction is more pronounced in softening than in hemorrhage. It occurs, whatever be the cause of the softening, but is most considerable in the old, whose brains are generally ill-nourished, and in embolism, especially when the plug comes from a septic source, such as ulcerative or septicæmic endocarditis. The previous elevation of temperature in these cases increases considerably after the occurrence of embolism. In occlusion of the basilar artery there may be hyperpyrexia, as in hemorrhage into the pons, but less rapidly developed, and it may be preceded by a greater depression. The febrile disturbance is often considerable and prolonged; the temperature is one or two degrees higher on the paralyzed side; sloughing of the skin is common; and inflammation of the joints is met with less rarely than in hemorrhage. The headache that accompanies the secondary inflammation is usually more considerable than in hemorrhage, and both delirium and convulsions are more frequent. Optic neuritis, almost unknown from hemorrhage, is also unknown as a result of senile (atheromatous) softening and of softening from syphilitic disease of the arteries.[*] If it coexists with these it is as the result of associated causes, as Bright's disease in the former, or a syphilitic tumor in the latter case. But it is met with occasionally in embolism when the plug comes from an inflamed endocardium, [‡] and it is apparently the consequence of the irritative character of the secondary inflammation. In ulcerative endocarditis, another ophthalmoscopic change may be met with;

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[*] An exception is a case of syphilitic disease of the pons recorded by Leyden (*Zeitschrift f. kl. Med.*, Bd. v), but the symptom probably depended on some coincident influence, since it is, as a rule, absent in obstruction of the basilar as in that of other arteries. In the rare cases, of which I have seen two, in which optic neuritis coincides with sudden hemiplegia due to syphilis, there has been other evidence of gummata or chronic meningitis, which are occasionally associated with the arterial lesion.

[†] Such cases have been recorded by Broadbent and Stephen Mackenzie, and another has come under my own observation.

rounded hemorrhages, with white centres, scattered over the retina. They are the result of the obstruction of minute vessels by septic plugs. Similar hemorrhages may be found in the pia mater.

Otherwise the local symptoms resemble closely those that are produced by hemorrhage, and consist in motor and sensory paralysis, etc., according to the seat of the softening. As in hemorrhage, the initial symptoms are more extensive than those that persist, and those that pass away during the first two weeks must be regarded as indirect in origin. We cannot explain them as pressure effects, but they are no doubt the result partly of inhibition and partly of the interference with function from slight damage that is wider than the area of actual destruction.

The chief differences between the local symptoms in softening and hemorrhage are the following. In softening, aphasia is rather more common on account of the frequency with which the middle cerebral artery is obstructed. Partial hemiplegia, "monoplegia," of arm, or face and arm, rarely of the leg, is also more common, because softening affects the cortex much more frequently than hemorrhage does, and partial hemiplegia is usually due to a cortical lesion. Moreover, the effect of softening is more restricted than is that of hemorrhage, on account of the absence of pressure on adjacent parts. Recurring convulsions are also far more frequent after softening than after hemorrhage. This is probably due chiefly to the more frequent affection of the cortex by softening; partly, perhaps, to the less strict limitation of structural changes. In the subsequent chronic stage, disorders of movement, choreoid, athetosis, etc., are far more common after softening than after hemorrhage, whatever be the explanation of the fact. Mental failure, loss of memory, emotional mobility, are also rather greater in degree during the chronic stage of softening, but the difference is less than is commonly imagined, and is in part related to three facts: (1) foci of softening are often more numerous than those of hemorrhage; (2) softening from atheroma is often accompanied by disease of other vessels interfering widely with the nutrition of the brain; (3) this is also the case in syphilitic disease, in which, moreover, there is often a marked tendency to nerve degeneration.

Another point of difference between softening and hemorrhage is that an entire absence of focal symptoms is rather more common in softening. A lesion of either kind, away from the motor and sensory tracts, causes no persistent symptoms, but if the lesion is merely near these parts and does not actually involve them, symptoms are more frequently absent in softening than in hemorrhage, no doubt on account of the pressure exerted by the latter. This is often seen in lesions of the central ganglia. It is rare for a hemorrhage limited to the caudate or lenticular nucleus not to cause hemiplegia by the pressure it exerts on the internal capsule, whereas it is not uncommon to meet with an extensive softening of one of these nuclei without any focal symptoms, past or present.

Bilateral obstruction of large vessels is rarely simultaneous, but sometimes occurs in quick succession, and may then cause bilateral symptoms very closely resembling those produced by ventricular hemorrhage. The initial apoplexy may or may not have passed away when the coma recurs or suddenly deepens, and quickly becomes intense, with general relaxation or rigidity of muscles. The patient may die in the course of a few hours. When the vessel occluded is less important, bilateral symptoms result, resembling those produced by softening of the pons. In rare cases of thrombosis or embolism, the corresponding areas in the two hemispheres have been affected, causing symmetrical paralysis of partial extent. Thus an affection of the lower part of each motor cortex has caused not only total loss of speech, but paralysis of the lips, tongue and throat, closely resembling that produced by a lesion of the medulla oblongata.

Complications.—In some forms of softening the symptoms produced by the lesion may be accompanied by others that are produced by associated morbid processes. Thus, syphilitic arterial disease is sometimes associated with syphilitic meningitis or with a syphilitic tumor. In ulcerative endocarditis, the circulation of septic material in the blood often causes general fever with rigors, headache and delirium, enlargement and tenderness of the spleen, or hæmaturia from renal embolism. Delirium may occur without softening, in consequence of the blood state or of capillary obstructions in the brain or meninges; and it is not therefore surprising to find that it often accompanies the symptoms of an actual softening. In all cases of embolism the indications of a similar process elsewhere are frequent. An interesting complication is embolism of the central artery of the retina. It usually occurs at some other time than the cerebral embolism, but I have recorded one case in which the two accidents were simultaneous.*

OBSTRUCTION OF PARTICULAR ARTERIES.—The distribution of the several arteries which determine the seat of softening and the symptoms produced has been already described (see p. 495).

Internal Carotid.—The simplest form of obstruction is that produced by ligature of the common carotid. This may cause no symptoms, or only transient hemiplegia. Sometimes there is severe hemiplegia, ending in death at the end of a week or ten days. In the former case the communications of the circle of Willis suffice to carry on the circulation in the hemisphere, the blood coming from the other carotid by the anterior communicating artery, and from the basilar by the posterior communicating. After a time these vessels are found considerably dilated. Persistent hemiplegia is usually the consequence of these vessels being unusually small, so that enough blood is not conveyed to prevent necrotic softening; it may be found post-mortem in the greater part of the hemisphere except in the region supplied by the posterior cerebral. In the cases in which the communicating arteries are absent this result necessarily follows. In embolism of the internal carotid the symptoms depend on the place at which the plug is arrested. If this is at the curve in the cavernous sinus the symptoms resemble those produced by ligature of the vessel, and the circulation is usually maintained in the part above the plug by the communicating arteries, so that softening does not result, while the connection between the ophthalmic and facial artery helps to maintain the circulation in the former. If the plug is arrested at the division of the internal carotid, the resulting symptoms are the same as in obstruction of the middle cerebral artery; the anterior communicating conveys a supply of blood to the anterior cerebral. Thrombosis in the internal carotid consequent on atheroma may cause the same symptoms as embolism, but more frequently produces grave results, because there is a greater tendency for the thrombosis to spread, since the disease of the walls of the artery is usually extensive. Thus a clot may spread into the anterior and middle cerebrals, and along the former it may pass beyond the communicating artery, producing softening of the anterior two-thirds of the hemisphere, only the occipital lobe and the lower and inner portions of the temporal lobe escaping. It may spread also into the ophthalmic artery, producing (as I have seen) a necrosis of the eyeball, so that the sclerotic after death was rotten and discolored. The symptoms of this extensive thrombosis are necessarily severe; hemiplegia with deepening coma and death at the end of (usually) four or five days. The opthalmoscopic appearances due to the thrombosis of the ophthalmic artery are not known, but they probably resemble that produced by embolism of the central artery, perhaps with rapid opacity of the media. It is very important in every case of apoplexy that the eyes should be daily examined, because the obstruction of the ophthalmic artery develops gradually during coma and subjective indications of the accident are absent.

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* "Medical Ophthalmoscopy," Case 47, 2d ed., p. 332.

The *anterior cerebral* is rarely obstructed except by plugging of the extremity of the internal carotid, because, on account of its direction at right angles to the carotid, an embolus very seldom enters it. Obstruction of the first part of the vessel may cause a small spot of softening in the head of the caudate nucleus, but not elsewhere in the region supplied unless the anterior communicating artery is absent, or a thrombus extends beyond its junction. Moreover, a collateral circulation from the branches of the middle cerebral may sometimes aid in maintaining the nutrition of the cortex. Hence softenings in the cortical region supplied by this vessel are rare, except in association with obstruction of the middle cerebral. Softening of the olfactory bulb and adjacent part of the orbital lobule has, however, been known to result from an embolism of the first branch of the artery. Very rarely softening is limited to the paracentral region, or to the precuneus. Except the loss of smell that results from disease of the olfactory nerve, the symptoms of softening in the region of the anterior cerebral are not distinctive. Theoretically a monoplegia affecting the leg only should result from softening of the marginal convolution and the paracentral lobule, but I do not know of an observed case.

Middle Cerebral.—Since this artery supplies the motor cortex and the motor path through the ganglia, its obstruction, partial or complete, usually gives rise to paralysis. One or more of its four cortical branches (see Fig. 217, p. 499) may be occluded, or all may be obstructed by a plug at the point of division of the artery at the island of Reil. Occlusion of the branches may cause softening in the area supplied by them, shown in Fig.

218. That of the first produces softening of the third frontal and, if on the left side, aphasia; of the second or third, softening of the ascending frontal or ascending parietal convolution and hemiplegia, sometimes partial when the softening is incomplete; of the fourth, softening about the posterior limb of the fissure of Sylvius, and if on the left side, there is sensory aphasia, defective perception of words, with corresponding impairment of speech. When all the cortical branches are obstructed the softening extends over all the area supplied, although usually in varying degree in different parts, in consequence of partial anastomoses, and the upper region of the hemisphere may escape (see Fig. 185, p. 463). The effect is a severe form of hemiplegia, with impairment of sensibility for a time, and sometimes affection of the special senses, and ptosis on the opposite side. Partial obstruction of the deep central branches, from the first part of the artery, either by embolism, disease of the wall at their origin, or thrombosis, causes softening in the lenticular nucleus, caudate nucleus, and anterior part of the optic thalamus, very variable in extent; when considerable, the internal capsule always suffers and hemiplegia results. Complete obstruction of the middle cerebral near its origin always causes softening in the central ganglia and hemiplegia, usually also softening of the cortex, but occasionally the cortex escapes wholly or partially, in consequence of the freedom of the anastomoses, although the central ganglia and capsule are involved (see p. 816).

Posterior Cerebral.—The rarity of softening from obstruction of the posterior cerebral, due to the freedom of its anastomoses, renders our knowledge of the symptoms produced much less definite than in the case of the middle cerebral. They are for the most part sensory; there may be hemianæsthesia from softening of the tegmentum of the crus, or internal capsule, or hemianopia from softening of the occipital lobe. Complete but transient loss of sight has also attended embolism of one posterior cerebral; it is probably inhibitory in origin. The symptoms of limited softenings of the inner and lower part of the temporal lobe are not known.

Basilar Artery.—The symptoms present considerable variations according to the seat of obstruction, whether in the middle, lower, or upper portions, or whether there is merely occlusion of the arteries that come off from it, in consequence of disease of the wall, or of incomplete embolism. In the obstruction of the transverse arteries, the softening produced may be unilateral, often small in extent, near the middle line or in the outer part of the pons, according as median or radicular branches are obstructed (see p. 500). Hemiplegia usually results, which may be alternate of limbs and cranial nerves, or may resemble that produced by a lesion of the corpus striatum, according to the seat of the disease (see p. 724). In some cases the occlusion of different branches takes place successively, and the patient may have many sudden seizures, with various palsy of limbs and cranial nerves. In occlusion of the whole basilar the limbs of both sides are usually affected, sometimes at the same time, sometimes successively in the course of a few days. The limbs on one side, for instance, may become weak and improve; a day or two afterwards the other side becomes weak, and then there is again loss of power in the side first affected. The affection of the facial or fifth nerves may present the same variations, and often does not correspond to the affection of the limbs. There is usually considerable impairment of articulation and of swallowing. Loss of the conjugate movement of the eyes, from palsy of external and internal recti, is occasionally observed, with or without palsy of the facial nerve on the side of the affected external rectus (see p. 610). This, however, less frequently results from softening than from tumor. When the obstruction involves the upper part of the vessel, as is usually the case in embolism, there are usually ocular symptoms other than the conjugate deviation, ptosis, contracted or dilated pupils, with loss of reflex action to light, sometimes transient. There is occasionally paralysis of the whole or part of one third nerve, or even of both. Less common is hemianopia, from extension of clot along one posterior cerebral beyond the origin of the posterior communicating artery. Bilateral loss of sight, theoretically possible, is excessively rare. Obstruction of the lower extremity of the basilar is not common, except as the result of the extension of a clot from one of the vertebral arteries. The onset of obstruction of the basilar is usually attended with a distinct apoplectic seizure, usually transient; it often recurs, and gradually deepens. The patient may lie for some days in a state of incomplete coma, can be roused to open his eyes, but does not speak. Convulsions occasionally attend the onset, but are on the whole not frequent; they may be general or partial; one limb or one side of the face may escape. Rigidity or clonic spasm in the legs or arms is much less common than in hemorrhage into the pons. Undue frequency and irregularity of the heart's action, and impaired respiration (labored or sighing, of Cheyne-Stokes rhythm, or with irregular variations) are very common before death, and may occur from the commencement, when the obstruction involves the lower extremity of the basilar, from which the arterioles arise that supply the pneumogastric centre. If there is an apoplectiform onset there may be an initial fall of temperature, much greater than is met with in the occlusion of other vessels, and resembling that met with in cerebral hemorrhage. In one case the rectal temperature fell to 95°*. Toward the end there is often the rise of temperature so common in lesions of the pons. In one recorded case the rise reached 109° before death and 109.5° after death. The duration of life is usually from three to seven days; occasionally death occurs within twenty-four hours. In the case mentioned above, in which the temperature fell to 95°, death occurred in five and a half hours. Rarely life is prolonged for a fortnight. Recovery probably occurs in some cases of syphilitic diseases, but not in atheromatous thrombosis, and perhaps not in embolism.

Vertebral Artery.—The frequent anomalies in the arrangement of the arteries of the medulla render the symptoms of thrombosis of the vertebral artery very variable. It usually supplies the "bulbar" nuclei, hypoglossal, accessory glosso-pharyngeal, in part directly, and in part by the anterior spinal, and through the latter it supplies the anterior pyramids and also the lower part of the medulla. The typical effect of its occlusion is one-sided bulbar paralysis—lips, tongue, palate, and larynx, and paralysis of the limbs on the same side.* The hemiplegia is usually transient, is often accompanied by tingling, and sometimes by anæsthesia. It is transient because the anterior spinal receives blood from the other vertebral artery and the supply to the lower part of the medulla is renewed. The paralysis of the bulbar nerves, the nuclei of which are supplied in part from the anterior spinal, is usually imperfect, but the impairment of swallowing and of articulation is always great. The median arterioles may come altogether from one anterior spinal (when there are two), and if this is occluded the bulbar paralysis is bilateral, and may resemble perfectly that from chronic degenerative disease. If, as is often the case, the clot extends up into the commencement of the basilar, the arrest of the blood supply to the respiratory centre causes rapid death. There may be softening of the posterior part of the cerebellum, the occurrence and extent of which is influenced by the freedom of its anastomoses.

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* Bastian, Clin. Soc., March 13th, 1885.

Cerebellar Arteries.—These vessels are rarely the seat of isolated obstruction, and although softening may result from occlusion of the arteries from which the cerebellar vessels spring, the softening is always less extensive than the area of distribution of the affected vessel, on account of the freedom of the anastomoses,† and any symptoms that are produced are lost in the more serious disturbance that results from the damage to the pons and medulla. Softening is most common in the region supplied by the posterior cerebral, the obstruction of which is usually secondary to occlusion of the vertebral. Incoördination of movement has been observed in one or two cases of this kind, and may have been due to the lesion of the cerebellum. Occasionally an isolated area of softening is found in the middle of one hemisphere, without any symptoms during life that could be ascribed to it.

Diagnosis.—The diagnosis of obstructive softening rests on the symptoms of a sudden cerebral lesion, occurring in the conditions that are known to be causes of vascular occlusion. The characters of the attack have less diagnostic significance than the causal indications. The chief difficulty is the distinction of atheromatous obstruction from hemorrhage. During the first forty years of life the question seldom arises. Hemorrhage would only be suspected if the apoplectic attack were one of considerable severity, and the coma prolonged. It must be remembered that heart disease, by producing aneurism, is a cause of hemorrhage even during youth, and hemorrhage should be suspected if a patient suffering from heart disease is seized with apoplexy followed by deepening coma. Such symptoms are rarely caused by embolism, unless the internal carotid is obstructed, and this can be excluded by an ophthalmoscopic examination. But in most cases during the first half of life the apoplectiform symptoms are generally moderate or slight in degree, or are absent, and one of the two common causes of vascular obstruction can be traced—heart disease or syphilis. Usually one is present and the other absent, and the diagnosis can be made with a high degree of probability. If, however both are present—if the patient is suffering from heart disease, and has had syphilis, the diagnosis is much more difficult, and rests, first, on the evidence of activity of one or the other of these causes, and secondly, on the presence of any other symptoms that may be due to syphilitic disease of the brain. If there is considerable valvular mischief, if there has been recent endocarditis, and especially if evidence of embolism elsewhere can be discovered (as an enlarged and tender spleen, or sudden hæmaturia, or embolism of the central artery of the retina), the probability of cerebral embolism is much increased. On the other hand, persistent headache for some days or weeks before the onset, transient attacks of tingling, numbness or weakness in the limbs afterward paralyzed, or a dull somnolent state before the attack, suggests syphilitic disease of the arteries. Evidence of present activity of the syphilis adds weight to the other symptoms, but is not often forthcoming, since the period at which arterial disease occurs is later than that at which the obtrusive manifestations of constitutional syphilis are common. Apparent quiescence of the constitutional disease is thus no contraindication, nor is past antisyphilitic treatment, however thorough. As an instance of the difficulty the diagnosis sometimes presents I may mention the case of a man who had an attack of hemiplegia at forty-five. He had a loud aortic regurgitant murmur, and it seemed probable that the hemiplegia was due to embolism, with which the onset was consistent. But he had had severe headache for some weeks before the attack. Shortly afterward, a node appeared on his skull, and it was ascertained that in youth he had had syphilis. This fact gave significance to the headache, and made it far more probable that the vascular obstruction was due to syphilitic disease than to embolism.

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* The limbs were affected on the same side as the thrombosis in most recorded cases, which seems to show that it is due to the anæmia below the decussation of the pyramids.

† The whole cerebellum can be injected from any one of its arteries, even if the basilar is tied (*Duret*).

A year or more after the onset, the absence of heart disease does not exclude embolism, or even render it less improbable, if the hemiplegia occurred during or soon after an illness known to cause endocarditis, especially rheumatism or chorea. A girl had an attack of chorea, attended by endocarditis, and mitral regurgitation. While still ill with the chorea she had an attack of hemiplegia, no doubt due to embolism. Some years later, the hemiplegia persisting, her heart presented no abnormal sign in sound or impulse.* Apart from a cause of endocarditis, the absence of disease of the heart renders embolism most unlikely.

During the degenerative period of life, after forty-five, the diagnosis of softening presents much greater difficulties. The help afforded by causal indications is more limited. The conditions associated with hemorrhage and softening are to a large extent the same. Atheróma, the chief cause of softening, is met with in four-fifths of the cases of hemorrhage. The state of the heart is of more significance than that of the vessels. A strongly-acting, hypertrophied heart suggests hemorrhage; a feeble, irregular heart, softening. Cancer, phthisis, a general illness or depressing emotion before the onset, are also in favor of softening. Other diagnostic indications are drawn from the characters of the onset, and these have been already described in the chapter on hemorrhage. It must be remembered that the diagnosis is always a matter of probability, sometimes high and sometimes low.

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* Yet several years later she presented signs of mitral constriction, a most significant fact, although not strictly relevant to the present subject.

The diagnosis is occasionally rendered still more complicated by the co-existence of heart disease, or of syphilis, with arterial degeneration, but the additional complexity relates to the cause of softening rather than to the distinction from hemorrhage. The latter is indeed facilitated since the presence of more than one condition capable of causing softening increases the probability, *cæteris paribus*, that the lesion is not hemorrhage. But the question which of several causes of vascular obstruction has been effective in a given case can only be answered by a careful consideration of the apparent activity of the several causal influences and by the symptoms at the onset as already described. It must be remembered that atheroma and syphilitic disease have more symptoms in common with each other than either has with embolism, since in each there may be interference with the circulation, and often, therefore, slight symptoms before the onset. In some cases a correct diagnosis is impossible. A woman, aged sixty, was brought unconscious to the hospital with symptoms that pointed to obstruction in the basilar artery. Her vessels were highly degenerated and she had a loud murmur of mitral constriction. Of the two lesions that seemed possible, atheromatous thrombosis and embolism, the rarity of the latter in the basilar artery made the former rather more likely. She died, and thrombosis of the basilar was found, as anticipated, but it was due, not to atheroma, but to intense and characteristic syphilitic disease of the vessel, which there was nothing during life to suggest.

Prognosis.—The prospect of recovery from the initial symptoms depends on their severity and duration, on the previous occurrence of similar attacks, on the artery affected, and on the nature of the cause. Intensity of apoplexy is a graver indication than its duration. In this respect softening differs from hemorrhage. It is not uncommon for a patient to recover after coma has lasted for five or six days, but deep coma with impairment of respiration is as rarely recovered from in softening as in hemorrhage. The stage of inflammatory reaction is attended with considerable danger in severe cases, and the occurrence of rapid sloughing or of convulsions is usually of fatal augury.

It is rare for a patient to die in a first attack of cerebral softening unless a very large vessel is occluded. But if the brain has been damaged by a preceding attack, recovery occurs less readily, and the immediate danger to life increases with the number of preceding attacks.

Thrombosis of the internal carotid or of the basilar entails imminent danger to life, and the latter, from whatever cause it arises, is rarely survived. Next in gravity is obstruction of the vertebral, and next that of the middle cerebral. Obstruction of the trunk of both middle cerebrals is almost always fatal. Subject to these indications, the danger to life is far less in embolism and in syphilitic disease than in senile softening, so far as concerns the cerebral lesion. In most fatal cases of cerebral embolism death has been due more to the state of the heart or to some general disease, as rheumatism, than to the disease of the brain. Death from syphilitic disease is rare unless the basilar artery or many vessels are affected, or the nature of the malady is not recognized and met by appropriate treatment.

The prognosis as to recovery from the persisting symptoms depends on the seat of the disease, and the reasons for regarding the symptoms as "direct" or "indirect" in nature. These indications are the same as in cerebral hemorrhage, and are described in the account of this (p. 787) and also in the description of the individual symptoms. The prospect of recovery depends also, to some extent, on age. Recovery of slightly damaged parts, and compensatory action of other parts, both take place more readily in the young than in the old. But the prognosis as regards recovery is *not* influenced by the nature of the disease. It is no better in syphilitic disease than in embolism. We may, by treatment, remove the disease in the wall of the occluded vessel, we may prevent an increase in the symptoms, but we cannot remove the clot that has finally closed it, and has extended on into the distal branches of the vessel. We cannot, therefore, restore the circulation through the vessel. Neither can we influence, by antisyphilitic treatment, the process of softening. In syphilitic disease of vessel, just as in embolism, this is a process of simple necrosis. Therefore, the syphilitic origin of the disease does not influence the prognosis of developed palsy. Most cases improve, and many recover, but they improve and recover in the same way as in every other form of acute cerebral lesion—because the symptoms are of indirect and not of direct origin, and sometimes, in obstruction, because a collateral circulation is possible. The improvement that occurs is not due to the antisyphilitic remedies that may be given, and if the softening involves the motor path or centres, enduring hemiplegia is the result. It is necessary to insist strongly upon this fact. Because a palsy is due to syphilis, it is often assumed, as a matter of course, that it will be removed by antisyphilitic treatment. The assumption is correct as regards the pressure effects of syphilitic growths and many syphilitic inflammations, but it is not true of necrotic softening from vascular disease. I have seen many patients who had been assured that, because their hemiplegia was of syphilitic origin, they would certainly be cured, and when, after a year or two, the paralysis remained, they were naturally indignant at the erroneous opinion they had received.

It must be remembered that convulsions *after* the onset are prone to recur, and the tendency to the return is greater, if some time elapses before they occur, and especially if the cortex is involved. They may continue even when the paralysis has passed away, and may constitute a disease resembling epilepsy in its course and even less amenable to treatment. Such recurring convulsions are far more frequent after softening than after hemorrhage, partly because the cortex is more often involved.

The probability of a recurrence of paralysis varies according to the nature of the disease. In embolism it is not great. Although a second attack is possible, and instances are occasionally met with, it is a rare accident, and in any given case a recurrence is unlikely. In syphilitic disease the probability of a recurrence is a question of treatment. Without proper treatment it is probable. Many vessels are usually affected, and if the disease is allowed to progress unchecked it is likely that another artery will be obstructed. In many cases, the severe attack was preceded by a slighter seizure, weeks or months before the onset, the cause of which was not suspected. Iodide of potassium effectually removes the disease of the wall, and although this does not restore the lumen of an occluded vessel, it does save other vessels from obstruction. The nature of the case is usually recognized and treated, and hence a recurrence of softening is extremely rare. In atheromatous softening, on the other hand, the tendency to recurrence is very great. Many arteries are always affected; the disease of their walls is progressive—an age degeneration beyond control—and sooner or later other vessels are almost sure to become obstructed. When another vessel will suffer cannot be foretold, but it is more likely to be speedy—to occur, for instance, within two or three years, if the first attack occurred without a removable exciting cause, *i. e.* independently of transient influences favoring thrombosis. If, for instance, the attack came on after depressing grief, during an attack of general illness, or in consequence of prostrating fatigue, a recurrence is more likely to be long deferred than if it came on without such excitant. On the other hand, a speedy recurrence is more probable if there are conditions favoring thrombosis that cannot be removed, especially cardiac weakness and dilatation. Exceptions to these rules naturally occur, because the rules take, and can take, cognizance of one element only in the morbid process. The other—the extent and degree of the vascular degeneration—must remain an unknown factor, which may often render our conclusions wrong, although they may be still more often right.

TREATMENT.—The treatment of the initial apoplexy is in part the same as in cerebral hemorrhage; in part it is different. The same precautions are necessary as to the avoidance of undue movement, and it is important to loosen the clothes around the neck so as to prevent any hindrance to the return of blood from the head. Sinapisms to the neck also probably do good; they hasten the recovery of consciousness, and by causing reflex contraction of the arteries probably quicken the cerebral circulation and lessen the tendency to stasis. The chief aim of treatment must be to maintain the cerebral circulation under conditions as nearly normal as possible, to steady the action of the heart, and at the same time to avoid too great a strain on the adjacent vessels in which the pressure is of necessity for a time increased. By doing this we may lessen the tendency to the spread of the thrombus (whether this is primary or secondary). Hence the patient should lie with the head and shoulders slightly raised, and flexion of the neck should be avoided. The bowels should be opened, but more gently than in hemorrhage. Violent purgation, by weakening the heart, may increase the tendency to an extension of the clot. If the patient is gouty and the heart regular, a somewhat stronger aperient may be given. Under the same circumstances a diuretic is useful—half-drachm doses of nitric ether by the mouth or by the rectum. Whenever the heart is feeble, and especially if it is irregular, digitalis should be given in small doses, five minims of the tincture repeated every four or six hours. An increased pressure in the vessels adjacent to that obstructed need not be feared from digitalis, because it contracts the arterioles as well as strengthens the heart, and by rendering the circulation more uniform and steady it lessens the tendency to the formation of clot. If it is at all probable that the disease is softening, the patient should on no account be bled. Bleeding has been advocated even in softening, but it is impossible to read the details of the cases in which it has been employed without being convinced that it has in many instances hastened death. It is not probable that a few leeches to the temples do any harm if the patient is fairly strong, nor is it probable that they do any good. Whether stimulants are given or not must depend on the state of the pulse and heart. If indicated, ether, ammonia, or alcohol may be given, the latter more freely than in hemorrhage. If there is doubt whether alcohol should be given or not in hemorrhage it is better to withhold it; in softening it is better to give it.

After the patient has regained consciousness, mental tranquillity is of the first importance. All excitement should be carefully avoided, since whatever disturbs the heart's action is liable to be followed by reactionary failure and extension of the thrombus. Food should be light, easily digested, and nutritious. Alcohol is better avoided after the first twenty-four hours (unless the state of the pulse urgently demands it) on account of the risk of increasing the intensity of the inflammatory reaction. During this stage the management of the case requires great care and should be conducted on the same principles as a local inflammation elsewhere. The diuretic given at the onset may be continued. The bowels must be kept carefully open. The patient's head may be raised a little higher, but the shoulders should always be raised as well as the head. If there is much headache and fever, ice may be applied to the side of the head, with or without a mustard plaster to the back of the neck. Leeching on the temple or behind the ear has been advocated, and may be employed if the pulse and heart are fairly strong; but in most cases it is safer to avoid their use. If convulsions occur during this stage ice may be applied and a few doses of bromide may be given. The same treatment is suitable for delirium. Extreme cleanliness is essential. The limbs and back should be daily examined, and on the least indication of trophic disturbances the patient should be placed on a water bed. If this cannot be obtained, pressure must be kept from any reddened spot by pillows or cotton wool. After the inflammatory stage is over, tonics are usually needed, especially iron and nux vomica or strychnine.

The variations in this treatment which the special form of obstruction renders necessary are not great. In embolism the general principles of treatment are also in the main the same as in thrombosis, because the latter process is always associated with embolism, clot forming not only on the distal side but also on the proximal side of the obstruction up to the origin of the first large branch, and it is important to avoid whatever may facilitate the extension downward of this secondary clot. Moreover, it is important in embolism as in thrombosis to strengthen and steady the action of the heart, in order to lessen the risk of further embolism. This is more likely to occur if the heart acts irregularly, because vegetations tend to increase during the feeble action and to be detached when it becomes excited. But in syphilitic disease something more is necessary. Although treatment is probably powerless to undo the mischief that has already occurred it prevents an increase. We know that syphilitic disease of the arteries is never limited to one spot; many arteries are usually affected, and the same artery may be diseased at separate spots, and it is of the utmost importance to avoid the extension of the area of obstruction by removing this disease. Iodide of potassium should therefore be given in doses of ten or fifteen grains every six or eight hours, or scruple doses may be injected into the rectum. If a patient has not recently taken iodide—and the probability that he has not is very great—thirty or forty grains daily removes the disease rapidly. It may be well also to rub in some mercury, especially if the symptoms indicate extensive disease. The importance of prompt treatment cannot be too strongly urged, because another artery may become occluded if any delay is permitted. Even if there is a possibility that the occlusion is due to syphilis, iodide should be given. In embolism and senile thrombosis iodide of potassium can do no good, and may perhaps do harm, because it has a distinct tendency, as its use in aneurisms shows, to increase coagulation. For the same reason it is better not to give iodide in syphilitic cases in unnecessarily large doses.

In all cases of vascular lesion of the brain the patient should be kept at perfect rest in bed for a period which varies with the severity of the disease, and may be two weeks in slight cases, and four or six weeks in those that are more severe. When the stage of inflammatory disturbance is over, and the process of repair of the less damaged structures has commenced, nervine tonics may be given, such as quinine, strychnia, hypophosphite of soda, and other tonics that may be indicated, such as iron. For the reasons to be mentioned presently, it is impossible to obtain satisfactory evidence that nervine tonics do good, but it is probable that they have some influence in the right direction. The better the general health the quicker is recovery in any disease.

For the treatment of the residual symptoms that depend on the destruction of tissue little, unfortunately, can be done. It is doubtful whether any therapeutic measures have a distinct influence upon them. The indirect symptoms pass away spontaneously. Many of the direct effects lessen after a time, with and without treatment, in consequence of the compensation effected by other parts of the brain. In hemiplegia, for instance, improvement always occurs in the leg, even when the motor path to the affected side is completely interrupted, in consequence of the other hemisphere gaining that power over the limb for which anatomical arrangements always exist.

In the early stage of hemiplegia gentle rubbing of the limbs is all the treatment admissible. It has a tendency to lessen the rigidity, and may be continued and made more vigorous when the early rigidity has passed away and the late rigidity has set in. Upward rubbing of the muscles has most influence in lessening the spasm. The influence of electricity on the paralysis is very small, as may, indeed, be anticipated from the nature of the palsy. Late rigidity in the flexors is lessened, for the moment, by faradisation of the extensors, and repeated applications sometimes seem slightly to diminish the contraction. If there is some power of voluntary movement, the stimulation of the muscles by any form of electricity is followed by a slight temporary improvement in power, but the improvement is not permanent, and it is doubtful whether any lasting good results from a course of such treatment. In any case, it is not desirable to apply electricity to the limbs during the first six weeks after the onset. The stimulation of the sensory nerves of the limbs has an influence on the brain, as the arrest of a commencing epileptic fit by such means clearly shows. The early application of electricity has been followed immediately by a fresh attack of paralysis. That the application caused the attack is very improbable, but it cannot be said to be impossible, and the mere sequence of events might have a very decided influence on the mutual relations of patient and practitioner. The application of the voltaic current to the cranium has been suggested as a means of influencing the recovery of damaged cerebral tissue, but from the powerlessness of the current on processes of repair in nerve elements that are quite accessible, I think it may be asserted that an influence on the cerebral disease is not within the range of therapeutic possibility. Theoretically, at least, the capacity for harm of this method of treatment is considerable. The tendency to improvement, by cerebral compensation, and by the spontaneous disappearance of indirect symptoms, is very marked, and makes it difficult to estimate the actual influence of treatment that is employed; at the same time it renders these cases a tempting field for the assumptions of the quasi-therapeutist. It is most important that, when there is evidence of destruction of the motor path, friends of the patient, if not the patient himself, should be made aware of the hopelessness of a search after a "cure," on the one hand, and, on the other, of the slow improvement that time will bring. In few diseases does more harm result, indirectly, from the mistaken kindness which conceals unpleasant truths.

In addition to the measures just mentioned, which tend to lessen late contractures, mechanical means are sometimes necessary for cases in which it becomes extreme in degree. The hand may be placed on a splint for a few hours each day; the spasm may be readily overcome, so as to apply the splint, by gentle continued extension, aided in severe cases by the immersion of the limb in warm water. A small cylinder may be placed in the hand within the flexed fingers, or an india-rubber ball to which a tube is attached may be introduced, and inflated after it is in the hand. When the paralysis of the leg is considerable, there is often shortening of the calf muscles, due to the weight of the foot, which brings it into the position of extension as the patient lies. This is a serious inconvenience, because it may prevent the patient standing and walking when he acquires sufficient muscular power. It can be more readily prevented than cured. The foot should be carefully kept at right angles to the leg during the early stage, by a foot-board or by a sand-bag beneath the sole. At a later period it may be necessary to get the foot up by attaching a cord with a spring inserted in it from the front of the boot to the knee, or by the use of a suitable splint. The active character of the contracture renders tenotomy undesirable.

The post-hemiplegic athetoid spasm is very difficult to influence by treatment. Electricity fails, as a rule, to lessen it. I have, indeed, met with one case in which the spasm presented a very marked and permanent diminution during a course of treatment with the voltaic current, but no similar effect was produced in a number of similar cases in which I employed the same treatment, and I am therefore disposed to regard the improvement as a coincidence rather than as a consequence. The incoördination is somewhat lessened by the persevering practice of hand gymnastics, regular rhythmical movements of the fingers and thumb. A set of "dumb piano" keys is convenient for this purpose. These are made for exercising the fingers of pianists, and are so constructed that the resistance of the keys can be varied by a spring. Systematic rubbing has also a slight beneficial influence on this as on other forms of spasm.

Softening of the brain is the most common cause of aphasia, and the question often arises whether any special treatment should be adopted for the loss of speech. So far as concerns general therapeutic measures, the treatment for the condition of the brain already mentioned is all that can be done; the only special treatment for the loss of speech is educational. The question only arises in a severe case of speech defect, in which there is destruction of the speech region, motor and sensory, on the left side of the brain. Under these circumstances speech is recovered by a process of training of the right hemisphere so as to bring the right-sided speech processes into a relation to the will similar to that of the processes in the left hemisphere. The readiness with which this can be effected varies in different individuals. In some it occurs speedily, in others it never occurs, and all treatment fails to evoke any power of voluntary speech. The precise method to be adopted must depend on the character of the defect, but must in each case consist in sedulous practice of the speech process that is deficient. If there is word deafness, simple directions must be uttered to the patient, and gradually varied and made more complex; if the patient has a difficulty in reviving word images, he must be exercised in naming objects; if there is motor aphasia, without word deafness, he should try to repeat words after another person. So with inability to read and to write. In motor aphasia, attempts to write are useless until the patient has made some progress with articulate expression. In every case the exercises must begin with the simplest word processes of each kind, and these should be mastered before more difficult processes are attempted. The practice should not be continued long enough to fatigue the brain, but should be repeated several times a day. Great patience and perseverance are required, but these will be rewarded by progress far more rapid than is possible if the patient is left alone.

In right hemiplegia it may be necessary to acquire the power of working with the left hand. A "type-writer" may with advantage be employed in some cases; the relative fitness of the various kinds is mentioned in the chapter on Writers' Cramp.

THROMBOSIS IN THE CEREBRAL SINUSES AND VEINS.

Coagulation of blood may occur in the cerebral sinuses and veins, and may cause mechanical congestion, œdema, capillary hemorrhages, and sometimes softening, in the parts from which the blood should be conveyed by the occluded vessel. The occurrence is usually of very serious significance, on account not only of its effects but also of the gravity of the conditions with which it is associated. In order to understand the pathology of the condition, it is necessary to bear in mind the chief facts regarding the venous circulation of the brain already described at p. 501.

Causes.—Sinus thrombosis may occur (1) from the state of the blood and circulation generally; (2) in consequence of disease adjacent to the sinuses. Having regard to the local process itself, the former may be called *primary*, the latter *secondary* thrombosis. The secondary thrombosis is more than twice as common as the primary.

Primary thrombosis usually occurs in association with general malnutrition and prostration, and hence is often termed "marantic thrombosis."* It occurs at all ages, but most frequently in children, and next in frequency in the very old. In children it is met with up to fourteen years of age, but the chief liability is during the first six months of life, and the chief cause is severe and exhausting diarrhœa. It may, however, follow almost any prostrating malady, lung disease, long-continued suppuration, or acute specific diseases. In adults it occurs occasionally during the last stage of phthisis, sometimes from acute diseases, in the puerperal state, or in the course of cancer.

These causes no doubt act partly by weakening the heart and so retarding the circulation, and partly by rendering the blood more prone to coagulate. Diarrhœa may act also by reducing the volume of the blood. The rigid walls of a sinus cannot contract in adaptation to lessened volume of the blood, and hence the circulation in it must be unduly retarded. The trabeculæ that cross its cavity doubtless also facilitate the formation of a clot, and so also does the irregular shape of the cavity. The seat of primary thrombosis is almost invariably the superior longitudinal sinus, very rarely the lateral, still more rarely the cavernous sinus. It is probable that the current of blood in the superior longitudinal sinus is normally more feeble than in any other vessel of the body. The veins that it receives open into it in a forward direction, so that the motion of the entering blood is opposed to that of the blood in the sinus. Moreover, these veins ascend to the sinus, and nowhere else in the body do ascending veins receive the blood of ascending arteries. Destitute, therefore, of the influence of hydrostatic pressure, they depend for the movement of the blood solely on the force of the heart, exerted through the capillaries. It is not surprising that thrombosis should occur in this vessel when the circulation within it is still further weakened by disease. This influence of the irregularity of the inner surface is shown by the fact that a clot is sometimes found on the wall of the sinus, in which a channel remains for the blood.

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* The term marasmus is currently used in a more restricted sense than is implied in this use of the adjective, although the latter is justified by the original meaning of the word.

Does primary thrombosis ever occur in the veins and not in a sinus? There is some pathological evidence that it does. Young children are sometimes seized with hemiplegia, commencing with unilateral convulsions that are prone to recur afterward. Sometimes the attack comes on without exciting cause, sometimes under the same conditions as sinus thrombosis. The symptoms point to a cortical lesion, and it is not improbable that this is sometimes thrombosis in one of the veins of the convexity. Very similar symptoms in the course of tubercular meningitis have been proved to be due to thrombosis in a vein. These cases of infantile hemiplegia are considered at a subsequent page.

2. *Secondary thrombosis*, the result of disease adjacent to a sinus. The most common cause is caries of the bone, and especially disease of the internal ear. Other causes are injury to the bone (especially when attended with inflammation of the diploë), meningitis, especially tubercular, compression of a sinus by a tumor or foreign body, and suppurative diseases outside the skull, erysipelas, carbuncle of face or neck, malignant ulceration, and even, it is said, suppurating eczema of the scalp (Tonnelé). This form occurs with nearly the same frequency throughout life, except that it is rare in the very young and in the very old.

The mechanism by which local disease acts is probably threefold. First, the inflammation may extend to the wall of the sinus, and the inflamed wall may cause coagulation of the blood within it. Secondly, a clot may extend along a vein that passes from the disease to the sinus. Almost every sinus receives veins from the exterior of the skull (see p. 502), many of them also from the diploë, while the superior petrosal and lateral sinus receive blood from the internal ear. The third mechanism is the simple compression of a sinus, but this is very rare. The first and second are common, and it is often difficult to say which has been effective in a given case. There is sometimes distinct evidence of inflammation of the wall of the sinus, and meningitis frequently coexists. But in other cases the wall of the sinus is healthy, and the clot is apparently extended into it by prolongation. Even when there is inflammation of the wall, this may be, in some cases, secondary to the formation of the clot. Hence it is not desirable to call the whole of this group "phlebitic thrombosis." The sinus affected is always that nearest to the local disease, and, as ear disease is the most common cause, the most common seat of thrombosis is the superior petrosal or lateral sinus. The superior longitudinal sinus is very rarely the seat of a secondary thrombus. In tubercular meningitis the clot may be limited to a vein.

Pathological Anatomy.—The affected sinus is usually filled with a clot adherent to the walls, and the more firmly, the older it is. A recent clot is dark red and soft, but after a few days it becomes paler, granular and friable. It is usually laminated. Sometimes the clot does not entirely fill the sinus. When due to adjacent caries, it is usually softened and puriform in aspect. The clot may be limited to one part of the sinus, or may extend through its whole extent, and even beyond it, into the tributary veins on the one hand, or into another sinus on the other, and even into the commencement of the internal jugular vein. The wall of the sinus is rarely inflamed in marantic thrombosis, but often when the condition is due to the extension of adjacent inflammation. Meningitis is common under the same circumstances. The veins from which the sinus receives its blood are always greatly distended, and the coagulation may have spread into one or more. The mechanical obstruction to the return of the blood causes intense congestion and œdema in the part from which the tributary vessels come. Capillaries often give way, especially in the gray substance, so that this is crammed with minute points of hemorrhage. This is most marked when and where the clot has extended into the tributary veins. If the clot is limited to a sinus, some blood can pass from the veins by other channels, but if a vein is plugged the circulation in the part is arrested. The result of the œdema and hemorrhage is to cause softening, often limited to minute foci, sometimes diffuse, so that a mulberry-colored pulp results. Very rarely meningeal hemorrhage results. Little is known of the changes that occur if the patient recovers, but it is probable that the destruction of tissue is rarely so great as in arterial obstruction, and that the affected part of the cortex ultimately presents atrophy and induration of the convolutions. When the clot softens, foci of inflammation may often be found in the lungs, sometimes purulent, constituting secondary septic abscesses. They are present in about half of the cases of secondary thrombosis.

Symptoms.—The symptoms vary much, and are often masked by those of the condition that gives rise to the thrombosis—the cerebral anæmia in primary thrombosis, the adjacent disease or the meningitis it excites, in the secondary form. The symptoms directly due to the thrombosis are of two kinds, those produced by the interference with the functions of that part of the brain in which the circulation is obstructed, and those due to the disturbed circulation outside the skull in the parts from which veins pass through the bone to the internal sinus. The former symptoms have little in themselves that is characteristic, and the latter, although characteristic, are comparatively rare.

Symptoms of Thrombosis of Special Sinuses: Superior Longitudinal.—External œdema and distention of veins may be present on the sides of the head and forehead. There may be epistaxis from the veins of the nose, which communicate with the anterior extremity of the sinus. In young children the fontanelle may become prominent, in striking contrast to its previous depression if there was collapse from diarrhœa. Cerebral symptoms are chiefly general—apathy, somnolence and coma; vomiting; and convulsions, usually general, but sometimes local; rigidity of the neck and sometimes of the muscles of the back. In adults convulsions are less common than delirium, quiet or active. There is usually headache. In both children and adults there may be strabismus, tremor of tongue or limbs, rigidity of the limbs or of the arms, or of the legs only. Unilateral symptoms are usually due to the extension of the thrombus into veins over one hemisphere, and then there may be unilateral convulsion, often beginning locally, and loss of power in one side, sometimes limited in extent. Thus, in a man in the last stage of phthisis, the superior longitudinal sinus contained an old, granular, partly-discolored clot, which did not quite fill it, and a more recent clot had extended into the veins over the right frontal lobe, which was intensely congested with capillary hemorrhage. The symptoms were mental dullness for a day or two, and then repeated convulsions beginning in the left arm, and first involving the whole of the left side, then ceasing on the left side and involving the right. The convulsions recurred every ten minutes until death, two hours later.

Cavernous Sinus.—There is usually some œdema about the eyelids and temples, and enlargement of the veins about the orbit, in consequence of the communication of the ophthalmic and facial veins. There may be distention of the retinal veins, usually transient, and there may be œdema of the optic papilla, usually slight. The eyeball may be prominent from the distention of the orbital vessels. Headache is common, but unilateral limb symptoms are rare. Sometimes there is paralysis of the nerves to the orbit, which run in the wall of the sinus, including the first division of the fifth. Neuro-paralytic ophthalmia has been observed (Lebert).

Petrosal Sinuses.—It is doubtful whether distinctive symptoms attend thrombosis of either of the petrosal sinuses.

Lateral Sinus.—There may be distention of veins and œdema over the mastoid process. Sometimes, especially if the clot extends into the commencement of the internal jugular vein, much of the blood that should pass into the external jugular finds its way into the former, and the latter on that side may be distinctly less full than on the other side (Gerhardt). The cerebral symptoms of occlusion of the lateral sinus are not distinctive.

Symptoms due to the Nature of the Thrombosis.—In *primary*, marantic thrombosis the symptoms vary in their accentuation. In adults the symptoms of a cerebral lesion of some kind are usually distinct; the significance of the somnolence and coma, of the delirium and headache, or of the motor symptoms, is unmistakable. But in young children the simple exhaustion from the diarrhœa is often attended by somnolence and inertia hardly less than that which attends the occlusion of the sinus. Epistaxis, œdema of the scalp, or retraction of the head, under such circumstances, should always attract attention. The temperature is normal or raised only one or two degrees.

Secondary.—In the phlebitic form the sinuses most often occluded are those near the ear, and the blood is able to escape by other channels, so that the damage to the brain is slighter, and the direct symptoms are less obtrusive than in thrombosis of the superior longitudinal sinus. Indeed, the cerebral symptoms that occur are due more to the meningitis which usually coincides than to the closure of the sinus. The clot that forms is usually septic, and breaks down quickly into purulent infective material, and the symptoms of septicæmia are often more pronounced than are those of a cerebral lesion. In some cases the local symptoms have been so slight that the occurrence of a morbid process was not suspected until the signs of secondary pulmonary abscesses were found. More frequently the septicæmic symptoms are of considerable severity, rigors, remitting or intermitting pyrexia, and a typhoid aspect. Gradually cerebral symptoms become more prominent. There is headache, sometimes local. The patient becomes dull, somnolent, and comatose; or, on the other hand, there may be delirium, quiet or violent. Motor symptoms may consist in varied paralyses, disordered sensation, convulsions, or rigidity, due chiefly to the meningitis. Their significance depends on their association with septic symptoms, or with the subcutaneous œdema already described. When the secondary thrombosis affects other sinuses the special local symptoms already described may be more pronounced.

The onset of the symptoms is sometimes sudden, but more often insidious. Their duration varies from a few days to a fortnight. Coma usually precedes death. Occasionally severe convulsions may exhaust the patient in a few hours, as in the case mentioned on p. 837. Recovery is extremely rare, but sometimes occurs in marantic cases, even when the external symptoms leave no doubt of the accuracy of the diagnosis.* It is probable that the clot may be absorbed and circulation reëstablished, but if the thrombus has extended into a vein, permanent damage to the cortex may result.

DIAGNOSIS.—In marantic thrombosis in the adult the occurrence of cerebral symptoms may lead to a suspicion of the condition if the patient has no heart disease and has not reached the degenerative period of life, but the diagnosis is only certain when there is external œdema or distention of veins. In an old person, without these symptoms, the cerebral disturbance is more likely to be due to thrombosis in an artery than in a vein. In young children, somnolence, coma, and even general convulsions may be due merely to cerebral anæmia (in the state termed "hydrocephaloid"), but if these symptoms continue after the diarrhœa has ceased and the collapse is slighter, thrombosis is probable; the occurrence of local brain symptoms increases very much the degree of probability, and the addition of the external symptoms renders the diagnosis certain. In phlebitic thrombosis the diagnostic difficulties are much greater. The cerebral symptoms are due to and suggest meningitis rather than thrombosis, and the indications of the latter are chiefly those of septicæmia (due to the softening of the clot) and the external œdema.

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* An instance of recovery with some permanent damage to the left hemisphere is recorded by Voormann (*Deut. med. Wochenschrift, 1882*, No. 36) in a child five months old, in whom the symptoms were tremor of tongue and right limbs, rigidity of neck, opisthotonos, with distention of the left temporal vein, and œdema of the scalp.

Prognosis.—This is extremely grave in all cases. Phlebitic thrombosis with septicæmia is probably always fatal. Marantic thrombosis is occasionally survived in children, but scarcely ever in adults, in whom it usually supervenes on a disease that is itself incompatible with the long continuance of life. If a patient recovers, the general symptoms pass away, but local symptoms may persist, and there may be some persistent impairment of intellect. Possibly some cases of so-called atrophy of the brain, that supervene on an acute illness in childhood, have this origin.

Treatment.—In cases of primary thrombosis the most important elements in treatment are to arrest the cause of the prostration, and to maintain and increase the strength of the circulation by stimulants and tonics, such as bark. The patient should lie on the back with the head and shoulders moderately raised so as to facilitate the movement of the blood in the posterior part of the sinus; a perfectly horizontal position lessens the influence of gravitation on the return of the blood. To facilitate the latter, flexion of the neck should be carefully avoided. No depleting measures are permissible.

In secondary thrombosis treatment usually fails to exert any influence on the disease. Free exit for all discharges from the wound must be secured. For the septicæmic form, quinine and salicylate of soda have been recommended. I think that full doses of tincture of the perchloride of iron more frequently produce a distinct effect upon this condition than any other drug, but it may be doubted whether recovery ever occurs in a case of this character in which thrombosis has occurred.

INFANTILE HEMIPLEGIA.

(ACUTE CEREBRAL INFANTILE PALSY.)

Hemiplegia of sudden onset is not uncommon in children, especially in young children. There is considerable difference of opinion as to the exact pathological condition on which it usually depends, and it is probable that the cause is not always the same. Hence it is convenient to give a brief account of the condition as a clinical variety of disease. It is probably not a distinct pathological variety. Many of the cases are examples of one or other of the forms of disease already described. In almost all cases there is paralysis—hemiplegia. Sometimes, perhaps, a lesion occurs and causes no local symptom, but such cases can scarcely be recognized. Very rarely there is general loss of power, but it is probable that in such cases both hemispheres are diseased; there is double hemiplegia.

It is to be understood that only the cases in which the onset is acute or sudden are now considered. Hemiplegia of chronic onset is generally due to a cerebral tumor. Another class of cases which must be excluded are those in which the paralysis dates from birth, and is due to injury received during the process of birth. These cases have been considered in the section on meningeal hemorrhage.

Acute cerebral palsy with a distinct onset during infancy or childhood is apparently rather more common in females than in males. Of eighty cases of which I have notes, thirty-five were in boys and forty-five in girls. In three-fifths of the cases the onset was during the first two years of life, three-quarters during the first three years, and seven-eighths during the first five years. It seems to be equally common in the first and in the second year of life, twenty-three cases having occurred in each. In my own cases males preponderate during the first year, and females after the first year, but it is doubtful whether the cases are sufficiently numerous to prove this relation.

In the majority of the cases the disease is not distinctly secondary to any morbid influence; it was apparently primary in fifty of the eighty cases. In some of these the onset was during hot weather, and in a few there had been distinct exposure to the sun, but the relation to season was not investigated in a sufficiently large number to justify any conclusion. We must remember that, by mere coincidence, nearly a third of the cases may be expected to occur during the time of the year when hot weather is occasionally met with. The onset occurred within a few days of a severe fall in six cases, two at the age of six months, two at a year, two at about two years.

Of the other cases under one year one was secondary to pneumonia (at one month) and one to severe diarrhœa (at four months). In all the other cases under one year the onset was apparently primary.

Of diseases to which the condition was distinctly secondary, two acute specific diseases take the first place with seven cases each—scarlet fever and measles. This relation has been noted by other observers.* In almost all, the onset was during the decline of the acute specific disease or within a fortnight of its termination. In three of the cases after scarlet fever, dropsy was present at the time of the onset; in the remaining four there was no dropsy. The cases after scarlet fever were all between two and five years of age; those after measles between one and four. In two cases the onset was during severe and prolonged whooping cough (at two and three and a half); in two it was during prolonged and severe bronchitis, and in two cases, both at the age of seven, the patient was suffering from what was said to be gastric fever. Only one case, at seven, occurred during rheumatic fever. In one case, not included in this series because the patient was eleven years old, the disease supervened on mumps.

The onset was attended by severe convulsions in more than half the cases;

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* Strümpell, Bernhardt, etc.

in some, the convulsions recurred at short intervals during several hours, and at the end of that time the child was found paralyzed. In other cases several distinct attacks of convulsion were separated by hours or days, and the hemiplegia was only found after two or three had occurred, or when the series was over. The initial convulsions are generally one-sided, affecting the limbs afterward paralyzed, and the later fits have almost always this distribution; sometimes the initial fits are general. In some cases the hemiplegia comes on without any convulsion. In most instances, whether there are convulsions or not, the onset is attended by severe cerebral symptoms, and the child often lies unconscious for several days. Sometimes there is fever and vomiting. In older children there may be some pain in the head, but this symptom is, on the whole, not prominent. In the cases that are secondary to some acute disease, attended with general prostration, or in which such prostration follows general convulsions, the hemiplegia may not be discovered for some days or weeks after the onset. If the paralysis is on the right side, it may be accompanied by distinct aphasia in those children who have already acquired the power of speech. The aphasia passes away completely in the course of a few weeks; rarely it lasts for a month or two.

The further course of the symptoms, and the persistent condition, vary in different cases. In most of them a considerable degree of hemiplegia remains, but it is probable that in some instances a cerebral lesion of the same character is so placed as to cause no persistent symptoms, and there may then be no indication of its existence. I remember, many years ago, finding a large cavity, the size of a hen's egg, in the posterior part of the left parietal lobe, in a man in whom no cerebrall esion was even suspected. With the exception of the half-vision centre in the posterior extremity of the hemisphere, it is probable that a lesion in early life causes lasting symptoms only when it involves the motor path or motor cortex.

The residual hemiplegia affects the right or left side with about equal frequency. Of the eighty cases there was left hemiplegia in thirty-seven, right hemiplegia in thirty-three; *i. e.* these acute infantile lesions are about as common on one side as on the other.* In some cases the amount of the persistent palsy is slight. If at first incomplete, it soon passes away from some part of the side, the arm or the leg. More often it is at first complete, and continues so for a few months, and then power slowly returns in some parts, especially the face and arm, in consequence of the compensation by the other hemisphere. Very rarely the paralysis has been bilateral from the first, and both sides have remained paralyzed. This is doubtless the result of a bilateral lesion, as one recorded case proves;† compensation is then impossible, and the palsy remains absolute in the legs as well as in the arms.

The ultimate condition of these cases necessarily depends on the severity of the symptoms. In a large number the arm remains considerably paralyzed. Some power is recovered in the shoulder and elbow, and a little in the hand, and as movement returns, contracture comes on. In the vast majority, the limb becomes the seat of mobile spasm (athetosis, post-hemiplegic chorea), of which these cases present the most typical examples.* There is varying flexor and extensor spasm at the several joints, the variations being greatest in the hand, in which there are often spontaneous movements, quick or slow, while voluntary movement is disordered and ataxic. The condition is that described at p. 520. The subluxation of the middle joint of the fingers, in consequence of the spasm in the interosseal muscles, is generally conspicuous. The active changing spasm is proportioned to the amount of voluntary power; when this is slight there is much fixed spasm, by which the wrist-joint is often strongly flexed. The spasm in the upper part of the arm may be considerable, and may keep the limb rigid, generally in extension. A strong effort with the affected hand causes a corresponding movement in the other hand, and *vice versâ*. In some cases recovery is so great that only a slight amount of incoördination remains, and occasionally even this is absent. The paralyzed arm is shorter than the other in most of the severe cases, and all the bones, even the scapula, present a diminution in size. The muscles may also be small, but are occasionally hypertrophied from the effect of their continuous over-action. The leg always regains considerable power, and the patient can walk without difficulty, although the growth of the limb may be so hindered as to cause a limp. The knee jerk is excessive, and a clonus can sometimes be obtained. Spasm is generally slight, but often causes some talipes equinus, or equino-varus, always to be overcome by gentle pressure; the toes are sometimes over-extended in the act of walking. In the face, the residual weakness is trifling, but often there is distinct over-action of the muscles on the affected side. This is seen best in the act of smiling, in which a slight movement occurs earlier, and is at first more marked than on the other side, although a strong movement may be distinctly slighter.

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* Bernhardt found right hemiplegia in two-thirds of his cases, but the small number (eighteen) from which his conclusions were drawn is evidently insufficient. (*Virchow's Arch.*, Bd. 102, p. 26.)

† Heubner (see p. 844).

As a rule, sensation is perfectly normal on the paralyzed side. It is probably impaired in some cases during the early stage, but recovery is constant, so constant that it can only be by the compensation effected by the other hemisphere. In the only case I have seen in which there was any loss of sensation some years after the onset, the loss was clearly functional, hysterical hemianæsthesia; a week later it had disappeared, and existed on the opposite side. It would, perhaps, be unjustifiable to say that there is never any persistent sensory loss, but the extreme rarity of such loss is evident from the fact that none could be found in any of the eighty cases that have come under my own observation. Mental defect is very common, and presents every conceivable degree, from a mere tendency to such functional derangement as hysteria, to pronounced iodicy. One of the most severe cases of hysteria that I have seen was in a girl, the subject of old infantile hemiplegia. Another frequent symptom is convulsion, recurring, and resembling idiopathic epilepsy in its course, although not in its origin. Such recurring fits are met with in at least two-thirds of the cases. Sometimes they continue from the onset; sometimes an interval of years may elapse before the fits begin, and occasionally the hemiplegia occurs in infancy, and the convulsive attacks are only added when the age of puberty is reached. These cases will be again considered in the chapter on Epilepsy. The convulsions are almost always one-sided, affecting only the paralyzed limbs, and they often begin locally, in some part of this side, especially in the hand. It is, however, a significant fact, indicating how profound is the influence of the repeated discharges on the brain, that the fits may ultimately be preceded by an aura identical with the most common aura of idiopathic epilepsy, and that minor attacks may become developed which consist only of loss of consciousness.

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* On account of the frequency of this symptom the cases have been termed "cerebral spastic paralysis," but the designation is too wide in its meaning to be very appropriate.

PATHOLOGY.—Very few observations have been made on the condition of the brain in these cases, and those are chiefly on cases long after the onset. The conditions found have varied, and are susceptible of different explanations. In some, a cavity has been found in the central region of the hemisphere, involving the central ganglia, and sometimes extending as far as the cortex of the motor region.* If there is an opening on the surface of the brain, the condition has been rather unnecessarily distinguished by a special name, "porencephalia." † The actual cavity may be very large, or may be small, and may appear as if cicatricial contraction had drawn the walls together. On the other hand, there is sometimes no cavity or evidence of extensive destruction of tissue, but some convolutions are small and indurated. In most cases the whole of the hemisphere in which the lesion is found is smaller than the other; the diminution in size may involve all parts of the cortex, and the tissue is firmer than normal, and contains more connective tissue.‡ The condition has been termed "diffuse lobar sclerosis," § or "chronic encephalitis." || Occasionally, such a diminution in size of the whole hemisphere is the only visible disease; the atrophy is, however, generally more marked in one part than in another. In the former case it is possible that there has been a primary lesion in one part of the cortex, and the wasting elsewhere has been secondary, although the two can scarcely be distinguished after the lapse of years. We are only considering now the cases in which there was a sudden onset, and it is scarcely conceivable

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* A good example is presented by a case under the care of Dr. Sturges, *Lancet, 1871*, vol. i, p. 369.

† It is curious that in some cases of such cavity there has been a defect in the bone corresponding to the hole in the brain. The origin of such defect in skull and brain is not known, but it is improbable that the condition belongs to the class now under consideration.

‡ Many examples of this are on record. A good instance has been described by Kast, *Arch. f. Psych.*, Bd. xviii, Heft 2.

§ Marie and Jendrassik, *Arch. de Physiologie, 1885*, No. 1.

|| Bourneville, "Recherches sur l'Epilepsie."

that any disease, affecting primarily all parts of a hemisphere, can have come on suddenly, or that actually sudden palsy should be the manifestation of a chronic process. It seems more probable that, in such cases, the diffuse change is really a secondary atrophy, although no theory that can be framed is altogether satisfactory.

Two kinds of lesion have been assumed, in current theory, as the cause of these acute symptoms. According to the one the primary lesion is in most cases vascular occlusion; according to the other, which has been put forward by Strümpell,* it is inflammation of the gray matter of the cortex, "polio-encephalitis," analogous to the inflammation of the gray substance of the spinal cord, "polio-myelitis." Attractive as the latter theory is at first sight, it rests upon no evidence, and the scanty pathological facts we possess are opposed to its validity, as are also the facts of etiology. A primary inflammation of any part of the cortex of the brain is a purely theoretical disease; if it occurs not rarely in childhood (as must be assumed on this theory), pathological evidence of it would certainly have been forthcoming, more abundant than that of the analogous spinal malady, as every kind of organic brain disease is more often fatal than is disease of the cord. It must be remembered that the general liability of the brain to primary inflammation is very much less than is that of the spinal cord, and we are therefore not justified in relying on analogy as a basis for theory. The circumstances of the onset present a marked contrast to those of the spinal malady, since the latter is scarcely ever secondary to a general disease, while the cerebral lesion, whatever its nature, frequently is.

The alternative theory, vascular obstruction, has much to support it. It is the common cause of such central cavities as have been found in some cases. It is a known consequence of such general diseases as frequently precede the cerebral lesion. It has been actually found in at least one case of the kind.† A cavity in the central ganglia can hardly be due to any other cause than arterial obstruction. But whether this is the result of embolism or thrombosis is an open question. The former has generally been assumed.‡ But in the vast majority of cases of proved embolism, there has been an obvious source for the occluding plug, in most cases endocarditis. In some of the conditions in which infantile hemiplegia comes on, endocarditis is most improbable § whereas we know that, at least, in the sinuses of the brain, primary thrombosis occurs in children, and sometimes occurs under the circumstances in which infantile hemiplegia comes on.§ Hence, while some cases may be due to embolism, it seems on the whole more probable that when the primary lesion is obstruction of an artery, this is the result of thrombosis *in situ*. In the cases in which there is no evidence of softening *en masse*, in which there is no cavity, but only shrinking and induration of part of the cortex, I have suggested that the lesion is probably thrombosis in a surface vein, and that the reason why this lesion is so seldom found post-mortem is because in fatal cases the clot usually spreads into a sinus before death, and the case is then regarded as one of sinus thrombosis. We know that the closure of a vein does not commonly cause softening of the whole of the cerebral tissue from which the blood should pass to the vein, but merely intense congestion, minute extravasations, and punctiform softening, a condition that may well leave the state of atrophy and induration met with in some cases. It is certain that thrombosis may be limited to veins. An important case has been published by Money, in which such thrombosis was found after scarlet fever, and the extravasations into the related brain substance showed that the coagulation had occurred during life.* We must remember that thrombosis in a sinus does not always involve its whole cavity: the clot may be limited to the wall or to one side, and the vessel may remain pervious. In this connection it is instructive to note that in one of my cases the history strongly suggested thrombosis in the superior longitudinal sinus. A child of four months old was prostrated by severe diarrhœa, and the fontanelle depressed; convulsions set in, and the fontanelle became extremely prominent; the child lay motionless for four days, and then, when improvement commenced, hemiplegia was found.

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* *Deut. med. Wochenschr.*, *1884*, No. 44.

† That recorded by Heubner, *Med. Wien. Blat.*, *1883*, No. 13. Bilateral palsy and rigidity with trismus developed during prostration after bronchitis in a child one year and four months old. Cavities were found in both hemispheres and the pons, with clots in the middle cerebral arteries, but these clots were "canalized," and the arteries beyond were pervious.

‡ See Abercrombie, *Brit. Med. Journal*, *1887*, vol. i, p. 1323.

§ Heubner assumes that in his case embolism had occurred. There was thickening of the lining membrane of the left ventricle, and he supposed that there had been a ventricular, not

We cannot yet give any trustworthy explanation of the mechanism of the spasm in the limbs which so constantly accompanies the hemiplegia. It seems to follow lesions of various kinds, degrees, and seat, in the cortex as well as the central ganglia. The far greater frequency of the symptom after a lesion in early life makes it probable that it is in some way due to the disordered action of centres that remain, and not to the direct effect of the disease itself.

The diagnosis of these cases has to be made chiefly from those in which birth palsy is one-sided, and this depends on the history of a distinct onset after birth, which is scarcely ever wanting. In cases in which similar symptoms are due to a stationary lesion of chronic character, such as a tumor, the early history is distinctive.

The treatment of the cases in the early stage must depend on the prob-

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valvular, endocarditis, from which the plug had come. This theory, to those familiar with diseases of children, will seem far less probable than that of thrombosis *in situ*, especially when the multiplicity of the cerebral lesion is compared with the fact that elsewhere in the body only one uncertain trace of embolism was found. Moreover, the canalization of a clot formed *in situ* is far more probable than in that of an embolic obstruction.

* Money, "Treatment of Disease in Children," p. 445. There had been symptoms similar to these in the cases now under consideration, but the clot did not seem of sufficiently old date to make the case a proof of the relation of the symptoms to the thrombosis.

able nature of the lesion. Whatever be its exact character, the treatment for thrombosis in veins and sinuses, described at p. 839, will be most suitable. The after treatment of the mobile spasm has been considered in the chapter on softening of the brain.

INFLAMMATION OF THE BRAIN.

(ENCEPHALITIS, CEREBRITIS.)

ACUTE INFLAMMATION.

The cerebral tissue, like most other tissues, may be the seat of inflammation, but in no organ has the part played by inflammation in producing morbid changes and symptoms been more variously estimated at different times. This is due to two causes: (1) Inflammation causes softening; hence it was once thought that all softenings are inflammatory. It is now known that most are not, but are, as we have seen, simply necrotic, and due to the arrest of the supply of blood. (2) Inflammation of the surface of the brain accompanies that of the membranes, and it is through the former that many symptoms of meningitis are produced. Hence, although the inflammation of the membranes is the primary condition, and the fact is now recognized in terminology, these cases were formerly called "inflammation of the brain," and are often still thus designated in popular language. These two classes must therefore be put on one side. The residual cases, in which acute inflammation is known to exist, are not numerous, and are almost entirely secondary in origin and local in position.

ETIOLOGY.—Acute inflammation of the brain usually results from one of three causes, a traumatic injury, contiguous inflammation, or some septic influence. *Injury* commonly sets up inflammation in the meninges as well as in the brain, but now and then the former escape, and the latter is affected beneath the surface, apparently because the white substance is more easily injured than the gray. All forms of injury may be effective, blows and falls on the head, fractures of the skull, and punctured wounds. Intensely acute cerebritis sometimes follow an operation on the brain. It may occur from mere concussion, but is doubtless set up by the mechanism of interstitial laceration. Usually the cerebritis is immediately beneath the seat of the injury; much less commonly it occurs at the opposite side of the brain, from *contre-coup*. The latter especially damages the centre of the convolution, so that there results ultimately an irregular shallow depression in its surface. This appearance is often seen near the apex of the temporal lobe in consequence of a blow on the vertex. The inflammation may subside, leaving only the changes above described, or it may go on to suppuration, and the resulting abscess usually runs an independent course. A punctured wound almost invariably causes an abscess. Another frequent cause of cerebritis is *bone disease*, usually actual caries, but sometimes osteitis which has not gone on to caries. The affection of the bone may be traumatic, syphilitic, or the result of contiguous inflammation. The latter is almost confined to the bones adjacent to or enclosing the organs of the special senses, the nose, orbit, and especially the ear. The inflammation of the brain thus excited usually accompanies meningitis, but sometimes occurs alone. It usually goes on to suppuration. *New growths* in the brain usually cause softening in the adjacent cerebral tissue, partly necrotic, the result of pressure, partly inflammatory, but the inflammation is slight in degree, is attended with œdema, and scarcely ever presents any tendency to suppuration. In *acute diseases*, especially erysipelas, diphtheria, and typhoid fever, minute foci of encephalitis, characterized especially by leucocytal aggregations, and sometimes by micrococcal infiltration, are often found on microscopical examination. Lesions of considerable size, in acute specific diseases, are generally due to embolism or thrombosis. In simple *vascular obstruction* the softening which results is chiefly necrotic; on its margin inflammation occurs, and is often considerable when the obstructing plug comes from a septic source, as in acute endocarditis. Even then suppuration is extremely rare. In pyæmia, however, the inflammation thus excited always goes on to the formation of pus.

Idiopathic cerebritis is practically unknown. It has been supposed to be the cause of sudden cerebral palsy in children, but until more evidence has been adduced in support of the hypothesis this lesion must be regarded as improbable (see p. 844). Acute functional disturbance of the brain may, however, be attended by minute changes, revealed by the microscope, and distinctly inflammatory in their character. For instance, in hydrophobia the medulla oblongata, the functions of which are so conspicuously deranged, presents such microscopical alterations—aggregations of leucocytes outside the vessels and even in the substance of the nerve tissue. These changes are not primary, but are secondary to the intense functional disturbance, although they have some of the anatomical characters of inflammation.

Pathology.—The alteration in the tissue of the brain which results from acute inflammation has been long and accurately known under the name of "red softening," since it is usually from the first much redder than the softening which results from necrosis or mere imbibition of effused fluid, and is "yellow" or "white." The red tint of the affected area depends partly on the distention of small vessels, but chiefly on minute points of extravasation, and, according to the number of the latter, the color varies from pale to deep red. The consistence is lessened, from the separation of the tissue elements by effused liquid and from their disintegration. For the same reason, the affected area is swollen, and, on section, stands up above the level of the adjacent brain substance. It is never sharply limited, and the consistence of the adjacent tissue is usually lessened. The microscope shows distention of the vessels, especially the capillaries, foci of hemorrhage, and accumulations of lymphoid (leucocytal) elements in the tissue and around vessels. These are always to be found, but vary in number; and, when numerous, the softened tissue may have the aspect of actual pus. The proper elements of the tissue—nerve fibres, ganglion cells, and neuroglia cells—are in various stages of degeneration, and from all these granule cells develop. The cell elements undergo first the stage of cloudy swelling, the nerve fibres become granular, the axis cylinder presents fusiform, highly granular enlargements, which, becoming detached, also form granule cells. At the margin of the inflamed area the ganglion cells also pass into the stage of cloudy swelling, but, instead of breaking up, may atrophy or become pigmented, or undergo a sort of vitreous degeneration. These changes in the nerve elements are the same as are met with in simple necrotic softening, and hence their occurrence does not show that they take any primary part in the process of inflammation.

It is only in the rare cases in which the inflammation is very slight in degree, and there is no actual destruction of nerve elements, that recovery of the affected part is possible. Small foci of considerable inflammation may ultimately cease to be visible to the unassisted eye, a different thing from actual recovery. In most cases visible damage persists. The complete disintegration of the tissue elements, and absorption of the remains of vessels, leaves a fatty emulsion in the resulting cavity, and to it the remains of blood pigment (often hæmatoidin crystals) give a yellow or reddish tint, always deeper in the gray than in the white substance. Such a cavity may at last be undistinguishable from one left by a simple necrotic process. Ultimately the fatty granules may be slowly removed, and a little clear fluid may alone remain. Occasionally a reticulated stroma persists (or is formed) in the softened area; the loculated cavities thus constituted may contract, so that ultimately a sort of fibrous cicatrix remains, with a little adjacent atrophy, the result of the initial œdema around the focus of inflammation. Now and then this cicatrix contains a central nucleus of fatty débris and hæmatoidin crystals. If, however, the inflammation is intense, the lymphoid (pus) cells are so numerous that the softening becomes actually purulent. A cavity thus results containing pus. This result of inflammation is further described in the chapter on "cerebral abscess." A distinctly purulent appearance has been acquired in so short a time as six or seven days. If a very acute and malignant form of inflammation affects an extensive area, rapid and infiltrating suppuration occurs, with acute sloughing of the cerebral tissue.

The traces of inflammation in typhus and typhoid fevers are seldom to be recognized by the unassisted eye. They consist of aggregations of lymphoid cells around the vessels or in the tissue, and may sometimes constitute a mass of relatively large dimensions, comparable in size to a tubercular granulation (Fig. 292). They are not always met with, and are more pronounced in typhus than in typhoid. They have been especially studied by Popoff, who states that the lymphoid corpuscles sometimes enter the protoplasm of the nerve cells, and may cause multiplication of the nucleus and other changes.*

The disseminated inflammation that occurs in some cases of erysipelas and diphtheria, and sometimes in septicæmia, is of especial interest on account of the organisms that are associated with it. Sometimes minute points of softening may be visible; more often the changes are only discovered with the microscope. In certain spots micrococci may be seen around the vessels scattered through the brain tissues, and even densely aggregated in definite "colonies." The condition has been termed "mycosis of the brain."*

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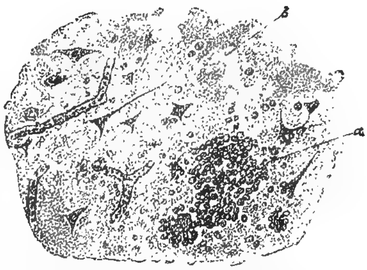
* Popoff, *Virchow's Archiv*, Bd. 87.

FIG. 292.



Section from the cortex of the frontal lobe in exanthematic typhus: *a*, accumulation of lymphoid elements; *b*, a cavity from which such cells have fallen. (Popoff, *Virch. Arch.*, Bd. 87; pl. i.)

SYMPTOMS, ETC.—Our knowledge of the exact symptoms of simple cerebritis is meagre, on account of the rarity with which it exists alone. Headache is certainly frequent; vomiting occasional; but optic neuritis is a rare result of the acute process. General convulsions and delirium may also be met with. These symptoms are irrespective of the seat of the inflammation. When the special motor or sensory region is involved, corresponding symptoms may be present, the most frequent being weakness in the limbs on the opposite side, and convulsions, local, or beginning locally. Such symptoms are not uncommon a few days after a cerebral injury, and are often attended by some elevation of temperature. They may pass away completely or may persist in slighter form. They differ from the actual injury to the brain by coming on, not immediately, but two or three days after the injury. The symptoms of an abscess, on the other hand, only develop after some weeks. A young child fell off a chair with a pair of scissors in her hand, and fell on the point of the scissors, one blade of which entered the skull just in front of the middle of the right temporal fossa. It passed in for about an inch and a half in such a direction that the point must have been very near the internal capsule. There were no immediate symptoms, but three days after the injury hemiplegia came on; probably the inflammation in the neighborhood of the wound reached the internal capsule. Sometimes the inflammation seems to set up a degenerative process in the brain, which runs an independent course, and is manifested by chronic mental failure and symptoms somewhat like those of general paralysis of the insane.

It is seldom that definite symptoms can be ascribed to the disseminated inflammation that occurs in acute specific diseases. The delirium and somnolence that so often exist may be in part due to these changes, but we cannot at present distinguish the effects of the definite lesions and of the condition of the blood, in the disturbance of the functions of the nervous system.

The *prognosis* of acute simple inflammation of the brain, as distinguished from that of the membranes, can seldom be made with certainty. Even in traumatic cases it is only when symptoms occur a few days after a punctured wound that inflammation of the brain substance can be diagnosed with confidence. In most other cases the symptoms are identical with those of meningitis over the corresponding region of the brain. In the form that results from specific blood states the condition may be suspected if cerebral symptoms become more pronounced and definite, but our knowledge of the effect of the lesions is still too meagre to permit any definite diagnostic rules to be laid down.

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* See Schüle, *Virchow's Archiv,* 67; Letzerich, ib., Bd. 75; and Blaschko, ib., Bd. 83.

The *treatment* of inflammation of the substance of the brain is, on the whole, similar to that of inflammation of the membranes.

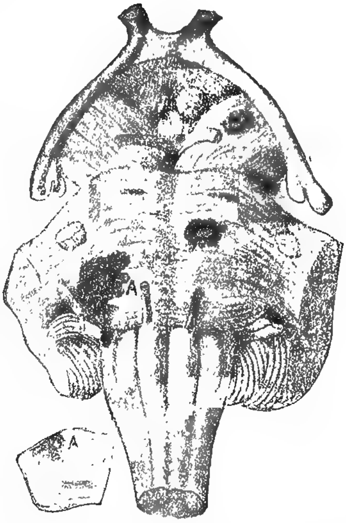
CHRONIC INFLAMMATION.

Of chronic cerebritis we have also very little exact knowledge. It is true the term "chronic encephalitis" or "meningo-encephalitis" is frequently employed, especially by French writers, but for the most part as a convenient designation for obscure cases, the exact nature of which is unknown. In this loose way the term is applied to all conditions in which traces of inflammation can be found, and the fact of inflammation is allowed to override the question whether the process was primary or secondary.

One class of these cases is that in which the chief alteration is an increase in the connective tissue in some part of the brain. The induration is regarded as the result of chronic inflammation, but it is certain that, in many cases, the inflammation is simply secondary. Thus, in the part of the brain compressed by meningeal hemorrhage in cases of birth-palsy, the induration has been thought to justify the designation of "chronic encephalitis." When the increase of connective tissue is primary, it is better to consider the case as one of "sclerosis of the brain."

The inflammation which surrounds new growths in the brain is often chronic as well as acute, and acute cerebritis, however caused, may pass into a chronic stage. When there are indications of a primary chronic cerebritis, these are almost always general and not local. Such indications are sometimes found in cases of diseases usually regarded as degenerative, as general paralysis of the insane. Leucocytal aggregations around the vessels and in the cortex are occasionally met with in this disease, but its extremely chronic course, and the absence of the headache which usually attends encephalitis, discountenance the view that it is primarily inflammatory in its nature. The term "chronic cerebritis" seem, however, strictly applicable to certain rare cases in which a patient suffers from headache and other cerebral symptoms (including optic neuritis)—symptoms that run a chronic course and may end in death. After death the only changes are the microscopical indications of general slight inflammation. For instance, a woman, aged thirty-four, had attacks of epileptoid nature, some vertiginous, others with transient loss of sight. Some months later she suffered from severe headache, with paroxysmal exacerbations, accompanied by vomiting, intense optic neuritis, slight pyrexia and terminal coma, the duration of the severe symptoms having been about six months. No naked-eye alterations were found in the brain, but slight diffuse inflammatory changes were found throughout its substance on microscopical examination.*

Fig. 293.



Chronic disseminated sclerotic inflammation of pons, etc. (See text.) (*After Charcot and Gombault.*)

A very rare variety of cerebritis is a disseminated form of chronic sclerotic inflammation, due probably to syphilis. A similar affection of the spinal cord is less rare. In the case described by Charcot and Gombault, of which the spinal lesions are shown in Fig. 94, p. 241, similar foci of disease existed in the pons, crura and optic nerves, and are shown in the adjoining figure (Fig. 293). The affected spots were gray in tint with yellowish centres, due to spots of caseation, such as are represented in the smaller figure, which is a section of the diseased area in the pons, marked A. Both sixth nerves and the right third nerve are partially degenerated. Thus, in the tendency to caseation, the morbid process has the characters seen in syphilitic growths, but the histological characters of the diseased areas were those of a sclerotic inflammation rather than of a growth. In these points, the process has considerable analogy to that of the foci of chronic syphilitic meningitis. The patient had had syphilis nearly twenty years before the onset of the symptoms. Extreme mental dullness existed, and obscured to a considerable extent the symptoms due to the intracranial lesions.† Such disseminated inflammation is related, on the other hand, to insular sclerosis (q. v.).

The *treatment* of chronic inflammation, if its existence is suspected, must be in the main directed against its cause. In all cases of uncertain origin iodide of potassium should be given, and if this fails very small doses of mercury may be tried, but tonics should be given at the same time. Improvement of the general health is of great importance.

ABSCESS OF THE BRAIN.

Collections of pus may form on the surface of the brain or within its substance. In the former case, the membranes, thickened by inflammation, constitute one wall of the abscess. In the latter case, which is the more common, the pus is separated from the surface by a layer of brain tissue, normal or only slightly damaged, although a communication sometimes exists, through this layer, between the interior of the abscess and the surface. The white substance of the brain appears to be more prone to suppuration than the gray. An abscess may occur in any part, but is most frequent in the cerebral or cerebellar hemispheres, and is rare in the central ganglia, the pons, medulla, or the middle lobe of the cerebellum. Usually there is only a single abscess, sometimes there are two or more, and occasionally there are many.

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* Hughlings Jackson, "Ophth. Hosp. Rep.," vol. viii, p. 445.

† Charcot and Gombault, *Arch. de Physiologie*, *1873*, vol. v, p. 143.

General Etiology.—Abscess of the brain is usually due to injury or to suppurative inflammation, near or distant, from which a septic material is conveyed to the brain. The most common cause is disease of the bones of the skull, and of such disease that of the bony investment of the organ of hearing is at once the most frequent and the most potent. Injury, moreover, often causes abscess, not directly but indirectly, as a result of bone disease thus produced. We may conveniently divide the immediate causes of cerebral abscess into two classes, the local and the distant. The local causes are the most frequent, and to them no less than 70 per cent of the cases are due (173 out of 241 cases).* They are ear disease (102 cases, or 42.5 per cent.), injury (57 cases, 24 per cent.), disease of the nose (6 cases), orbit (3 cases), non-traumatic caries of other bones (5 cases), tumor of the brain (1 case). The distant influences cause 15 per cent of the whole. They are—suppuration elsewhere than near the bones of the skull (25 cases), pyæmia (9 cases), oïdium albicans (2 cases). In the remaining 15 per cent. no cause was detected. These causal influences will be presently considered in greater detail.

Age and Sex.—Cerebral abscess is far more common in males than in females. Of 232 cases, 174 occurred in males, 58 in females, the ratio being as 3 to 1. The various causes do not, however, influence the two sexes in the same proportion. From ear disease the ratio of males to females is 2 to 1, from injury 5 to 1, from suppuration elsewhere, 4 to 1. The greater liability of males to traumatic abscess is readily explained by their more frequent exposure to injury; their greater liability to abscess from other causes is less easy to understand.

No time of life, from birth to old age, is exempt, but the affection is very rare during the first year of life. The following is the relative distribution of 223 cases:—

1 – 9,	24 cases.	30 – 39,	29 cases.	60 – 69,	7 cases.
10 – 19,	48 "	40 – 49,	26 "	70 and over,	1 case.
20 – 29,	72 "	50 – 59,	16 "		

Thus, one-third occur in the third decade of life, and one-fifth in the second. The twenty years between ten and thirty yield more than half the cases. Those due to ear diseases are distributed through life nearly as the total number. Traumatic abscess is relatively more frequent in early life, two-fifths occurring under 20, and nearly one-fifth under 10. Pyæmia never, and distant suppuration rarely, causes cerebral abscess under 20.

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* Collected from various sources, and including the seventy-six cases tabulated by Gull and Sutton ("Reynolds' System of Medicine," vol. ii, 2d ed., art. "Abscess of the Brain").

General Pathology.—Suppuration in the brain, as elsewhere, must be considered as a result of inflammation, and the first stage of the process is apparently "red softening" (see p. 847). In all red softening, pus corpuscles are found in the diseased tissue, and if they are numerous the softened tissue gradually assumes a distinctly purulent aspect. It is even thought that, in some cases, pus is formed from the first without an initial stage of red softening (Huguenin). The pus formed has a greenish tint, and usually an acid reaction. It is fetid in one-fifth of the cases (Mayer); on what the fetor depends is uncertain, but it is probably the result of some special septic influence. Under the microscope, the pus cells, especially in old abscesses, are indistinct, having undergone granular disintegration. The wall of the cavity is irregular at first (in some cases for a long time), and there is a tendency to the increase of the abscess by the necrosis of portions of the limiting tissue. The "capsule," which forms after a time, is at first thin and delicate, and gradually increases in firmness and thickness. It has a smooth inner surface, and its substance is composed of connective-tissue elements, looser externally than in the middle. Beyond it the brain tissue is often softened by slight œdema, and in the immediate vicinity of the capsule there is some fatty degeneration of the cerebral elements. Doubtless most of the pus cells are escaped leucocytes, but some may be formed from the cells of the neuroglia. After the capsule is formed, pus cells still increase in the cavity of the abscess, and, if there is a channel by which the contents may escape, the formation of pus may be continuous and abundant.

Taking together all cases of cerebral abscess, it is about equally common for a capsule to be present or absent. The time at which one is formed is important, because, if known, its presence affords some indication of the age of the abscess. The question can only be decided by traumatic cases, in which the commencement can be accurately determined. In such cases the first indication of a delicate membrane has been seen at the end of the second week,* but it is rarely distinct before the end of the third week,† and it only assumes its character as a well-defined membrane with a smooth surface at the end of two months. But an abscess may remain for a much longer time without a capsule. The absence of this is therefore of less significance than its presence. The abscess may be entirely closed, but not unfrequently an opening, sometimes a fistulous channel, connects it with the surface of the brain, especially when there is bone disease, and it may thus communicate with the exterior of the skull. It may also burst into the ventricles.

The shape of the encapsuled abscess is more or less rounded, but a recent abscess with adjacent sloughing may be irregular and ill-defined. Rarely it consists only of a fistulous channel extending from the surface of the brain to the ventricles. The size varies in most cases between that of a walnut and a hen's egg, but it may be so large as to occupy apparently two-thirds of the cerebral hemisphere, or, on the other hand, it may be no larger than a pea. Multiple pyæmic abscesses are usually small. In about four-fifths of the cases the abscess is single (80.5 per cent.); in a fifth, more than one collection of pus exists. The abscess is almost always single, when of traumatic origin (93 per cent.), and usually when due to ear disease (87 per cent.). On the other hand, when due to suppuration elsewhere, this abscess is single in less than half the cases (46 per cent.), and in general pyæmia the abscesses are multiple in two-thirds of the cases, and they are generally numerous and small. When multiple, it is about as frequent for there to be two, a few, or many, as the following figures show: Two, 14 cases; a few, 15 cases (three, 10 cases; four, 3; five, 1; seven, 1); many, 10 cases. In half the cases of multiple abscesses these are situated in the same hemisphere of the cerebrum. Suppuration occurs in both hemispheres only when due to distant septic causes. In one-quarter of the cases the abscesses are situated in the same side of both cerebrum and cerebellum. Less frequently there are multiple abscesses in the cerebellum, and the cerebrum is free.

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* Lallemand, quoted by Lebert.

† Lebert, *Virchow's Archiv*, Bd. x, 1856, p. 95.

Suppuration occurs in the cerebrum four times as frequently as in the cerebellum, and is very rare in the pons, or medulla oblongata.* Cerebral abscess may result from any cause, but cerebellar abscess is produced almost exclusively by ear disease or distant influences. In the cerebrum, suppuration is a little more frequent on the right side than on the left (as 87 to 70), but the two hemispheres suffer almost equally from distant causes and from ear disease. The general excess of abscess in the right hemisphere depends entirely on the influence of other local causes than ear disease; these show a remarkable proclivity to affect the right rather than the left hemisphere.† Cerebellar abscess, generally due to ear disease or distant causes, corresponds in this respect to the cerebral abscesses of the same origin, and the two cerebellar hemispheres are affected with equal frequency. Suppuration is very rare in the middle lobe of the cerebellum. Any part of the cerebral or cerebellar hemispheres may be affected, but the position of an abscess due to local disease is determined by the situation of its cause. The temporo-sphenoidal lobe suffers more than any other part, on account of the frequency with which the cause of abscess is disease of the internal ear, over which this lobe lies.

An abscess, if of any size, exerts pressure on adjacent parts, although not to the same degree as a tumor. The convolutions over it are flattened, and the adjacent brain is anæmic and often softened. Moreover, an abscess in the middle lobe of the cerebellum may cause internal hydrocephalus, just as does a tumor in the same situation.

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* Cerebrum 186 times (not cases); cerebellum 41; pons 3; medulla oblongata once.

† Thus, injury caused abscess in the right hemisphere 22, in the left 15 times; disease of the nose, right 7, left 1; disease of the orbit, right 3, left 0; caries of other bones than the temporal, right 4, left 1. The greater influence of these causes on the right hemisphere seems to be too uniform to be accidental.

Anatomical Course.—An abscess, which has become encapsuled, may remain for a long time stationary. The capsule becomes thick and tough, may even become calcified, and very rarely the contents may undergo a similar change.* More often, before a capsule is formed, or while this is still thin, the abscess enlarges, usually more in one direction than in another, and the patient may die from the extensive interference with the cerebral functions. The abscess may ultimately burst into the lateral ventricles, less commonly on the surface of the brain. The former occurs in one-sixth of all cases; most frequently (one in three and a half) in abscess from distant suppuration (not distinctly pyæmic); less commonly in abscess from ear disease (one in five); still less frequently in traumatic cases (one in nine). The effect of rupture, external or internal, is to excite purulent inflammation of the meninges in the one case, of the lining membrane of the ventricles in the other, and these cavities become filled with pus. Purulent inflammation, of the ventricles may also be excited without actual rupture. Occasionally the ventricles contain an excess of turbid fluid but no pus. An abscess due to bone disease may communicate with the latter by a perforation through the thickened and adherent membranes, and pus may thus be discharged from the interior of the brain through the ear, nose, etc. A very rare accident is hemorrhage into the sac of an abscess; in one case extensive meningeal hemorrhage from a vein occurred over an abscess just beneath the surface. Small abscesses are occasionally found in the vicinity of a larger one, due apparently to its influence. Several contiguous abscesses may coalesce.

Special Etiology and Pathology.—(A) *Local Causes.*—(1) *Injury.*—While the actual proportion of abscesses referred to injury is about one-quarter (23.2 per cent.), it is probable that the actual proportion is larger, and that many of the cases in which no cause could be traced were due to some unnoticed or forgotten traumatic influence. The common form of injury is a blow or fall on the skull, causing actual fracture of bone in some cases, in others necrosis, while in many a careful examination revealed no injury to the bone. In the latter cases the abscess is usually situated deeply in the brain, beneath the part struck, and is apparently due to the interstitial laceration of the tissue. When traumatic necrosis of the bone exists, the abscess is sometimes deep, sometimes superficial, and its cavity often communicates with the pus about the bone. In fracture, the inner table is often splintered, spiculæ project into the brain, and the suppuration may be near the surface, or it may be deep-seated and connected by a fistulous passage with the irritating splinter. After depressed fractures, which have been trephined, hernia cerebri often accompanies the suppuration. Another occasional traumatic cause is a penetrating wound, as a stab or a fall on a projecting nail, etc. Traumatic abscesses are usually single, and situated beneath the injury, very rarely in the opposite part of the brain. Thus a fall on the occiput has caused an abscess in the frontal lobe. In one case a fall on the

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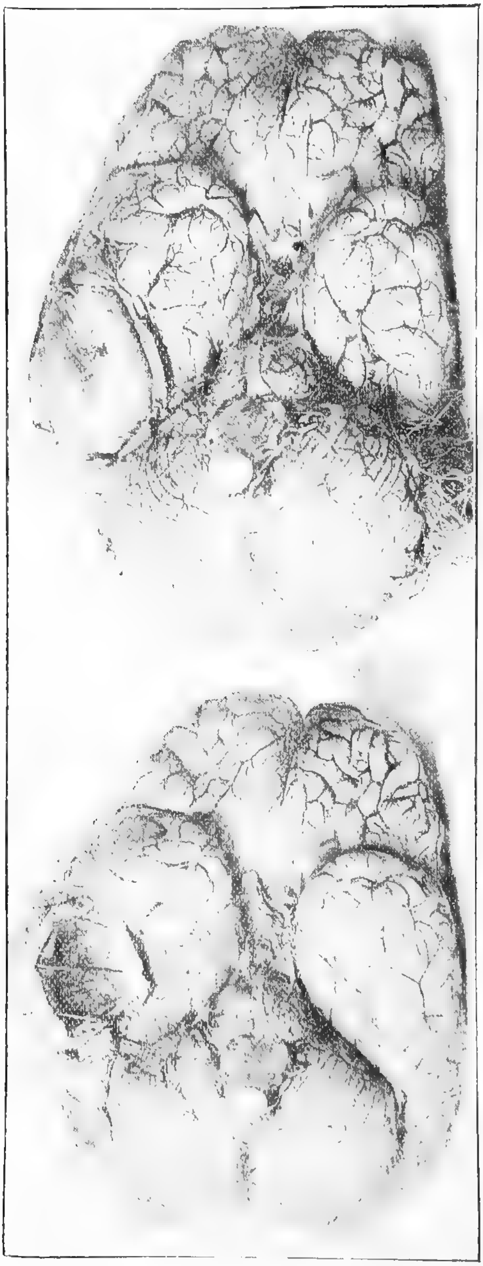
* Fenman, *Edin. Med. Journal*, October, 1879.

Figs. 294, 295.



Figs. 294, 295.—Abscess of right temporal lobe, due to ear disease. The enlargement caused by the abscess is shown in Fig. 294; in Fig. 295 the cavity is opened.

forehead, fracturing the bone, caused an abscess in the corresponding frontal lobe, and another in the cerebellum.

(2) *Disease of the ear* is the most frequent cause of abscess. The ear disease is usually chronic, and has existed for several years—five, ten, fifteen, and even twenty or twenty-five years—before it caused the abscess. Very rarely the mischief had existed only for a few weeks or months. There is usually caries of the bone, following an inflammation of the middle ear or mastoid cells, set up by cold, injury, or more frequently by extension from the throat, sometimes by a polypus in the external meatus. A purulent discharge from the ear (the tympanitic membrane being perforated) has usually existed for a long time. In many cases the abscess has followed the arrest of this discharge; less commonly it has followed an increase in the ear mischief, due to fresh cold or to a blow on the ear. Occasionally there is suppurative inflammation of the middle ear or mastoid cells, and no bone disease. The tympanic cavity and mastoid cells are separated from the interior of the skull only by a thin layer of bone, which is readily destroyed, and, moreover, this is perforated by small veins which pass from the tympanum to the superior petrosal, and from the mastoid cells to the lateral sinus. Abscess from ear disease is twice as frequent in the cerebrum as in the cerebellum. In the former it is usually in the temporo-

sphenoidal lobe, occasionally in the frontal, rarely in the occipital, and still more rarely in the parietal. Abscess of the cerebellum is almost invariably in the hemisphere. In rare cases the abscess is seated in the pons Varolii. Multiple abscesses from ear disease (met with in only 13 per cent. of the cases due to this cause) may be in the same cerebral or cerebellar hemisphere or in both; they are always on the same side.

The membranes are usually thickened over the diseased bone, but are sometimes normal. Coagula of old date, sometimes breaking down, are occasionally found in the petrosal or lateral sinus. The abscess is rarely superficial; commonly it is seated within the brain, separated from the surface by normal cerebral substance. In other cases an opening exists in this tissue and in the adherent membranes, so that the sac of the abscess may communicate with the diseased bone, and thus with the exterior. In some of these cases it is probable that the abscess commenced in the interior of the brain, and extended outward toward the diseased bone, until a communication was established.

The mechanism by which an abscess within the brain is produced by ear disease, not directly continuous with the cerebral suppuration, has been much discussed. There is no direct evidence as to its nature. That it is by the passage of septic material from the bone disease can scarcely be doubted. The arrest or retardation of the circulation in the sinuses may permit this material to reach the brain by the veins which, from ear and brain, pour their blood into the sinuses; but while the current is still flowing this can scarcely occur.* It is not improbable, however, that the perivascular lymphatic canals are the paths by which the infection generally occurs. The internal carotid artery sends twigs to the interior of the tympanic cavity, and the perivascular sheaths of these have been thought to be the chief channels of infection (Binswanger), but the rarity of abscess in the frontal lobes in consequence of ear disease renders this improbable.

The roof of the tympanum forms part of the middle fossa of the base of the skull, on which lies the temporo-sphenoidal lobe, and the superior petrosal sinus receives blood from both. The bone which separates the mastoid cells from the intracranial cavity forms part of the posterior fossa beneath the tentorium, and on it the cerebellar hemisphere lies, while the lateral sinus receives blood from both. Hence, as Toynbee first pointed out, disease of the tympanum causes chiefly cerebral abscess, that of the mastoid cells cerebellar abscess. Exceptions to the rule are, however, occasionally met with.

(3) *Chronic disease of the nose* is an occasional but rare cause of cerebral abscess (6 out of 240). There is usually bone disease, involving the nasal, sphenoid, or ethmoid bones, and often syphilitic in origin. In some cases the disease is confined to the nasal mucous membrane, just as the ear disease may be limited to the mucous membrane of the tympanum. Almost invari-

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* Adams, *Glasgow Med. Journ.*, vol. xv, 1881, June, p. 424. The problem of the mechanism is well discussed in this paper.

ably the abscess is situated in the frontal lobe; in only one recorded case was the suppuration in the parietal lobe. In one-third of these cases there are more than one abscess. Meningitis frequently coexists. When there is bone disease the sac of the abscess may communicate with this, so that pus is discharged from the interior of the brain through the nose.

Still rarer, as a cause of cerebral abscess, is *orbital disease* (three cases). In two there was an abscess in the orbit, in one a growth. The suppuration was always in the frontal lobe, and single.

Caries of other bones than temporal, and not due to injury, very rarely causes cerebral abscess. Five cases only were of this nature. In one the caries was syphilitic, in one cancerous. The abscess in each case was single.

The last local cause of abscess is a growth in the brain. This form of abscess is extremely rare, and occurs chiefly as a result of a tubercular tumor. Such growths usually caseate and harden, but have been known to break down into a collection of pus.* Other tubercular tumors, and other evidence of tubercle, are usually present, and indicate the origin of the abscess.

(B) *The distant causes* of cerebral abscess are morbid processes in some other part of the body, by which septic material is produced, and this, passing into the blood current, doubtless causes the abscess of the brain by septic embolism. The reality of this mechanism has been demonstrated in one case; a cerebral abscess, secondary to suppuration in the lung, was found to contain lung pigment.† Since embolic processes are rarely single, cerebral abscess from distant causes is frequently multiple. From local causes the proportion of cases in which there are more than one abscess is only 13.5 per cent.; from distant causes it is 61 per cent.

In general pyæmia the brain is far less frequently the seat of the secondary suppuration than are some other organs, and it is probable that the brain is less frequently affected in pyæmia due to injury than in pyæmia due to non-traumatic causes. Only nine of 234 cases of cerebral abscess occurred as part of general pyæmia, and in only one of these was the pyæmia secondary to injury; in one it was post-puerperal; in three it was secondary to abscesses elsewhere, and in two to abscess of the liver, produced by dysentery. In one-third of the cases the abscess was single, in another third there were from two to five abscesses, in the remaining third numerous small foci of suppuration were scattered through the brain, as many as sixty-eight being counted in one case.‡

In an important group of cases an abscess of the brain is the result of suppuration elsewhere, but no indications of general pyæmia can be discovered. Such cases are separable from the pyæmic form practically, but not theoretically. The occurrence of the secondary suppuration in the brain, and not elsewhere, is remarkable and unexplained. The cases are more numerous than those in which there is general pyæmia, and about ten per cent. of all cases of cerebral abscess are of this origin (25 of 234 cases). In the majority of these the suppuration is in connection with the lung. Pneumonia, imperfectly resolved and breaking down; suppurating cavities in the lungs, left by such pneumonia, or the result of bronchial dilatation; simple fetid bronchitis; and especially empyema, are the chief causal conditions.* It never results from true tubercular cavities. Suppuration in the abdominal cavity is a less common cause, and still rarer is suppuration connected with the limbs. The abscess is single in about half the cases, and is generally situated in the cerebral hemispheres, especially in the posterior lobe. The cerebellum is not often affected from this cause, and never suffers alone. Numerous small foci of suppuration have been met with, but far less frequently than from general pyæmia.

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* For an instance see D'Espine, *Rev. méd. de la Suisse Romande, 1886*, p. 371.

† Böttcher, *Petersb. Med. Zeitschrift, 1869*, and *Virchow's Jahresbericht, 1869*, ii. 51.

‡ It should be noted that pyæmia is not always the cause of a cerebral abscess with which it coexists. The abscess may be of local origin, and the pyæmia may be the result either of the abscess or of the local cause of the abscess.

Oïdium Albicans.—Strange as it may seem, in two recorded cases† thrush in the mouth was a cause of abscess of the brain. One patient was an infant, the other an adult. An abundant growth of oïdium occupied the mouth and throat, and the brain was studded with small abscesses, containing the same fungus. The oïdium in the pharynx has been traced into the epithelial layer of the mucous membrane,‡ and into the interior of the vessels, and it doubtless thus finds its way, with the blood current, to distant parts.

There remains about a sixth of the total number of cases in which no cause for the abscess could be discovered. In two or three of these congenital heart disease existed, and was regarded as a cause of the abscess, but no mechanism by which the result could be produced has been suggested. In many of these unexplained cases it is highly probable that the abscess was really traumatic, the result of some forgotten fall or blow. In a few cases in which the abscess was situated in the temporo-sphenoidal lobe, it may have been the consequence of disease of the tympanum, overlooked because there was no bone disease. It is still an open question whether all cases can be thus explained, and whether there is or is not an idiopathic form of abscess of the brain.

SYMPTOMS.—A cerebral abscess originates in inflammation, and constitutes, when developed, a foreign mass within the brain. Ultimately, secondary processes occur, adjacent œdema and inflammation, meningitis, effusion into the ventricles; or the abscess ruptures externally or internally. In some cases of acute and severe disease, the initial suppurative inflammation progresses with more or less rapidity until it destroys life. In other cases it passes into a stationary condition for a time.

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* In diseases within the lung substance capable of causing it, cerebral abscess appears to occur in about eight per cent., according to some facts recently published by R. Näther, (*Deutsche Archiv f. klin. Med.*, xxxiv, p. 169. Of ninety-eight cases forty-nine of gangrene of the lungs, thirty-seven of fœtid bronchitis, and twelve of bronchial dilatation) there was cerebral abscess in eight.

† Zenker, *Bericht der Gesellsch. f. Nat. u. Heilk*, Dresden, 1861, p. 62; Ribbert, *Berl. kl. Wochenschr.*, *1879*, p. 617.

‡ Wagner, *Jahrb. f. Kinderheilk.*, i, p. 56.

The symptoms correspond to the morbid process. The initial symptoms are those of inflammation, but vary much in intensity. In many cases they are so trifling as to be overlooked. In severe cases they are correspondingly grave. This is always the case in acute abscess; the early inflammatory symptoms continue and increase to the end. In the cases of chronic abscess, during the period in which the abscess is comparatively stationary, there are few or no symptoms, and this is termed the "latent period." Ultimately acute symptoms supervene, it may be suddenly, and run a rapid course, to end in death. These constitute the "terminal stage." The symptoms are in part those of inflammation; in part they are those of an irritating foreign body, such as a tumor. Their character is determined by the character of the morbid process, and by its seat. Local symptoms, often such as afford an indication of the position of the disease, are absent far more frequently than in the case of tumor. This is due to two circumstances. First, abscesses are often situated in parts of the brain, as the temporo-sphenoidal and frontal lobes, in which local disease often causes no local symptoms, whatever be its nature. Secondly, the pressure of a slowly-developed abscess produces less grave effects than the pressure of a tumor.

Every gradation is met with between the cases of acute abscess with severe cerebral symptoms, running a rapid course, and ending fatally in two or three weeks, and those in which the initial disturbance is so slight as to be unnoticed; the stationary period is marked by complete latency, and the existence of such grave cerebral disease is unsuspected, until the rupture of the abscess, or some acute consequence, causes rapid death. In another class of cases the existence of suppuration may also be unsuspected—those in which abscesses are secondary to a grave general disease, such as pyæmia, by the symptoms of which those of the cerebral mischief are altogether obscured.

Early symptoms are most frequent in cases of traumatic abscess, because the initial inflammation is most extensive. When present they resemble those of meningitis, which often coexists; there is headache, frequently local, vomiting and febrile disturbance, attended in severe cases by rigors. Convulsions are less common; when general they indicate that the mischief is extensive and severe. Local convulsions occur only when the disease is in or beneath the motor region of the cortex. Paralysis is still less common. Delirium is rarely an early symptom. In the cases of acute abscess these symptoms pass on into those presently to be described, which attend the terminal stage of chronic abscess, and usually consist in delirium, convulsions, and paralysis on the side opposite to the abscess. The temperature is raised, and severe rigors may occur. Delirium gives place to stupor, which deepens into final coma.

These cases of acute abscess are most frequently the result of injury, next of distant suppuration, and pyæmic embolism, while they are not common as a result of otitis. The duration of the symptoms is in most cases from ten to thirty days; sometimes death occurs at the end of a week; occasionally life is prolonged for four or five weeks.

The "latent period" of chronic abscess varies much in duration; and when, as is often the case, the initial symptoms were slight, its commencement cannot be fixed. It may vary from two or three months to several years. Indeed, it sometimes exceeds these limits, and an abscess which has become enclosed in a thick capsule has remained for many years, in one case for seven, in another for twenty, without exciting symptoms. But the latency is often imperfect. Slight symptoms may exist, and their nature is usually misunderstood. Headache is the most common, usually not severe. In cases of ear disease it sometimes alternates with otorrhœa. Convulsions have occurred at short intervals, and have been thought to be due to idiopathic epilepsy. In other cases slight mental disturbance, usually melancholic, has been the only symptom.

The latent period may end suddenly or gradually. The terminal stage usually commences suddenly, but it is sometimes preceded by a gradual increase in the headache or mental symptoms, by restlessness, irritability of temper, or depression. Very rarely acute symptoms develop, and then subside, and the latency continues as before. Such an attack is usually due to intercurrent meningitis. It is, however, rare; when the latency is once broken, the symptoms usually increase until death occurs.

The symptoms of abscess, therefore, are essentially those of the terminal stage; but these, in acute abscess, are continuous with the initial disturbance, and in chronic abscess they sometimes exist in slighter degree during the latent stage, and especially toward its close. Like those of tumor, they may be divided into general and local (focal), but the former are the more important, and exist alone more frequently than in the case of tumor.

Headache is as frequent as it is in tumor and presents similar characters. It is more often moderate in degree than in tumor, but now and then extremely severe, and has even been known to kill the patient by its violence. It is often increased by posture, and by muscular effort. It may correspond in position with the seat of the disease, and does so more frequently than in the case of tumor, but it is sometimes referred to a different part of the head, and the statements made in a subsequent page regarding the pain of tumor are applicable also to that of abscess. But in traumatic abscess it is generally referred to the situation of the disease. In abscess from ear disease, pain in the ear often blends with that in the head. Occasionally the pain varies in seat from day to day. Once established it usually persists.

Vomiting is often associated with the headache, and is especially frequent in cerebellar abscess. The same is true of giddiness. Although less frequent in cerebral than in cerebellar abscess, both these symptoms are occasionally very marked in the former.

Optic neuritis is less common in abscess than in tumor, but is certainly more frequent than statistics would indicate. It may occur in both acute and chronic abscess, and probably the ophthalmoscope would show that, during the latent stage, it frequently precedes the onset of the more acute symptoms. It is probably rather more common in traumatic abscess than in other forms. It has been met with in one case of abscess of the cerebellum (Pflüger). The aspect of the neuritis is similar to that which results from cerebral tumor.

Convulsions are frequent, usually general, and resembling epileptic fits. They may occur either at the beginning or end of the terminal period. Convulsions which are local in their distribution or commencement are less common, and occur chiefly in abscess beneath or near the motor area of the cortex. They are usually associated with paralysis. Rigidity of the neck, with retraction of the head, and slight opisthotonos, occurs chiefly when rupture has excited purulent inflammation about the pons and medulla.

Paralysis, usually hemiplegic in distribution, occurs in about half the cases. It is often slight and very rarely absolute. Sometimes it succeeds a unilateral convulsion. It is considerable only when the disease involves the motor fibres or cortex, and sometimes affects only the arm or the arm and face.

Sensation is affected much less frequently than motion. When there is hemiplegia there is occasionally diminished sensibility, but this has been a conspicuous symptom only in a few cases of abscess in the optic thalamus, or posterior part of the cerebral hemisphere, or in abscess of the cerebellum compressing the pons. The cranial nerves sometimes suffer, but more often from bone disease or meningitis than from the abscess itself. The olfactory nerves are scarcely ever affected. The optic nerves are often damaged by optic neuritis, and this may cause loss of sight, but vision is rarely affected by the direct influence of the abscess. Intolerance of light may accompany headache. The pupils may be unequal, and sometimes irregular, but as a rule they are normal until the terminal stage, when their reaction to light may be lessened and ultimately lost. The ocular muscles are sometimes involved, but there is rarely paralysis of a single nerve. The most frequent symptom is ptosis, generally on the same side as the abscess. The movement of the eyeballs is sometimes impaired in cases of cerebellar abscess pressing on the pons, although rarely in a definite manner. Pains in the region of the fifth nerve have been observed from the same cause, but it is scarcely ever paralyzed. Affections of the facial and auditory nerves are common in abscess from ear disease, but are the direct result of the caries of the petrous bone. Speech is often slow, but articulation and deglutition are definitely impaired only when the pons is the seat of abscess or is compressed by adjacent suppuration in the cerebellum.

Mental disturbance is among the most frequent of the terminal symptoms. Stupor, deepening to coma, is almost invariable at the end, and it is often preceded by delirium, or by mental depression, sometimes with suicidal tendencies. Chronic mental disturbance, or simple mental failure, is, as already stated, common toward the end of the latent period, before the final acute symptoms come on. When the abscess is seated on the left side, in the lower central or temporal regions, aphasic impairment of speech is occasionally observed.

Of the symptoms outside the nervous system, pyrexia is the most important. It is very frequent during the terminal stage, and sometimes precedes it, while in cases of acute abscess it is present, in greater or less degree, throughout the disease. There is often a dry, brown tongue, and rigors frequently accompany the pyrexia, followed by sweating, so that the case may resemble intermittent fever. The pulse may be frequent, especially when there is fever, but toward the end and sometimes throughout it may be infrequent, falling to 50, 40, or 30. Now and then there is profound anorexia, even during the latent stage. Constipation is common. The frequency of vomiting has been already mentioned. The sphincters are rarely affected until the terminal stage; during the final coma there is usually retention of urine.

Symptoms according to Seat.—For the reasons already explained, the local symptoms produced by abscess are less frequently distinctive of its seat than in the case of a growth in a corresponding situation. The general symptoms, headache, delirium, terminal coma, optic neuritis, etc., are irrespective of the seat of the abscess. The chief local symptoms have been already mentioned. They are most common when the suppuration is in or near the central (motor) region of the cortex, and consist in unilateral paralysis, partial or complete, and unilateral convulsions, beginning locally. In the precentral region of the frontal lobe, and in the temporo-sphenoidal lobe, an abscess rarely causes localizing symptoms. A small abscess in the corpus striatum or optic thalamus may cause no symptoms, but in some cases there is hemiplegia, doubtless from implication of the internal capsule. In one case of thalamic abscess there was anæsthesia and loss of the muscular sense in the opposite limbs, probably from implication of the posterior part of the internal capsule.

Abscess in the cerebellum may cause only general symptoms. The headache is usually occipital, often, in ear disease, darting from the ear to the occiput, and sometimes it is very severe. Vomiting is frequent, and occasionally there is typical cerebellar incoördination, probably in consequence of the pressure of the abscess on the middle lobe. Ptosis, and defect in the ocular movements, may occur from damage to the pons by pressure on or secondary softening, and in the same cases hemiplegia has been met with. Rigidity of the neck occurs in rare cases of cerebellar abscess without rupture; it is probably due to slight meningitis.

A small abscess in the pons usually causes no symptoms till it ruptures, but alternate hemiplegia, or even bilateral paralysis of limbs and face, has been observed.

When several abscesses are present, unless they are very small, one is usually much larger than the rest and determines the symptoms. The minute abscesses which are occasionally scattered through the brain in great numbers in pyæmia seldom cause definite symptoms.

Rupture.—The grave disturbance which marks the terminal period is usually due to the occurrence of inflammatory œdema and softening round the abscess, induced by its extension, and sometimes by some secondary influence, such as a blow on the head, or exposure to cold. Death is sometimes due to the rupture of the abscess, the frequency of which has been already mentioned. External rupture of a cerebral abscess may cause symptoms of violent meningitis, rapidly fatal, but frequently the adhesions of the membranes prevent the escape of the pus on the surface of the brain. A cerebellar abscess more often ruptures into the membranes; and the acute purulent inflammation which results around the pons and medulla usually causes rigidity of the neck, retraction of the head, slight opisthotonos, and even rigidity of the masseters, and convulsive shocks.

Rupture into the lateral ventricles occurs in abscesses in all parts of the cerebrum, but is especially frequent in those of the temporo-sphenoidal lobe. The symptoms produced bear considerable resemblance to those of ventricular hemorrhage, consisting of convulsions and of coma which rapidly deepens to death.

Occasionally a patient with abscess of the brain dies suddenly and the post-mortem examination fails to reveal the mechanism by which death was produced.

Course.—The varieties which the disease presents in its course have been already mentioned. They may be divided into the following groups, between which, however, intermediate forms occur:—

I. Acute abscess, with the early inflammatory symptoms progressing without considerable diminution into a terminal stage and the whole affection running its course in from one to four weeks.

II. Chronic abscess, in which a latent period intervenes between the early and late cerebral disturbance, and in which the early symptoms are often so slight as to be unnoticed.

(*a*) The latency is incomplete; some chronic cerebral symptoms occur, headache, convulsions, or mental depression, and these increase and pass gradually into the more severe terminal stage.

(*b*) The latency is complete and the existence of any cerebral mischief may be unsuspected until—

1. The terminal stage develops, usually suddenly.

2. Or death occurs from the cause of the abscess or else from some sudden effect of the cerebral lesion. For instance, a boy was struck on the head by a cricket ball; after a day or two he seemed well. Some months later he was found on his face in bed, dead, a sure sign of death from a convulsion. Post-mortem an abscess of the brain was found.

3. Or death occurs from some intercurrent disease. Such cases are extremely rare. Almost invariably death is the result of the abscess or its cause. But in a few cases the abscess has remained stationary for a long time. The capsule has become thick and even calcified, and the contents also have become inspissated and probably calcified. Such cases are the nearest known approximation to a recovery from an abscess. But the disease is still there, and its quiescence can hardly be accepted as proof of entire innocuity, since death has resulted from an abscess after a latent period of twenty years.

When nervous symptoms are once developed they usually run a rapid course. Taking all cases in which the duration was noted, the symptoms last less than five days in 20 per cent. (a fifth), in 33 per cent. (a third) they do not exceed ten days, in about half the cases they do not exceed a fortnight, and in three-quarters of the cases they do not exceed a month. On the whole, the symptoms are more often rapid in chronic abscess from ear disease (in which they usually succeed a latent stage) than in traumatic cases (in which there is often no latency). Of traumatic cases, only an eighth lasted less than five days, a quarter did not exceed ten days, and five-eighths did not exceed a month. On the other hand, of cases secondary to ear disease, in no less than a quarter the symptoms lasted less than five days, in a third they did not exceed ten days, and in seven-eighths they did not exceed a month.

DIAGNOSIS.—The extreme variability and frequent latency of the course of cerebral abscess often render its diagnosis difficult and even impossible, and no brain disease of equal extent and gravity so often escapes recognition. The cerebral symptoms are in themselves equivocal, and are common to other diseases, as simple inflammation and growth. They may derive significance from their association with general symptoms suggesting a snppurative process, especially fever and rigors. But the most important element in the diagnosis is the combination of these cerebral symptoms with a cause which is known to be capable of giving rise to cerebral abscess, especially chronic ear disease, suppuration in the lungs or elsewhere, or preceding injury. Fortunately, such indications are present in about three-quarters of the cases. The greatest difficulty is presented by those in which the stage of latency is complete, and the terminal symptoms are sudden in their onset and rapid in their course; the indications of a chronic cerebral disease are absent and the terminal symptoms resemble those of *apoplexy*. This is sometimes the case when sudden rupture into the ventricles causes only convulsions, coma and death. The nature of such a case can be suspected only when a cause for abscess exists. It is more common for slight symptoms to have preceded the terminal attack, and these increase much the probability of the diagnosis. In many cases, moreover, an ophthalmoscopic examination would reveal optic neuritis, a proof (in the absence of renal disease) that there is other disease than a mere vascular lesion.

When chronic brain symptoms are pronounced, the chief distinction has to be from *tumor*. Here also the etiology is of great importance. The absence of any cause is in favor of tumor, and the only cause which gives rise to both diseases is injury. Although tumor is much less frequently the result of injury than is abscess, yet the absolutely greater rarity of abscess compensates for the difference, and the absolute frequency of chronic abscess and growth, after injury, is probably nearly the same. Hence, in this case, the symptoms must be the chief guide. Definite focal symptoms, slowly increasing, especially with progressive paralysis of cranial nerves, are in favor of tumor rather than of abscess, and so also in very intense optic neuritis. The rapid development of severe cerebral symptoms, after slight indications of brain disease have existed for some time, is in favor of abscess, especially if accompanied by considerable fever and by rigors. Retrogression of severe symptoms is in favor of tumor, in which they are often caused by intercurrent meningitis. If the symptoms are equivocal, and etiology gives no help, the greater frequency of tumor turns the diagnostic scale.

Meningitis and abscess may readily be confounded, and not without reason, since meningitis may cause the terminal symptoms of abscess, and it may result alone from the most frequent causes of abscess, injury and caries of bone. The distinction, therefore, cannot always be absolute. Preceding symptoms are of longer duration in abscess, and meningitis affects the cranial nerves in greater degree, unless the abscess is seated in the pons. The terminal meningitis which results from abscess is usually purulent and due to rupture; it is more rapid in its course than that which exists alone, and the cerebral symptoms which often precede it materially assist the diagnosis. When these are absent the diagnosis may be as impossible as in the cases in which there are only terminal symptoms of apoplectic character.

The distinction from simple inflammation of the brain (*acute red softening*) has to be made chiefly in the cases of acute traumatic abscess. Since both these conditions often precede or accompany suppuration due to injury, the diagnosis consists less in a distinction between the two than in the recognition of the indications of suppuration over and above those of inflammation. One of these is afforded by the duration of the symptoms. Prolonged traumatic inflammation of the substance of the brain usually results in suppuration, and hence, if severe local symptoms continue for more than ten days, the formation of pus is probable. Another is the presence of the general symptoms, especially rigors, that usually attend suppuration. A third is the subsidence of the acutest disturbance, while slighter symptoms persist and subsequently increase.

The difficulty of distinguishing the existence of abscess in a case of chronic ear disease is sometimes very great, because some general cerebral symptoms are occasionally produced by the ear disease, and these may include (strange to say) distinct optic neuritis. It is, indeed, impossible to lay down any absolute diagnostic rules for these cases. The important fact is that pronounced cerebral symptoms, and especially considerable optic neuritis, are exceedingly rare results of simple ear disease, and in nine cases out of ten in which they are found, an abscess exists.

Functional diseases and abscess can only be confounded in the rare cases in which slight general disturbance, such as convulsions or melancholia, attend the latent stage, and the distinction can only be made by the recognition of some other symptom, such as persistent headache or optic neuritis.

Cerebral abscess can only be mistaken for a general disease, febrile or septic, in cases in which such general brain symptoms alone are present as might be produced by a toxic blood state. Usually, however, the coexistence of headache with delirium and stupor, and retardation of the pulse, will prevent this error, if the general relation of the symptoms is carefully watched and duly weighed.

Prognosis.—In a case of abscess, the diagnosis of which is certain, the prognosis is most grave unless the surgeon can give relief. This conclusion is not invalidated by the very long latency occasionally met with, because, in the few cases in which this has continued for several years, symptoms have been completely absent during the stationary period, so that the diagnosis of the disease was impossible. Only in the extremely rare cases in which relative subsides into absolute latency can the prognosis be at all hopeful, even as to the duration of life. The longer a stationary abscess lasts the thicker is its capsule, and the less easily is fresh mischief excited. The proved occurrence of calcification of the wall, and inspissation of the contents, together with the fact that an abscess may remain latent for as long as twenty years, permits the hope that, in some cases at least, the abscess may not shorten life, and such hope may reasonably be somewhat stronger if the cause of the abscess has ceased to be active.

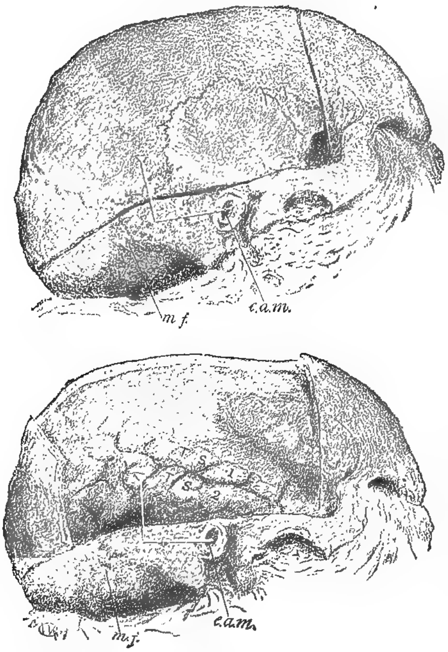
Treatment.—Although almost beyond the range of direct treatment, abscess of the brain is not altogether beyond the range of prevention, when due to its most frequent cause, local bone disease. This should be subjected to treatment, sedulous and persevering, and, above all, the free discharge of the products of suppuration should be carefully secured, by due and constant attention to the freedom of exit. This remark applies especially to ear disease, in which the first cerebral symptoms have often followed an arrest of the discharge. These measures should be continued, even when there is reason to fear that suppuration in the brain has already occurred, for the unfavorable course of cerebral abscess has been certainly accelerated by subsequent increase in the inflammatory and septic mischief in the ear.

The improvement of the health of the patient is of the greatest importance. Tonics, cod-liver oil, and fresh air, tend thus to lessen the tendency of the cerebral disease to spread. Of especial importance also is the avoidance of exposure to cold, and of blows or falls on the head. Each of these causes, in several recorded instances, has distinctly excited the disease to fresh progress. If the symptoms should fortunately become quiet and complete latency be developed, the same care must be maintained for years.

Acute cerebral symptoms must be treated in the same way as those that are due to simple inflammation. Rest, cold to the head, sometimes counter-irritation, as by a blister to the occiput, are the most important measures. The treatment of special symptoms must be conducted on the same principles as in the case of cerebral tumor.

Surgery has endeavored, and with success, to afford the direct relief which medicine is unable to give. The pus has been evacuated by means of trephining and the patients have recovered. But success has been obtained almost exclusively in cases of traumatic abscess. In these, the abscess is seated, as a rule, beneath the place of injury, and the guidance thus afforded enables the operation to be exactly localized. For instance, a boy struck his forehead and suffered afterward from headache and retching. At the end of seven weeks hemiplegia came on, with double optic neuritis. The frontal bone was trephined; the dura mater beneath was healthy, but an aspirator needle being plunged into the brain three drachms of pus escaped. The boy recovered, but with loss of sight from the optic neuritis.* This case illustrates forcibly a rule which is also indicated by the facts of many unsuccessful cases. If pus is not found on the surface of the brain a fine trocar is plunged into the cerebral substance. In more than one case of unsuccessful trephining the post-mortem showed that this procedure would have reached the abscess. Occasionally the operation has merely aided a process which nature was endeavoring to effect. For instance, a girl, aged eight, fell and cut her forehead. The wound healed quickly, but four months afterward an abscess formed over the bone, and opened spontaneously. A probe passed through carious bone into the interior of the skull. The trephine was applied, a wineglass of pus escaped, and the patient recovered.† In any other than traumatic cases, it is seldom that the symptoms are so localized as to indicate with precision the seat of the abscess. I am aware of no case in which the indications of cerebral localization have alone guided the operator successfully. The most they have done is to corroborate the indications of local injury, and in these cases they have been more useful in strengthening the surgeon's will than in guiding his hand. Roser, indeed, has been led, by a detailed study of the question, to the conclusion that trephining is only justified when there is actually a foreign body to indicate the precise locality of the abscess. This is certainly an error on the side of caution, as is shown by Hulke's case, given above.

FIGS. 296, 297.



Figs. 296 and 297.—Dissections showing the guide adopted by Mr. Barker in successful trephining for abscess from ear disease.

Until recently no case of abscess from ear disease had been cured by trephining. The practicability of the surgical treatment of these cases has, however, now been proved. In a case recently under my care at University College Hospital, Mr. Barker trephined over the temporo-sphenoidal lobe, in which an abscess was supposed to be, and the trocar, on its first introduction, reached the pus; the abscess was emptied, and the patient made a good and apparently a perfect recovery. The cerebral symptoms were slight, as in most cases of abscess in this situation, and consisted only in inequality of the pupil, vomiting, and optic neuritis of considerable intensity and very rapid development. With these symptoms the patient had had several rigors. The precise seat for the trephining was determined by Mr. Barker, by means of a careful dissection, represented in Figs. 296 and 297, and was at a point one inch and a quarter behind the external auditory meatus, and the same distance above its level. This point is over the second temporal convolution, and a trocar plunged in at this spot and directed forward will probably enter an abscess. The details of the surgical procedure will be found in the paper* in which this case is detailed. Another successful case has since been published by Dr. Greenfield.†

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* Hulke, *Medico-Chirurgical Trans.*, vol. lxii, 1879.

† Watson, *ib.*, 1870, p. 353.

INTRACRANIAL TUMORS.

The brain is often damaged by new growths which arise in its substance or spring from the enclosing membranes or from the bone of the skull. Almost all forms of tumor are met with, but some, simple fatty tumors, for instance, that are common elsewhere, are extremely rare in this situation. Others, such as massive tubercular growths, are more common in the brain than in other parts. Sarcoma is not rare, and one variety, glioma, is almost confined to the brain and spinal cord, being met with elsewhere only in the retina. Besides these, the list of intracranial tumors includes syphilitic growths, which are also common, cancer, fibroid and bony tumors, cholesteatoma, vascular or erectile growths, psammomata or tumors containing brain-sand, and parasitic tumors, echinococci and cysticerci. Intracranial aneurisms are considered in a separate chapter.

Etiology.—The cause of most of these tumors is not less obscure than that of similar growths in other situations. But the comparison of a series of cases brings out certain general etiological facts. The liability of males to suffer is twice as great as that of females. Of 650 cases of various forms of intracranial tumor, 440 occurred in males, 210 in females.‡ The greater liability of males is not due, as might be imagined, to the influence of syphilis, for it is true (as will be seen) of all forms of tumor, with the apparant exception of sarcoma. The difference has been ascribed to the influence of traumatic causes, but it is not probable that this influence accounts for more than a small part of the excess, since it is rarely to be traced, and the difference in the sexual liability is as marked in the case of children as in that of adults.

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* *British Medical Journal, 1886,* ii, Dec. 11th, p. 1154.

† *British Medical Journal, 1887,* i, p. 317. In a preceding case trephined successfully by Schede, a fistulous tract led from the surface to the abscess.

‡ This, and the other statistical conclusions given in the text, have been obtained by a fresh analysis of the cases contained in the collections made by Ladame ("Symptomatologie und Diagnostik der Hirngeschwulste," Würzburg, 1865) and Bernhardt ("Beiträge zur Sympt. und Diag. der Hirngeschwulste," Berlin, 1881).

This difference is not, however, equally marked in all forms of tumor. It is greatest in the case of tubercle and of glioma. Of 171 cases of tubercular tumors, 119 (or 69.5 per cent.), and of 65 cases of glioma, 45 (or 69.2 per cent.) were in males. Of 23 cases diagnosed as cancer and apparently such, 16 were in males. On the other hand, of 56 cases of sarcoma 26 were in males and 25 in females.

No time of life is exempt, unless it be the first six months; but cerebral tumors are not common in old age. The largest number of cases occur in childhood and in the active period of adult life. The first twenty years furnish one-third of the cases, the second two-fifths, and the third one-fifth. The proportion in the first decade, 18.5 per cent., falls to 14 in the second, and rises to a maximum of 20 in the third. In the fourth it is the same as in the first, 18.5 per cent., and it falls to 14 per cent. in the fifth, while after fifty years of age the cases rapidly become less numerous. The frequency in early life is largely due to the fact that at this period tubercular growths are so common; if these are excluded the proportion in the first twenty years of life falls to one-fifth, while that in the second rises to one-half, and that in the third to one-quarter of the total number. Age has little influence in relation to sex. The relative affection of males and females is nearly the same in each period of life, but there is a marked tendency, as in so many diseases, for the sexual difference of the early and adult periods to disappear in the decline of life; over fifty the two sexes suffer equally.

Tubercular tumors of the brain occur at all ages up to seventy, but, as just mentioned, they are most frequent in early life. Three-quarters of the cases occur during the first twenty years, and in one-half of the whole the patients are under ten years of age. Glioma is most common during active adult life; one-half of the cases occur between twenty and forty, one-quarter between forty and sixty, and one-fifth during the first twenty years. The relative distribution of the cases of sarcoma is nearly the same as that of glioma. Parasitic tumors are most common between ten and twenty, next between twenty and thirty, while under ten and over thirty they are very rare. The youngest sufferer was aged six, the oldest sixty-six. Tumors diagnosed as carcinoma have been met with at all ages, but one-half were in patients between forty and sixty years of age, and only two in the first twenty years of life. The other forms of tumor are too rare to permit their relation to age to be stated, further than that none of them seem to have been met with hitherto in patients under twenty.

Two forms of cerebral tumor, tubercular and syphilitic growths, depend upon diathetic influences. In most cases of tubercular tumors there is a family history of phthisis; the patients are frequently ill-nourished, and if adults, often present evidence of chronic lung disease. Syphilitic growths, although not unknown in the inherited form, occur chiefly in the acquired disease. The period after infection at which they develop is especially from the fifth to the twelfth year, but they have been met with as early as twelve months and as late as fifteen years after the primary sore.

Little is known of the influences which determine the occurrence of other forms of intracranial tumor. It is rare for any indication to be obtained of an inherited tendency to morbid growths elsewhere. There is little doubt, however, that traumatic influences, falls and blows on the head, are occasionally the immediate excitants of a growth, since the symptoms have been observed to follow a blow, and the tumor is found to correspond in position to the seat of the injury. Such a relation has been observed in almost all forms of tumor, in syphilitic and tubercular growths, as well as in those which are supposed to be of purely local origin. Traces of the traumatic mischief may or may not be visible after death, and apparently, the nutritive changes consequent on a mere concussion may be the starting-point of a tumor. But the cases in which a traumatic cause can be traced form a very small proportion of the total number, and the extent of this influence may readily be overrated.

Pathology.—Various classifications of intracranial growths have been proposed, founded, for the most part, on the structures in which they originate, but such classifications are of small practical value. It is more convenient to describe the various growths in the order of their frequency. If they are to be grouped, as an aid to memory, they may be placed in six categories:—

I. Diathetic—Tubercular and syphilitic.
II. Sarcomatous—Glioma, sarcoma, myxoma.
III. Carcinoma.
IV. Osteo-fibroid—Fibroma, osteoma, osteo-fibroma.
V. Miscellaneous—Cholesteatoma, lipoma, vascular or erectile tumors, psammoma, neuroma.
VI. Parasitic—Echinococcus and cysticercus.

Excluding syphilitic growths (the frequency of which is not fairly represented in published lists of fatal cerebral tumors), tubercular growths constitute more than half the total number of cases, and gliomata and sarcomata together about a third, glioma being rather more frequent than sarcoma. Thus the two groups, tubercular and sarcomatous, constitute together about four-fifths of non-syphilitic tumors of the brain. Carcinoma is much less common, but its actual frequency is uncertain, since many cases of glioma and of sarcoma have certainly been recorded as cancer by older writers. The statement sometimes made that cancer is one of the most common forms of cerebral tumor is entirely unsupported by authenticated facts. According to published cases, parasitic tumors are nearly as frequent as carcinoma, but their actual frequency doubtless varies much in different localities, and they are more likely to be published than are cases of cancer. The other forms of tumor are rare; all that can be said is that fibroma, cholesteatoma, and osteoid tumors are more common than vascular tumors (angioma), psammoma, lipoma, or neuroma, the last two being the most rare of all the tumors of the brain.

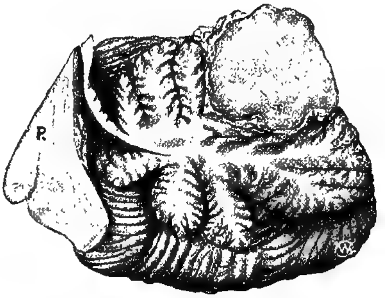
The relative frequency with which tumors are situated in different parts of the brain is as follows: Cerebral hemispheres (excluding central ganglia), 297; cerebellum, 179; pons, 59; central ganglia, 48; medulla oblongata, 31; corpora quadrigemina, 13; crus cerebri, 10. Thus the cerebral hemispheres are the most frequent seat of new growths, but when the smaller bulk of the cerebellum is considered, its tissue is evidently far more prone to give rise to neoplasms than is that of the cerebrum.

If we take all forms of intracranial tumor, the relative frequency with which the lesion affects the different parts of the brain is as follows. Of 718 separate growths (several existing in some instances), the order of damage was: cerebral hemispheres (white substance and cortex), 297; cerebellum, 179; base of brain, 76; pons, 59; central ganglia, 48; medullâ oblongata, 31; corpora quadrigemina, 13; crus, 10. Growths are also occasionally met with in the pineal gland, pituitary body, and the choroid plexus.

Tubercular tumors of the brain occur in the form of solid, firm, rounded masses, having little resemblance to tubercle in its miliary form. They vary in size from that of a nut to the size of a walnut, and occasionally attain still larger dimensions, that of a hen's egg, or even of the closed fist. Their section presents a uniform, opaque, cheesy aspect, sometimes softened here and there, but never in a large area. The periphery of the mass is softer, grayish, and translucent, and the adjacent brain tissue may be softened. Hence they are readily detached. It is in this gray zone that the growth of the tumor occurs by the development and coalescence of miliary granulations, which quickly degenerate and blend with the caseous mass. The growth apparently takes place in the lymphatic sheaths of vessels, and within the vessels thrombosis may occur. Hence the absence of vessels within the mass, and the quick and uniform degeneration of its elements. If such a tumor ceases to grow, the peripheral layer may undergo a fibroid change so as to form a capsule. Occasionally the degenerated mass undergoes partial calcification. Rarely the tissue softens, and a collection of pus results, increased, perhaps, by adjacent inflammation. Usually miliary tubercle is found elsewhere in the body, sometimes in the meninges. Occasionally these growths (like bone disease) may be the sole lesion, and hence they are sometimes called "scrofulous" tumors, but of their tubercular nature there is no doubt. They compress the brain tissue, which atrophies before the growth (Fig. 298); they do not infiltrate like some other tumors. They generally occur within the cerebral substance, without connection with the membranes; very rarely a small growth springs from the pia mater. Now and then they spring from the dura mater and merely compress the brain without invading it.

The most frequent seat of tubercular tumors is the cerebellum, either the hemisphere or, as in Fig. 298, the middle lobe; next (and almost as frequent) the cerebrum; while the other parts are affected less frequently and in the following order; pons, central ganglia, crura cerebri, medulla oblongata, and corpora quadrigemina. There is frequently more than one tumor. Of 183 cases, in 83 there was only one tumor, in 100 there were more than one. It is equally frequent for there to be two or more than two, and there may be many—ten, twelve, or even twenty, of various sizes. When there are more than one it is equally frequent for the tumors to occupy the same or different parts of the brain (the white substance and cortex of the cerebral hemispheres being considered as one part). In very rare cases there has been no mass of tubercle, but the whole brain was infiltrated with minute miliary concretions, none exceeding a barley-corn in size (Gee, Baly).

FIG. 298.



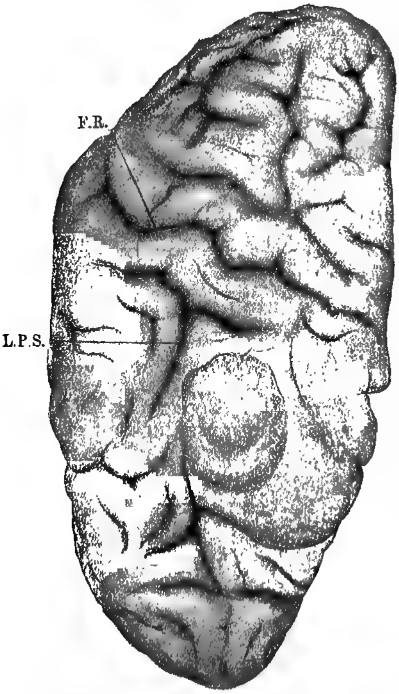
Tubercular tumor of the middle lobe of the cerebellum. The patient, a young child, was admitted into University College Hospital dying from general tuberculosis. P. Pons.

Syphilitic growths (syphilomata, gummata) are certainly more common than is suggested by the frequency with which they are found post-mortem, because they are more amenable to treatment than any form of tumor. They vary in size from that of a nut to that of a chestnut; in shape they are somewhat irregular and nodular. Their consistence is not uniform; they are firm, but not equally so in all parts. The section presents an irregular caseation; cheesy spots, isolated or coalescing into irregular tracts, are separated by a firm, gray or reddish-gray fibrous tissue. The growing surface is usually gray and gelatinous. The adjacent brain substance is softened and more or less displaced, but is not infiltrated by the growth. The more irregular surface and the irregular caseation are distinctions from tubercular tumors. Sometimes these tumors are met with in a state of more advanced degeneration, probably under the influence of treatment; they are shrunken, very hard, and fibroid, and may be surrounded by an indurated capsule. The process of obsolescence may go so far that only an indurated cicatrix is left. Syphilomata, rare in the cerebellum and central ganglia of the brain, are commonly situated in the cerebral hemispheres or the pons. They are usually superficial in position and attached to the pia mater; even when apparently more deeply seated a connection may usually be traced with a fold of pia mater between two convolutions. The dura mater is often adherent to the growth, which sometimes has obviously commenced in this membrane and thence invaded the brain. In such a case the invasion may be clearly observed to take place along the walls of vessels, probably in the perivascular sheaths. Hence it is probable that in the very rare cases in which no connection with membranes can be traced, the growth proceeds from the vessels. These tumors are rarely met with in the central ganglia, but occasionally occur about the optic thalamus, as an ingrowth from the neighborhood of the crus. When the growth is superficial, the membranes in the vicinity often present the indications of chronic inflammation with much thickening.

The sarcomatous group comprehends a variety of tumors differing much in structure, but all connected by intermediate forms. They consist of round, oval, or spindle cells, varying in delicacy, and with a more or less intercellular and fibroid tissue. There is, however, an important difference in their mode of growth. Some infiltrate without displacing the cerebral substance, and have no sharp outline. Others displace rather than invade the brain ; have a well-marked outline, and are surrounded by a zone of softening in consequence of the destruction of the nerve elements before the morbid growth. The former, from their infiltrating character, must grow in the neuroglia, and arise by a modification of its elements. Hence they are termed *gliomata*, and the tumors, before which the brain tissue undergoes destruction or displacement, are alone to be regarded as true *sarcomata*. The former are composed of more delicate, the latter of less delicate, tissue elements.

Gliomata.—The infiltrating character of gliomata was not that on which their designation was primarily based. Their elements were supposed to resemble those of the neuroglia; but the resemblance is never close, and they rarely present the stellate cells which characterize the normal interstitial tissue of the brain. Their elements vary as much as do those of other forms of sarcoma; sometimes round, oval, or fusiform cells predominate or may alone be visible. But the cells are always delicate, their outlines are difficult to recognize, and they are easily destroyed, so that their round or oval nuclei may alone be visible in a scraping. The interstitial tissue varies in amount; it is usually very delicate and homogeneous, but sometimes is so abundant that the tumor has a mucoid character (myxoglioma), rarely fibrous (fibro-glioma). Their tint is gray or reddish gray, and often closely resembles that of the brain tissue, being a little paler than the gray matter of the cortex, and their consistence is nearly that of the cerebral substance. The rare fibroid form is firmer ; the mucoid form softer than the brain. Moreover, they are prone to undergo softening, which may be so considerable and extensive as to convert the growth into a cyst, the wall composed of new growth, and containing, in its cavity, only the débris of its cells. The tint of the tumor depends on the varying amount of vessels which it contains. In soft tumors the vessels easily rupture and

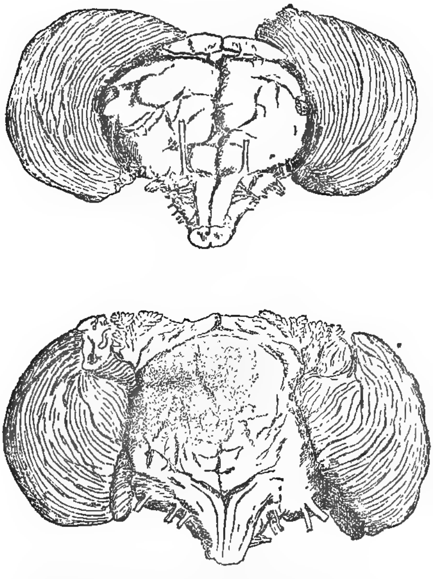
FIG. 299.



Infiltrating glioma of the cortex of the left superior parietal lobe.

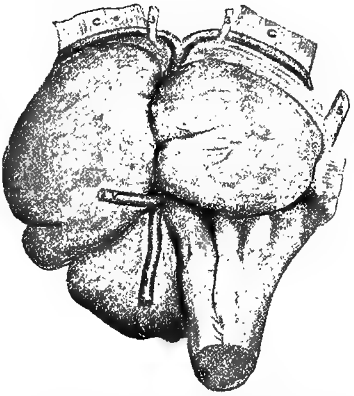
give rise to hemorrhage, so that extravasation may infiltrate a large part of the growth (as in Fig. 300), or an extensive hemorrhage may occupy a cavity with it. It is rare, however, for the hemorrhage to escape beyond the limit of the growth. From the infiltrating character of a glioma, it usually causes simple enlargement of the part of the brain in which it occurs; adjacent parts may be compressed by the enlargement, and may be displaced to some extent (as in Fig. 303), but there is not such simple displacement as by non-infiltrating growths. When the pons is the seat of the new growth, the whole of the pons may be greatly enlarged; the basilar artery may restrain the swelling, and ultimately be concealed in the bottom of a deep fissure between the two parts, as in Fig. 302. When a glioma reaches the surface, outgrowths, sometimes pedunculated, often form. Sometimes small, separate growths are met with on the surface, as in Fig. 303. It is rare for adhesions to the dura mater to occur. The degree and rapidity of the destruction of the nerve elements by the infiltration varies much in different cases. Sometimes nerve fibres persist, and retain their conductory power even in a part which is almost entirely infiltrated with the new growth. As already stated, there is no sharp boundary to the tumor, and it is only in the white substance of the brain that the difference in tint enables its limit to be recognized by the naked eye. In consequence of these characters, curious errors in pathological diagnosis sometimes occur. Cases of uniform infiltration of the pons with a myxoid glioma have been described as

FIGS. 300, 301.



Figs. 300, 301.—Infiltrating glioma of the pons. The dotted shading on the upper surface indicates the position of an hemorrhagic infiltration.

FIG. 302.

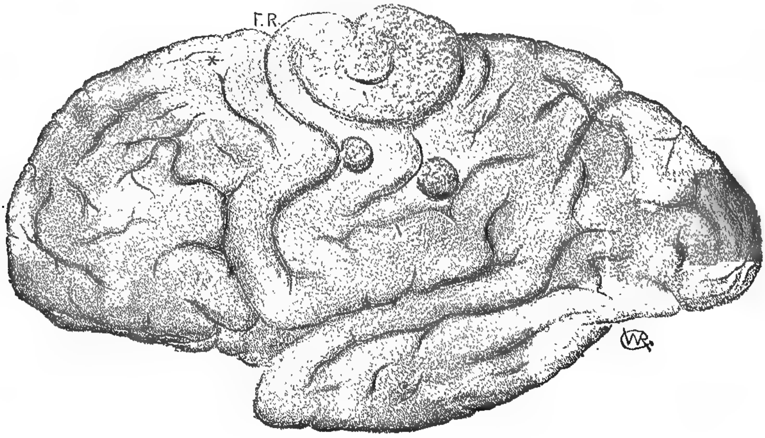


Glioma of the pons. V, V, vertebral arteries, the basilar is entirely concealed in the deep groove between the projecting growth on each side. On the right an extension downward has compressed the right side of the medulla; C, C, crura cerebri; 3, 3, third, and 5, fifth nerve. The patient was a boy, aged eight, who presented during life paralysis of the right fifth, sixth, and seventh nerves, and of the left arm and leg.

"hypertrophy of the pons."* In cases in which the growth has broken down, or has been the seat of hemorrhage, the existence of the tumor may readily be overlooked, and such cases have been described as simple softening or simple hemorrhage. Most of the cases in which optic neuritis has been described as accompanying hemorrhage or softening have been cases of glioma. A microscopical examination of the margin of the softened part will always prevent error. On the other hand, the rare fibroid forms have been mistaken for sclerosis of the brain.

Gliomata are single nine times out of ten (sixty-three out of seventy cases). Rarely several coexist (multiple glioma). The order of frequency with which they occupy the several parts of the brain is as follows: cerebral hemispheres (in one-half of the cases), cerebellum (in one-quarter), central ganglia, pons, medulla oblongata, crus cerebri, corpora quadrigemina.

FIG. 303.



Tumors, gliomata, of left hemisphere, a large one infiltrating the superior parietal lobule, two smaller growths, one pedunculated, on and just behind the middle of the ascending parietal convolution. At * another growth lay beneath the cortex. The symptoms were right-sided convulsions, beginning with pain in the elbow or shoulder, and affecting the arm chiefly, a few limited to the leg. There was also general right-sided weakness. See Hughlings-Jackson, *Med. Times and Gaz.*, *1875*, i, p. 661.

Sarcomata may occur within the substance of the brain, or may spring from the membranes, pia mater or dura mater, or from the bone, especially at the base. When they commence in the bone they may perforate the dura mater, but in this case, and also when they commence in the dura mater, they do not usually invade the pia mater, but merely compress the adjacent part of the brain, sometimes causing a depression of considerable size. Within the cerebral substance they do not infiltrate like gliomata, but have a well-marked limit, beyond which the brain tissue is usually softened, so that the tumor can be easily detached. In each situation they may be hard or soft in consistence; the softer forms are often very vascular, and constitute one form of "fungus hæmatodes." Such tumors growing from the dura mater or bone may perforate the skull and be felt as swellings on the surface, softer than nodes, for which they are sometimes mistaken. Occasionally these growths have a spongy section. Those which grow from the dura mater over the convexity often attain a large size; I have seen one that had the dimensions of a turkey's egg. Those which spring from the membranes at the base are usually small, firm, and more or less rounded. The softer forms may consist chiefly of round cells, but the firmer varieties are composed of spindle cells, varying in size, less delicate than those of glioma, and often separated by tracts of fibrous tissue. Occasionally the spindle cells

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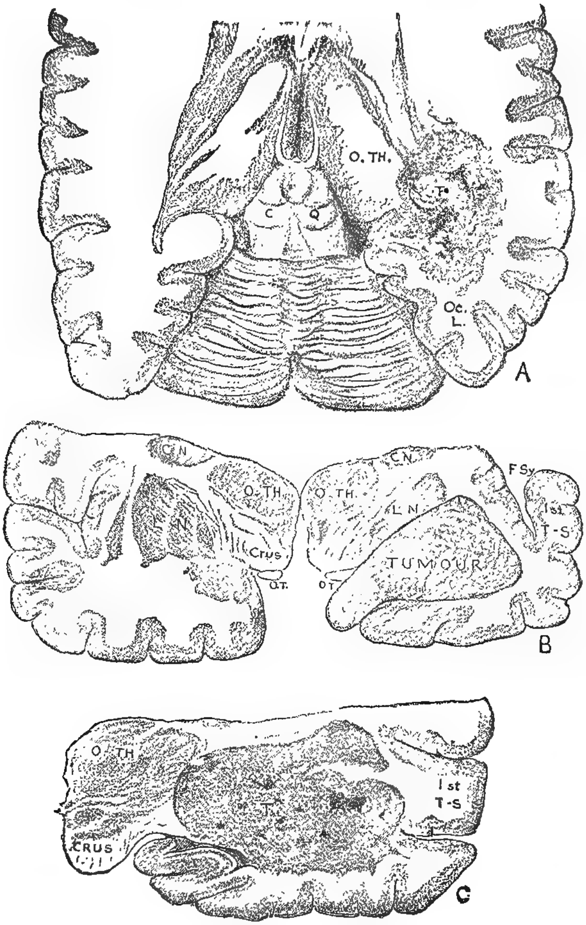
* It is certain that all the alleged examples of hypertrophy of the pons are cases of infiltrating glioma (see Money, *Med.-Chir. Trans.*, vol. lxvi, 1883).

FIG. 304.



Tumor, beginning between the right optic thalamus and first temporal convolution and extending inward from the temporal lobe, so as to compress the optic tract (Fig. 241, p. 572). A, horizontal section through the posterior parts of both hemispheres; T, tumor, with small irregular cavities, due to softening of its tissues; B, transverse section through the middle of the thalamus; C, through the posterior part of the thalamus. The patient suffered from hemianopia, fits beginning with an auditory aura (sound of bells referred to the opposite ear), optic neuritis and hemiplegia.

are arranged in concentric nests.* They may contain nucleated cells, such as are so abundant in the so-called "myeloid" tumors. In the substance of the brain these growths are usually single, but occasionally more than one exists; when they spring from the dura mater or the base they are often multiple. They are more frequent outside than within the brain, but may occur in any position, and are seated in the central ganglia more frequently than gliomata.

Carcinoma of the brain may be primary, or secondary to cancer in any other part. The form is almost always soft. It may spring from the dura mater or grow within the brain in any situation, but especially in the cerebral hemispheres. It is relatively more frequent in the central ganglia than other forms of tumor. It partly infiltrates and partly displaces the brain tissue. Usually single, carcinoma is sometimes multiple, and now and then a symmetrical growth occurs in each hemisphere. In one case, for instance, a large secondary tumor was situated in the central ganglia on each side.

FIG. 305.

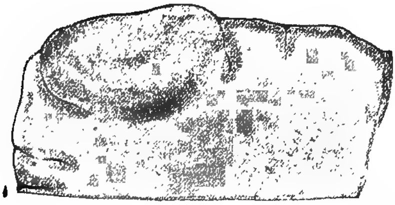


FIG. 306.

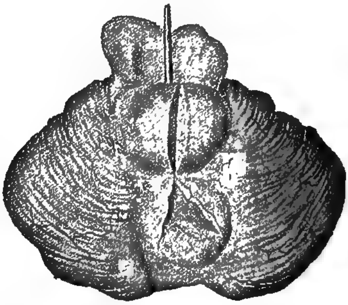


FIG. 305.—Cancerous growth in the cortex of the brain.

FIG. 306.—Tumor (cysto-myxoma) of corp. quadrigemina with a cyst in the superior vermiform process of the cerebellum. The tumor seemed to have sprung from the posterior quadrigeminal body and had involved the medullary velum. The patient was a boy, aged seven, who suffered from unsteadiness of gait and divergent strabismus. Acute symptoms, occipital headache, and vomiting, with a tendency to fall forward or backward, and optic neuritis supervened after a fall. (Kohts, *Virchow's Archiv*, lxvii, p. 425, and pl. xiv.)

Cerebral cancer is usually vascular, and forms sharply limited tumors, containing large cells, which sometimes have several nuclei. When seated in the dura mater the tumors are usually small and vascular, and may perforate the bone, or grow into the brain, and, like the similar form of sarcoma, are often termed "fungus hæmatodes."

The rarer forms of tumor may be briefly described. The so-called *myxomata* are merely mucoid forms of glioma. *Melanotic* tumors have been met with, always associated with similar growths elsewhere. *Fibroid tumors* are very rare, and have been met with chiefly in the cerebellum or cerebellar peduncle, small in size, usually single, but in one case multiple. *Bony tumors* are usually partly fibroid, "osteo-fibromata." They have been met with chiefly in the cerebral hemispheres, once in the cerebellum, once in the central ganglia, and once were multiple. (It must be remembered that

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* See *Medico-Chirurgical Trans.*, vol. lix, 1876, p. 217.

calcification may occur in any degenerating tumor, tubercle, glioma or sarcoma.) *Psammomata* are small tumors consisting of fibrous tissue mingled with particles of "brain sand," such as occurs in the pineal gland or choroid plexus—calcareous particles, which are sometimes aggregated into larger irregular masses. These growths usually spring from the dura mater at the base of the brain, or from the choroid plexus, and form small hemispherical tumors, reddish gray, smooth and hard. *Cholesteatomata* are small, hard bodies, which are usually found in recesses in the base of the skull, and are composed of epidermoid cells arranged in concentric layers. They are perfectly innocent in this situation, but they have also been met with of considerable size, as large as a hen's egg or even as a fist, growing into the cerebral substance and even arising within the hemisphere, in the central ganglia, or the pons. They are said to contain cholesterine and stearine; hence their name. Among the rarest of intracranial tumors are *neuromata*, small growths containing nerve elements; *lipomata*, which have been observed on the surface of the corpus callosum and corpora quadrigemina, in the ventricles and on the dura mater; and *vascular or erectile* tumors, which have been met with, once or twice, in the substance of the cerebral hemispheres. Fœtal tumors, *teratoma*, are among the most rare.*

Cysts in the brain are generally the relic of an acute destroying lesion, hemorrhage or softening; the effused blood, or degenerated nerve elements having, in the course of time, become absorbed. Such cavities have usually an irregular shape, and no well-defined wall. Simple serous cysts, of more regular form and with a fibrous wall, are sometimes met with in the membranes, the cerebral hemispheres, or the cerebellum; their origin is uncertain. Small cysts are often found in the choroid plexuses, and now and then these attain a considerable size. More frequently, cystic cavities are developed in connection with morbid growths, especially glioma and sarcoma. Such a growth may contain one or more cavities within it, or a cyst may be attached to one side of a growth, or the cyst may occupy almost the whole area of the tumor, its walls only consisting of the new growth. We have seen that a glioma often breaks down in part, so as to form a cavity, containing débris of its elements; in process of time these débris may be removed, and it is probable that the cyst thus formed may enlarge by the flow of liquid into it. Such cystic tumors are met with especially in the cerebellum, occasionally in the white substance of the hemispheres, very rarely in other parts of the brain. Dermoid cysts have been already mentioned.

Parasitic cysts may be either hydatid or cysticercous, the former usually single, the latter, which are less common, are often multiple. Hydatid tumors have been found outside the dura mater, but the usual position of both varieties is the cerebral hemisphere, where they are seated in four-fifths of the cases, either in the white substance or within the ventricles. In rare instances they have been found in the membranes, in the central ganglia, in the crus, pons, cerebellum, medulla oblongata, or at the base. Occasionally as many as fifty or a hundred cysticerci have been found in various parts of the brain.

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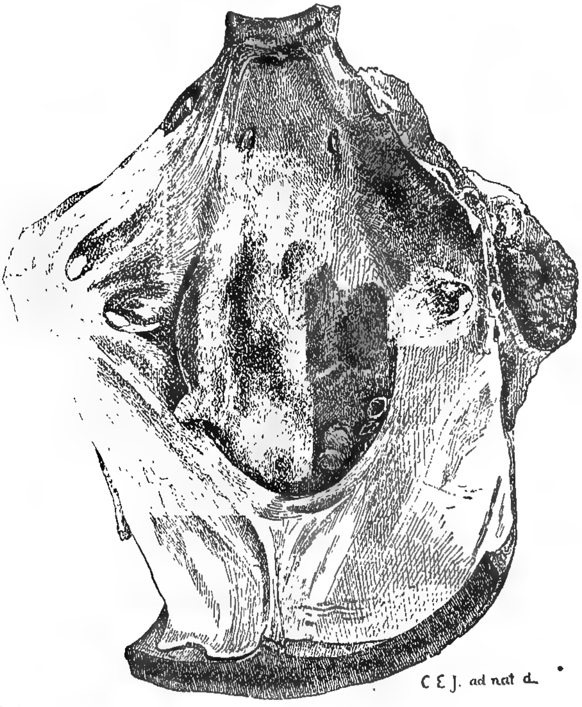
* In a case recorded by Hugo Beck (*Zeitschrift für Heilkunde, 1884*), a walnut-sized tumor occupied the position of the pituitary body, and contained bony and cartilaginous tissues and teeth. The patient was a woman, aged seventy-four, and the tumor had caused no symptoms. Dermoid cysts containing hair have also been found in the lateral ventricle.

The *pineal gland* may be invaded by tumors springing from the corpora quadrigemina, and has occasionally been found the seat of independent growths, usually firm and hard, attaining the size of a nut or a pigeon's egg. In one case the body was the seat of a cystoid enlargement, which occupied the whole of the third ventricle.

FIG. 307.

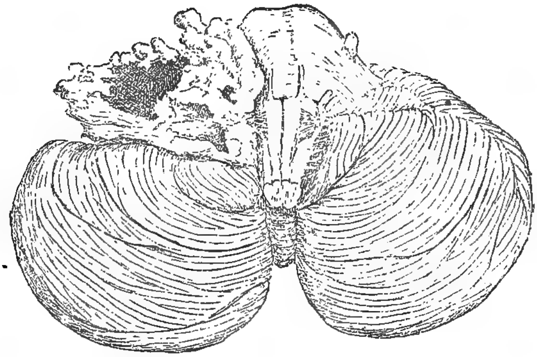


Growth from the occipital bone encroaching on the foramen magnum and compressing the medulla. (*Drawn by Mr. C. E. Jecks.*)

Tumors of the base of the brain deserve special mention. They usually spring from the dura mater, sometimes from the bone. Sarcoma and carcinoma are most frequent; less common are psammoma, cholesteatoma, syphilitic gumma, enchondroma, fibroid growth and hydatid cyst. The petrous part of the temporal bone, or the dura mater over it, is the part from which growths most frequently spring; the neighborhood of the sella turcica is another common seat, and sometimes they originate in the posterior fossa, adjacent to the foramen magnum. They may spring from the bone in this part (Fig. 307). They often attain a considerable size, even that of a hen's egg, and compress the superjacent brain. From their position, the cranial nerves are frequently damaged by basal tumors, which in the anterior fossa often compress or invade the optic chiasma and nerves to the eyeball, and, in the posterior fossa, the pons (Fig. 308), medulla, and the nerves which arise from them. The fifth nerve and Gasserian ganglion are affected with especial frequency by the growths which originate from the petrous part of the temporal bone.

Intracranial growths entail certain pathological effects, to which their symptoms are due. (1) By the process of growth they destroy directly the adjacent nerve elements. This destruction is partly the result of pressure, partly the effect of the growth of the morbid tissue elements. In the infiltrating tumors, these tissue elements grow between and enclose, and gradually destroy, the nerve elements. In the non-infiltrating growths, the nerve elements perish before the advancing tumor, and the zone of softening around these growths is due to this destruction. In both cases, however, the slower the process of growth the less extensive and less complete is the damage. Nerve fibres may persist within a slowly-growing glioma, or may long resist the pressure of a slowly-growing tubercle.

FIG. 308.

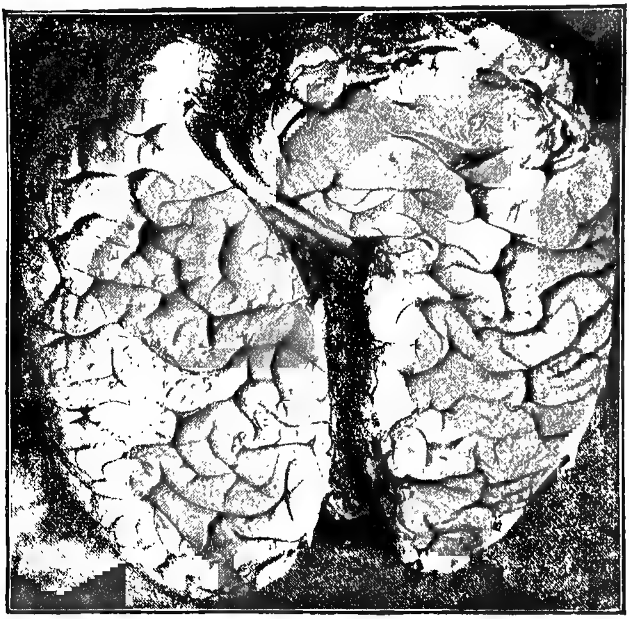


Tumor, containing cysts, compressing the right side of the pons Varolii.

(2) Intracranial tumors also exert distant pressure. A growth is a new mass within the skull, which occupies more space than the tissue which it has destroyed, and so exerts pressure on all parts in that region of the skull. The nearer the parts are to the growth the greater is the effect of pressure upon them. The more the pressure is limited in range by resisting structures the greater is its immediate effect. The falx offers some resistance to the extension of pressure from one cerebral hemisphere to the other, but is often displaced by it. More effective is the resistance of the tentorium, and tumors in the small space beneath it may compress all the structures therein contained. Thus the pons is often considerably damaged by tumors of the cerebellum.

One pressure effect is of especial importance, that by which internal hydrocephalus is produced. The cerebro-spinal fluid is chiefly secreted by the choroid plexuses of the lateral ventricle, and passes thence by the third ventricle, aqueduct of Sylvius, and fourth ventricle. This passage may be closed by tumors of the pons, corpora quadrigemina, third ventricle, or (at the aqueduct) by the distant pressure of tumors of the middle lobe of the cerebellum. The fluid is still secreted, but, unable to escape, distends the lateral ventricles. Moreover, the veins of Galen, which return the blood from the choroid plexuses to the straight sinus in the tentorium, are often compressed by tumors of the middle lobe of the cerebellum or corpora quadrigemina. It has been thought that the compression of these veins will alone cause internal hydrocephalus; it certainly increases the distention of the ventricles when the exit is closed, and under these circumstances the convolutions are much flattened, and even the sutures of the skull may be separated by the powerful distending force.

FIG. 309.



Large infiltrating tumor of the right frontal lobe causing displacement of the falx, and compression of the opposite hemisphere. (*From a photograph*)

(3) The growth of a tumor causes irritation, due in part to the pressure, in part to the vascular disturbance which attends it, in part, perhaps, to the influence of the process of new cell formation. The acute destruction of the nerve elements is an irritative process, sometimes even inflammatory in nature. To this irritation the circumjacent softening is chiefly due, and by it some of the most characteristic symptoms are produced. But the irritation is not confined to the brain substance. The meninges, especially the

pia mater, are more prone to inflammation than any other intracranial structure. Hence, if the growth reaches the surface of the brain, there is usually distinct evidence of meningitis over it. The pia mater, if not invaded, is vascular, opaque, and thickened, and adhesions form between it and the dura mater. Traces of inflammation, recent or old, sometimes extend for a considerable distance from the tumor, often more in one direction than in another. Now and then there is evidence of meningitis at a distance, without any traceable connection with the growth, so that it must apparently be ascribed to distant pressure. Thus, in a case of tumor of the corpora quadrigemina, which had caused internal hydrocephalus, there were signs of inflammation beneath each orbital lobule, and nowhere else. In the diathetic forms of tumor, syphilitic and tubercular, meningitis is especially frequent and important, because it also results from the constitutional condition. In tubercular tumors the meningitis is usually general and acute, in syphilitic growths it is local and chronic. The tubercular meningitis is frequently the immediate cause of death in cases of tubercular tumor, especially in children. In syphilitic tumors, the adjacent pia mater and arachnoid are often extensively thickened by a mixed process of inflammation and growth, matting together the structures, and damaging nerve roots at some distance from the growth.

An occasional effect produced by tumors of the brain is to cause a remarkable thinning of the cranial bones.* It is sometimes local, corresponding to the position of the growth, more often it involves all the roof and sides of the skull, less commonly the bones of the base also. In extreme cases the bone may be reduced to the thickness of card-board. Actual perforation has been observed only over the tympanum (Hale White). The inner surface of the bone is rough, in consequence of the unequal erosion. The outer surface of the dura mater may present corresponding inequalities, and the membrane is often thickened. The atrophy occurs when the tumor is within the substance of the brain as well as when it is at the surface, but the condition is only found when the tumor is large or there is internal hydrocephalus. It seems to be the result of the increase of intracranial pressure, which slowly causes an atrophy of bone similar to that produced by aneurism.

SYMPTOMS.—The nerve disturbances produced by intracranial tumors are commonly grouped into two classes, *general* or *diffuse* and *local* (see p. 508). The former are those symptoms which are produced by growths in various situations, and are of various kinds. Such are headache, vomiting, giddiness, general convulsions, optic neuritis, etc. The local symptoms (focal symptoms; German, *Herd-symptomen*) are those which depend on, and afford some indication of, the position of the tumor; they comprehend the local palsy and spasm, the affections of sensibility and of cranial nerves, which are the effects, more or less direct, of the existence of the tumor in a certain situation. It is important to remember that the division is not absolute. Some of the general symptoms may be produced to a greater extent by disease in one situation than in another, and so may have a focal significance (see p. 508).

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* See, on this subject, a valuable paper by Hale White, in the *Guy's Hosp. Rep.*, vol. xliii, 1886.

In most cases the general symptoms precede the local, and the onset of both is usually slow and gradual. Sometimes they are trifling compared with the size of the growth, and, in rare cases, they may be absent, or, at any rate, so slight as to have attracted no attention, and a tumor is found post-mortem, the existence of which was not suspected during life.

Of the general symptoms, *headache* is the most constant, absent only in very rare cases. It is usually constant, with paroxysmal exacerbations; less frequently it intermits. In character the pain varies much; it may be dull or acute; sometimes it is described as rending, tearing, stabbing, boring. Its severity is usually such that sleep is more or less disturbed by it, and in the acute paroxysms the mind may be unhinged by the intense agony. It is usually increased by whatever causes passive congestion of the brain, as a cough, or muscular effort. It may be general, felt equally in all parts of the cranium, or it may be felt chiefly in the front or in the back of the head, or on one side, or may even be referred to a limited area. The locality of the pain does not always correspond to the locality of the disease. It may be referred to the forehead, when the disease is in the cerebellum, or to one side when the disease is on the other. But when the tumor is at the surface of the brain, the pain often corresponds to the disease in situation. When the growth is in the white substance, the pain is often frontal, whether the disease is in the frontal or parietal lobe. When the disease is beneath the tentorium, the pain is usually occipital, and often seems to pass down the neck. Unilateral occipital pain usually corresponds in side to the disease. Percussion of the skull by gentle tapping often causes pain over the seat of the disease, and not elsewhere. Neuralgic pain in the region of the fifth nerve indicates irritation of the nerve, and is not a "general" symptom, but this pain, when in the first division of the fifth, often blends with headache on the same side. The local pain produced by superficial tumors is doubtless due to irritation of the meninges. The more general headache has been referred to the influence of the increased intracranial pressure on the sensitive membranes, but it is, perhaps, better to confess that there is no evidence as to the mode of its production (see p. 530).

Optic neuritis occurs in a large proportion (probably about four-fifths) of the cases of intracranial tumor, whatever be the seat or nature of the growth, whether it commences in the brain or membranes. Nevertheless it is certainly less frequent when the growth is in the membranes over the convexity, and merely compresses the brain, than when the cerebral tissue is actually invaded. The size of the tumor, *per se*, seems to have little influence, and the optic neuritis is certainly not produced by the mechanism of increased intracranial pressure. It has been produced by a tubercle of the pons no larger than a cherry, and was absent in a case which I watched throughout, in which a sarcoma the size of the closed fist, growing from the dura mater, had compressed but not invaded the brain, and must have raised the intracranial pressure as much as it is ever raised by the direct agency of an intracranial growth. It is probable that the neuritis is due to more than one mechanism, which varies in relative degree in different cases. One of these is the extension of a process of tissue irritation to the optic tract and nerves which, reaching the papilla, lights up a more considerable inflammation. Another is the distention of the sheath of the optic nerve by fluid from the subarachnoid space, containing, it may be, irritating products. A third is the meningitis which, as we have seen, often occurs in cases of intracranial tumor, and may extend directly to the optic nerves. Optic neuritis is usually a transient event in the history of intracranial tumor. The growth may exist and may cause symptoms for a considerable time, even for years, without any affection of the optic discs, and then neuritis may be rapidly developed, run its course, and pass into atrophy, and the symptoms of the tumor go on as before. Often, however, the occurrence of the neuritis coincides with an obvious increase in the other symptoms of the growth, and probably always indicates progress of the disease. In the majority of cases both are affected, one perhaps more than the other. Rarely the neuritis is unilateral, and this although the disease may be one which usually causes bilateral neuritis.

The course of neuritis varies considerably in different cases. It may develop rapidly and reach, in a few weeks, a considerable degree of intensity, with extravasations and great distention of veins, and then subside, leaving "consecutive atrophy." On the other hand, it may develop slowly and remain apparently stationary for months, or even for a year, before it passes away. The course of the neuritis is some indication of the course of the tumor. It is true that a rapid neuritis is sometimes developed in the course of a slowly-growing tumor, but a chronic form of neuritis never results from a rapidly-growing tumor. If the neuritis is acute and the tumor is not influenced by treatment the neuritis usually goes on to atrophy. It often does so also in the chronic form, but now and then a slight neuritis, slowly developed, may subside, although the tumor continues to increase. But if the morbid process within the skull can be influenced by treatment, the neuritis may subside and leave little trace of its presence, and the diminution in the intra-ocular affection may be the first indication of that which is taking place within the skull. For other particulars regarding the mechanism, symptoms and consequences of neuritis, the reader is referred to p. 561, but it may be again pointed out that sight may be unimpaired as long as the neuritis is moderate in degree, and that it often fails much more while the neuritis is subsiding than when the inflammation is in the active stage. The stage of atrophy is never reached until after many months, and this fact often affords very important indications. A tumor, for instance, may have caused marked symptoms for only a short time, and the condition of the optic disc may prove that the growth must have existed for many months before the symptoms were produced.

Mental Disturbance.—Stupor and coma are common as terminal phenomena in most forms of intracranial tumor immediately preceding death. Apart from this final state, during the course of the disease, psychical symptoms are not unfrequent. The most common form is simple mental failure, loss of memory, depression, sometimes with emotional mobility, now and then varied by excitement. This condition may accompany tumors in any situation. It is sometimes, in tumors of the mesencephalon, produced through the agency of internal hydrocephalus compressing the cortex. Occasionally there is more pronounced mental disturbance, hallucinations and delusions, which may even render it necessary to send the patient to an asylum. In a small percentage of post-mortems in asylums a cerebral tumor is found, often unexpectedly, to have been the cause of the mental derangement. In most cases in which the derangement is pronounced, the tumor is situated in the cerebral hemisphere, and when there are no other symptoms it is generally in the anterior portion of the frontal lobe. In some of these cases there is a peculiar childishness of action. As instances of these symptoms may be mentioned the case of extensive tumor beginning in the temporal lobe, in which the patient had vivid hallucinations that his wife, who had died two years before, was committing adultery in the ward before his eyes. In another case of glioma of the frontal lobe the only symptoms were headache, optic neuritis, attack of petit mal, and mental disturbance with childish acts. The patient would strip the bedclothes off other patients who were in bed, turn pictures to the wall, etc. It is important to remark, moreover, that cerebral tumor, like every other form of brain disease, may evoke in predisposed persons the manifestations of hysteria. Many errors of diagnosis have occurred in such cases; the unmistakable symptoms of hysteria have caused the physician to overlook the symptoms of organic disease.

Vomiting is a frequent symptom of tumor in all parts of the brain, but especially in tumors of the medulla oblongata, middle lobe of the cerebellum, and corpora quadrigemina, rather less frequent in tumors of the cerebellar hemispheres, the pons, the base (all parts), and the central ganglia. It is less common, occurring in about a quarter of the cases, when the tumor is situated in the cerebral hemispheres, white substance or cortex. In cerebellar tumors, vomiting, headache, and optic neuritis are often for a time the only symptoms.

Giddiness, constant or paroxysmal, attends tumors in various situations. It is sometimes produced through the agency of the paralysis of an ocular muscle disturbing the projection of the visual field, and so destroying the harmony between the various sensory impressions which regulate the centre for equilibration; less commonly it is due to disturbance of the auditory nerve. It occurs, however, in tumors of every situation, without any peripheral disturbance to which it can be referred. It is most severe and frequent in cases of disease of, or compressing, the pons, corpora quadrigemina, and cerebellum (especially the hemisphere) and middle cerebellar peduncle. In the latter case it is sometimes most intense, and is frequently associated with a tendency to a forced position of the head or body. It is less frequent in tumors of the cerebral hemispheres than in those of the central ganglia.

Affections of Speech.—A peculiar slowness of speech is sometimes met with in association with tumors of almost every position, but with especial frequency in tumors of the cerebrum. A tendency to separate the syllables is conspicuous in some cases of tumor of the pons. With mental dullness from tumors in any situation it is not infrequent for words to be clipped, or syllables run together in "confluent articulation." Marked difficulty of articulation occurs chiefly when the growth is situated in the pons or medulla, or in the posterior fossa of the base. It is also produced, now and then, by tumors of the cerebellum when these compress the pons or medulla. Aphasic difficulties of speech are chiefly due to tumors of the left cerebral hemisphere, cortex, or white substance; as a permanent symptom only when the growth directly damages the speech centres already described (p. 540). But it occurs also as a variable and intermitting symptom in disease of neighboring parts, even of the upper part of the central region, chiefly in association with convulsions, and probably as an effect of irritative inhibition. It is very rare in tumors of the central ganglia. I have once known very marked aphasic defect to be produced by a tumor of the pons (in a patient under the care of my colleague, Dr. Hughlings Jackson); the characters of the defect were so similar to those of disease of the motor cortex that they were probably produced by an upward inhibitory influence.

Motor Disturbance.—*Paralysis* occurs: (1) Under the form of ordinary hemiplegia, almost always gradual in onset, affecting the lower part of the face, the arm and the leg, from tumors situated in or pressing on the upper part of the pons, crus, or internal capsule.

(2) Partial hemiplegia, paralysis of the arm, or arm and face, less commonly of the leg, is usually the result of growths in or beneath or adjacent to the motor part of the cortex, and is often associated with convulsions beginning locally in the limb paralyzed. Both the local palsy and the local convulsion usually commence in the extremity of the affected limb, and have the same significance. The positions of the growths causing such limited symptoms are those already described in the chapter on Localization (p. 708).

When the tumor is seated below the hemispheres, in the crus, pons or medulla, the hemiplegia is often accompanied by paralysis of one or more cranial nerves on the side corresponding to the tumor, and opposite to the affected limbs, as described at p. 708.

Basal tumors only cause hemiplegia when they are seated, or extend so far back as to compress the crus, the pons or medulla; and the character of the hemiplegia is similar to that produced by tumors in these parts, but the nerve palsy is usually more extensive, and occurs earlier, than in the case of tumors within the brain substance.

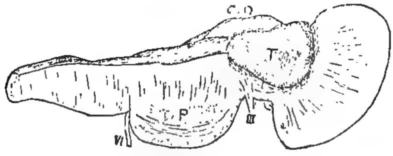
(3) The paralysis produced by intracranial tumors is sometimes not unilateral, but bilateral. This may result (*a*) in rare instances from symmetrical tumors on both sides of the brain, generally in the central ganglia, or (*b*) from a single tumor in such a position that it compresses the

motor tracts of both sides. This can only happen when the tumor is situated in or near the mesencephalon, where the two motor tracts are still contiguous, and it is generally due to a tumor in or compressing the pons or the medulla oblongata. In the latter situation the weakness may be chiefly marked in the legs, causing paraplegia, usually greater in one leg than in the other, rarely limited entirely to the legs, and distinguishable from that due to disease of the spinal cord by the affection of the cranial nerves. When the damage to the motor tracts is by pressure from a distance (as in the case of tumors of the cerebellum or corpora quadrigemina) the bilateral weakness is often slight in degree.

Contracture, persistent tonic spasm, with excess of myotatic irritability, is a frequent sequel to the paralysis in the limbs that results from cerebral tumor, and has no special significance. Confined to one limb it usually indicates that the disease is in or near the motor cortex. The chief difference from the contracture which follows other lesions is that it often accompanies, instead of following, the loss of power, doubtless because the damage to the motor tract is not only gradual but irritative in its character, and hence descending degeneration occurs the more rapidly. From the same cause the contractured muscles often waste early, the more irritative degeneration of the pyramidal tract extending to the spinal motor nerve cells (see p. 518). General tetanic rigidity, without paralysis, may occur from tumors of the pons or cerebellum; in the latter case there is occasionally retraction of the head, but as this is also produced by basal meningitis, it is probably due to pressure on the medulla.

Spontaneous choreoid or "athetoid" spasm also occurs in association with hemiplegic weakness and contracture, but less commonly than after some other cerebral lesions. It is accompanied by a slow spastic incoördination. It has been sometimes observed in cases of tumor of the parietal lobe, and of the neighborhood of the optic thalamus. One form of incoördination in the arm is not at all infrequent, in cases of tumor (especially tubercle) of the pons or crus, as in the case shown in Fig. 310. It is a wild, jerky incoordination, precisely resembling that which is seen in disseminated sclerosis.* The limb, apart from voluntary movement, is free from spasm. This symptom, in disseminated sclerosis, seems to be due to the wasting of the white substance of the nerve fibres, by which the conducting power of their axis cylinders is lessened unequally. The pressure of a tumor on the motor tract may cause a similar interference with structure, and the

FIG. 310.



Longitudinal section of pons (P) and left crus cerebri, showing a tubercle (T) in the crus, beneath the corpora quadrigemina (CQ). The patient was a child, and the first symptom was jerky incoördination of the right arm exactly like that of disseminated sclerosis, followed by palsy of the arm, then of the leg and face, then of the left third nerve (III), and later of the right also.

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* It is exceedingly probable that some cases published as examples of disseminated sclerosis in childhood have really been cases of stationary tubercle of the brain.

phenomenon, which is certainly identical in aspect, may be due to a similar mechanism in each case. A finer tremor is not an uncommon accompaniment of the movement of weak limbs, and has no special significance.

Unsteadiness in the upright posture, most marked on walking, is a common symptom in tumor of the cerebellum, especially of the middle lobe. The body sways like that of a drunken person, and the patient may tend to fall forward, backward, or to one side. The movements of the arms are usually steady.

A tendency to the assumption of forced positions, or to certain involuntary movements, is occasionally seen. When there is vertigo, the patient may tend to one side in walking, irrespective of the cause of the giddiness. A tendency to rotation on the horizontal axis of the body has been noted as an extremely rare symptom of tumor of the middle peduncle of the cerebellum. Rotation of the head to one side, with corresponding conjugate deviation of the eyes, occurs in some cases of cerebral tumor. Deviation toward the side of the lesion, so frequent at the onset of cerebral hemorrhage, occurs in tumors of various positions, although not frequently. Occasionally there is a deviation from the side of the cerebral lesion, due to irritation and spasm, when the disease is in the cerebral hemispheres, or to paralysis, when it is in the pons.

Convulsive attacks are frequent, and are of four chief forms. Two are of merely general significance, occurring, like headache and optic neuritis, with tumors of various seat, while two are of local significance, met with chiefly when the growth occupies certain situations. (1) Attacks of general convulsion with initial loss of consciousness, resembling the fits of ordinary epilepsy, occur in tumors of every part, but are not common when the medulla oblongata or cerebellum is the seat of the disease. They are more frequent in tumors of the posterior fossa of the base than in tumors of the anterior or middle fossa. The mechanism by which these convulsions are produced is uncertain. They are often ascribed to the general increase of intracranial pressure caused by the growth, on the ground that similar convulsions are produced in animals when the intracranial pressure is raised (to 130 mm. of mercury, Leyden). But it is scarcely justifiable to conclude that, because a rapid increase in the pressure causes convulsions, these are, therefore, thus produced by a slowly-growing tumor, and the explanation is not easy to reconcile with many clinical facts, which show, first, that large tumors do not necessarily cause convulsions, and, secondly, that even in the most severe form such convulsions may be produced when there is no increase of intracranial pressure. An instance of the latter is afforded by a case in which general convulsions persisted after other symptoms of syphilitic brain disease had been removed by treatment, and the patient actually died from the violence of the fits. The only brain disease discovered was a small, shrunken gumma, in the anterior part of the right frontal lobe.

(2) Attacks of brief loss of consciousness, resembling the *petit mal* of epilepsy, occur occasionally, but not frequently, with tumors of various situation, especially in the cerebral hemispheres. They are often associated

with the general convulsions just described, and, like them, are of only general significance.

(3) The convulsions may commence in a manner which indicates that the discharge begins deliberately at some one part of the brain, and remains limited to it, or slowly spreads to other parts. The characters and significance of the convulsions have been described at pp. 524 and 709. They are more frequently due to morbid growths than to any other lesion. The tumor is almost always situated on or near the motor region of the cortex. The occurrence of occasional general convulsions does not lessen the significance of those which commence locally. If the convulsion commences in the foot, the tumor is probably in the upper part of this region; if in the face, it is probably in the lower part; if in the arm, it may be in or near the middle region. Commencement in the hand has, however, been observed when the tumor is not contiguous to the central convolution, *e. g.* in the supramarginal convolution or even in the occipital lobe and cuneus. It is possible that in such cases the convulsions are produced through the agency of meningitis. Some fits may commence in one part, some in another. The convulsion is the result of irritation, which may be greatest now in one and now in another region which is being invaded. Moreover, a growth may be seated in one part, but the chief irritation may be in another part to which it is advancing. In these cases there is often paralysis in the part related to the seat of the tumor, which is of assistance in determining the significance of the various fits. Thus, in one case in which some fits began in the face, some in the arm, some in the foot, the latter only was permanently paralyzed, and the tumor occupied the highest part of the central convolutions.* Fits which begin locally often leave transient weakness in the part first convulsed, and this, if the attacks are frequent, may persist during the intervals, passing away when they cease. The frequency of these local fits is sometimes very great. In the case just mentioned a hundred or a hundred and fifty fits sometimes occurred daily, and in the course of eleven months the patient had 17,000 fits (carefully recorded). Occasionally local clonic spasm may continue for several hours or even days.

The local commencement is sometimes by a sensation, tingling, "pins and needles," or pain, in the hand or in the foot, as mentioned at p. 525. This sensory commencement in the limbs has the same significance as the motor commencement, and the part in which the sensory discharge begins is that in which the chief irritation exists; the *secondary* spasm is not of localizing significance. In rare cases a cerebral tumor causes fits which commence by a special sense warning. It is probable that, in these cases, the tumor is situated in the cerebral hemisphere, in, near, or beneath that part of the cortex in which the special sense centre is situated. Some instances of this are mentioned on p. 525.

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* A similar case has been recorded by Osler (*Amer. Journ. of Med. Sciences, 1885*, p. 31). The fits began in the left hand but the palsy was in the leg, and the tumor was just beneath the highest part of the ascending frontal convolution.

(4) Attacks of brief tetanic rigidity with retraction of the head have been observed in some cases of tumor of the cerebellum. It is uncertain whether they are due to the irritation of structures in the cerebellum or to pressure on the pons or medulla oblongata.

Sensory Symptoms—(1) *Loss.*—Motor hemiplegia from tumor is often accompanied with unilateral diminution of sensibility of various distribution and character, according to the seat of the disease, as described in a preceding section (p. 526). Well-marked hemianæsthesia, extending up to the middle line, and usually with impairment of all the special senses on that side, occurs in cases of tumor damaging the posterior part of the internal capsule and subthalamic region, a not uncommon seat of syphilitic growths. Affections of sensation are rare and trifling in tumors of the anterior and middle fossa, of the base, the corpora quadrigemina, and the cerebellum. Bilateral loss may occur, like bilateral motor palsy, from growths in the pons and medulla.

(2) *Sensory irritation*, pain, tingling, and other paræsthesiæ, often accompany the anæsthesia produced by cerebral tumor, just as spasm often accompanies the motor palsy, in consequence of the irritating character of the lesion. Pain in the limbs is most frequent in tumor of the central region, and is often felt in the limb which is chiefly paralyzed or convulsed. The pain may be increased by movement, active or passive, and local convulsions may be sometimes induced by movements that cause pain. Other paræsthesiæ are especially frequent in tumors of the pons and medulla.

Cranial Nerves: Olfactory.—Loss of smell has been noted in rare cases of tumor in various parts of the brain, but with singular infrequency in any part, even in tumor of the anterior fossa of the base. Its occurrence in tumor elsewhere, even in the cerebellum, has suggested that its loss may depend on an olfactory neuritis, such as affects the optic nerve, but this is a mere hypothesis at present unconfirmed.

Optic Nerve.—The frequency of optic neuritis renders impairment of sight a very common symptom of intracranial tumors, irrespective of their seat. Apart from this affection, which has been already considered, sight may be damaged by tumors situated in or near the visual path. The characters of the loss, according to the seat of the disease, have been already described (see p. 574). Symptoms of irritation of the optic nerve are extremely rare, except as the aura of convulsions.

Third Nerve.—Isolated paralysis of the whole of one or both third nerves occurs only in tumors of or adjacent to the crus cerebri, first on the side opposite to the hemiplegia. Both third nerves may be affected by an interpeduncular growth, as probably is the case shown in Fig. 263, p. 604. Partial paralysis usually involves chiefly the levator or the internal rectus.

The *fourth nerve* rarely suffers alone, and chiefly in tumors in or compressing the corpora quadrigemina.

The *sixth nerve* is frequently paralyzed alone on one or both sides by subtentorial disease, which exerts pressure, probably because its long and exposed course over the prominent part of the pons causes it to suffer very readily by pressure from above. It also suffers in tumors of the pons, medulla oblongata, and posterior fossa of the base, but then usually in association with other nerves, with the facial in disease of the pons, with the hypoglossal in disease of the medulla. In the former case it is affected on the side opposite to the limbs; in the latter, hemiplegia may be on the same or on the opposite side.

Combined paralysis of ocular muscles, slight and various, occurs in tumors of various situation, that exercise pressure on the base—tumors of the deeper parts of the hemispheres; corpora quadrigemina, pons, and basal tumors. All the muscles of one eye are only paralyzed by growths in the neighborhood of the sella turcica, which usually causes also loss of sight.

Conjugate deviation of the eyes and head toward the side of the lesion (paralytic) or from it (spasmodic) has been already described. Its characteristic is that the axes of the eyeballs remain parallel in all positions. Another condition sometimes confounded with this is the associated paralysis, described at p. 610, in which the external rectus on one side, and the internal rectus on the other, are affected together, so that the eyes cannot be moved toward the side of the lesion beyond the middle line. The variations in this symptom, and their localizing significance, have been already considered. Tumor is by far its most common cause. Defective movement upward or downward is sometimes observed in cases of cerebral tumor, but its exact significance is uncertain (see p. 609). Nystagmus is an occasional symptom of tumor in many parts, but is not frequent in tumors of the cerebellum. An unsymmetrical position of the eyes, one directed upward and inward, the other outward and downward, has been observed as an extremely rare symptom of tumor of the middle peduncle of the cerebellum. The pupils are often unequal, dilated, or contracted, but, alone, their condition is of little significance, except as evidence of some intracranial disease.

The *fifth nerve* is almost constantly affected in tumors of the middle fossa of the skull, frequently in tumors of the pons and of the posterior fossa, occasionally and in slight degree from growths in the cerebellar hemisphere which exert distant pressure. Both the sensory and motor parts of the nerves are usually affected (especially by tumors outside the pons); sometimes the sensory only, rarely the motor part alone. All divisions of the nerve may be involved or only the upper and middle. Neuro-paralytic ophthalmia is frequent from basal tumors, which damage the Gasserian ganglion, but the eye often escapes when the tumor is situated within the pons. The gradual damage of the sensory part usually causes irritation, and neuralgic pain, sometimes most intense and accompanied by hyperæsthesia, precedes loss of sensibility. Taste is also often lost.

Facial Nerve.—Paralysis of all parts supplied by the facial nerve, with the reaction of degeneration in the muscles, occurs only in tumors of the pons or posterior fossa, very rarely, and in slight degree, from the distant pressure of a cerebellar tumor, and is associated with paralysis of the limbs on the opposite side. Sometimes both facial nerves are paralyzed.

The significance of association of paralysis of the facial and other nerves is described at p. 657.

Auditory Nerve: Hearing.—Deafness is an occasional symptom in tumors of the lower part of the pons, of the medulla oblongata, and of the posterior fossa of the base. Its localizing significance has been mentioned at p. 674. Bilateral deafness has been twice produced by growths in the corpora quadrigemina which had damaged the upper layer of the tegmentum, in which probably the auditory tract passes up to the hemisphere. Subjective sensations of hearing have been produced by tumors of the pons and base, and also by tumor of the temporal lobe. In the former case they are referred to the ear of the same side, in the latter to the opposite side.

The Hypoglossal Nerve.—Besides the general relations of palsy of the tongue mentioned at p. 704, the nerve may be paralyzed on the side opposite to the hemiplegic limbs, in tumors of the posterior fossa of the base, sometimes in tumors of the pons, and especially in those of the medulla oblongata. It is sometimes an early symptom of a morbid growth on the front of the occipital bone. In these cases there is often conspicuous wasting of the paralyzed half of the tongue.

The Spinal Accessory nerve is also occasionally paralyzed from tumors in or near the medulla. Only those outside the medulla impair the action of the muscles of the neck, but more frequently the vocal cord on one side, less commonly on both, is paralyzed. The palate usually, and the tongue often, suffers at the same time. Bilateral palsies are occasionally met with, and the lips may also be involved, causing symptoms similar to those of progressive bulbar paralysis. Very rarely the same combination of symptoms has resulted from symmetrical tumors of the cortex in the lower part of the central convolutions.

The *pulse* is often infrequent in tumors anywhere within the skull, and the symptom has no special significance. *Respiratory* disturbance is chiefly met with in growths near the medulla.

The *Sphincters* are not often paralyzed from intracranial tumor, and the loss of power over them is of no special significance. Untimely evacuation of bladder and of rectum is, however, very common in association with the mental apathy and dullness which are so common in tumors of all parts. An increased secretion of *urine*, and the presence in it of sugar and of albumen, have been met with in rare cases of tumor in various parts of the brain; glycosuria occurs most frequently in association with tumors of the pons and medulla, but not so frequently as to have any strong significance apart from other symptoms.

Intracranial tumors rarely give any other evidence of their existence than that which is afforded by the symptoms already described. Only those growths which spring from or invade the bone of the skull are recognizable externally; swellings on the surface of the skull occur in rapidly growing tumors springing from the dura mater. Growths in the bone of the middle fossa of the skull are sometimes recognizable on the under surface, in the palate. More commonly, tumors of the anterior or middle fossa invade the orbit, and cause bulging forward of the eyeball.

Regional Symptoms.—The symptoms of tumors in various parts of the brain are those that have been described in the chapter on Localization, p. 708, and need not be here repeated in detail.

The symptoms of tumors of the base, however, need brief enumeration. Growths in the *posterior fossa* cause nearly the same symptoms as tumors of the pons or medulla, subject to the same variations according to their seat. The most important difference is that the cranial nerves suffer earlier than the motor tract, and the combination of the nerves affected is somewhat different. Thus the facial and auditory suffer together from external pressure, rarely from internal disease. The sixth (abducens) is paralyzed without the conjugate internal rectus, which suffers with it in disease of the centre. The combination of palsy of one-half of the tongue, palate, and glottis is especially frequent from tumors beside the medulla. Neuro-paralytic ophthalmia, from implication of the Gasserian ganglion, sometimes occurs, but less frequently than in disease of the middle fossa. Lastly, pressure on the middle peduncle of the cerebellum often gives rise to intense vertigo, and an inclination to deviate to one side in walking. Convulsions occur in cases in which the pons is compressed, and are rather more frequent than when the tumor is in the substance of the pons.

Middle Fossa.—The course of the fifth nerve, and the position of the Gasserian ganglion, render it very liable to suffer in tumors of the middle fossa, so that its irritation and paralysis, on one side only, with neuro-paralytic ophthalmia, is a characteristic symptom of disease in this region. Now and then, from extension backward of the growth, or of meningeal irritation excited by it, the facial and auditory nerves sometimes likewise suffer. Other nerves are rarely affected unless the growth is situated near the sphenoidal fissure, when those to the eyeball may be involved. Hemiplegia is rare and slight, and is produced by the extension of the growth to the neighborhood of the crus. Convulsions are extremely rare, and it is not common for these to be conspicuous mental symptoms.

Anterior Fossa.—Tumors are rarely limited to the anterior fossa of the skull, and the chief symptoms they produce is by their extension beyond it, backward to the optic nerve (unilateral loss of sight), the chiasma (temporal hemianopia, once transverse hemianopia, probably from damage to all the higher fibres of the chiasma), and even back to the crura, causing slight paralysis of the limbs, usually on one side only. From compression or invasion of the frontal lobes of the brain, mental symptoms occasionally result, and a peculiar childishness has been thought to have some diagnostic value.

Tumors of the pituitary body cause, usually, vague symptoms resembling those just mentioned, with very frequent failure of sight, and indications of a lesion of the chiasma are present in many cases. Sometimes, however, the tumor causes very slight symptoms, and may run an almost latent course, and this although the size attained is considerable.*

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* *e. g.* case recorded by Cunningham, *Journ. of Anat. and Phys.*, July, 1879.

Basal tumors sometimes extend over a large area; occasionally a growth has occupied almost the whole of one-half of the base of the skull. The symptoms, when the growth has been soft, have been in several cases singularly few, and a correct diagnosis impossible.

Multiple cerebral tumors sometimes cause only "general" cerebral symptoms. More often one growth causes conspicuous disturbance, and there is no indication during life of the existence of the others. Occasionally two or more growths exist in the same part of the brain and cause extensive but connected symptoms. This is especially the case with tumors in the posterior fossa of the base, where multiple growths are common. Now and then tumors in different parts of the brain cause symptoms so distinct and disconnected as to afford clear evidence of their separate causes.

Course.—The great characteristic of cerebral tumors is that their symptoms are gradual in onset and slowly progressive in their course. The earliest symptoms vary much, according to the seat of the tumor. In most cases the general symptoms precede the local, and no initial symptom is so frequent as headache. Optic neuritis is sometimes a very early symptom, especially in disease of the cerebellum or corpora quadrigemina. In disease of the hemispheres it is often late. Convulsions now and then occur before any other disturbance has raised suspicion of brain disease, and this is especially true of the local convulsions from cortical tumor. The early fits are sometimes excited by an accessory influence, as digestive disturbance (p. 735). Symptoms of irritation or paralysis of some cranial nerve often occur early, but hemiplegic weakness very rarely precedes headache.

Although the course of the symptoms is usually slow and gradual, it is rarely uniform, and exceptions to the gradual course are occasionally met with. In slowly-growing tumors the progress may intermit, stationary intervals alternating with periods in which the symptoms increase. Sometimes the pressure effects are produced rapidly; in a few days, for instance, a cranial nerve, as the third or facial, which before was unaffected, may become completely paralyzed. In these cases it is probable that a neuritis is induced by the pressure, just as a tumor compressing the spinal cord often causes paraplegia by setting up myelitis.

In other cases, however, a rapid increase of symptoms may result from local meningitis excited by the growth. This is to be suspected if the symptoms increase rapidly in their range; and especially if the increase in symptoms is attended with febrile disturbance; and it is certain if symptoms so arising subsequently lessen. Often, as already stated, the traces of past meningitis are distinct after death, but if a long time has elapsed, its indications may not be discoverable, although the symptoms may have been distinct in the past. For instance, in a case of tumor occupying the third ventricle, and compressing the corpora quadrigemina, the patient suffered, six months before death, from an attack attended with severe occipital headache, vomiting, and paralysis of the right fifth nerve, the left half of the face and tongue, the right half of the palate, and right vocal cord, and much difficulty in swallowing. These symptoms lasted for a few weeks, and then passed away almost entirely, slight weakness of the right masseter alone remaining. In this case no evidence of meningitis could be discovered at the post-mortem examination six months later, but no other mechanism could produce transient symptoms of damage to nerve roots extending as far back as the medulla oblongata.

Hemiplegic weakness often follows unilateral convulsions, as a transient effect of the "discharge," and is most marked in (and sometimes limited to) the limb in which the convulsion commenced. It may also follow, apparently as the result of inhibition, sensory discharges which do not cause spasm. But sudden and persistent hemiplegia may occur in the course of cerebral tumors, without preceding convulsion. In rare cases it is the result of associated or independent vascular disease,* or of vascular occlusion from the effect of the growth on adjacent vessels. It may also be the result of a hemorrhage into the growth, such as is common in the case of soft glioma. This is also rare, because the hemorrhage is into, and not outside, the tumor, and does not cause paralysis unless the growth occupies a motor region, but a growth so situated has usually caused paralysis, from its size, before the hemorrhage occurred. Lastly, sudden hemiplegia occasionally occurs in the course of a cerebral tumor, without there being any mechanism discoverable after death by which the sudden onset can be explained. The fact is important, its explanation obscure.†

In the vast majority of cases, the course of an intracranial tumor is progressive, and ends in death. But the rapidity of progress varies much, according to the character and position of the new growth; the former determining the rate of growth, the latter the symptoms which the increase in size of the tumor produces. Thus a tumor of the same kind and size, in the anterior part of the cerebral hemisphere, may cause far slighter symptoms than if seated in the pons. Other things being equal, the slower the growth of a tumor the slighter are the symptoms, since the slower the pressure the better is it borne.

The duration of the symptoms varies from a few weeks to many. In a case recorded by Osler the convulsions produced by the tumor continued during fourteen years. In the majority of cases the duration is between six months and two years. The most rapid cases are tubercle, soft sarcoma, cancer, rapidly-growing gliomata, and syphiloma. The cases of longer duration are tubercles, firm sarcoma, slowly growing glioma, and the various hard tumors, fibroma, enchondroma, etc. Tubercles furnish some of the cases of shortest and also of longest duration.

The most common causes of death are: (1) Exhaustion, induced especially by the violence of the pain, and the interference with nutrition entailed by the frequent vomiting, and the difficulty of feeding occasioned by mental dullness or by dysphagia. (2) Coma coming on gradually, and usually due to the general increase of intracranial pressure, sometimes also produced by hydrocephalus. The lower centres participate in the apathy of the higher, and from the lowered sensibility of the respiratory centre, mucus accumulates in the lungs, and the resulting interference with respiration is generally the immediate cause of death. (3) Death is sometimes sudden. This occurs most frequently in tumors of the medulla oblongata, almost as frequently in tumors of the cerebellum, next in tumors of the central ganglia, occasionally in tumors of the cerebral lobes, corpora quadrigemina, pons, and base, but rarely in tumors of the cortex (Bernhardt). It is probably to be referred to pressure on the respiratory or cardiac centres of the medulla. Occasionally it seems to be produced by the mere intensity of headache, conjoined with syncopal exhaustion from vomiting; sometimes it occurs from the violence and frequency of general convulsions, as in a case already mentioned. (4) Lastly, death may occur from some other effect of the cause of the tumor. The mechanisms of the class are extremely varied. Some are intracranial. Tubercular meningitis, for instance, is a frequent cause of death in tubercular tumors of the brain, even when the position of the latter makes it certain that the meningitis is in no way the direct result of the presence of the growth. A patient with syphilitic tumor may die, not from this, but from the effects of syphilitic disease of the vessels. Other fatal effects of the cause of the tumor are extracranial. In cases of tubercular tumor, it is frequent for death to result from the general disease. When a cerebral tumor, cancer, or sarcoma is secondary to such disease elsewhere, the latter, and not the former, is occasionally the cause of death. Lastly, the subjects of very chronic tumors not unfrequently die from intercurrent, unrelated diseases.

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* As in the case of a man who had a firm tumor, three-quarters of an inch in diameter, below the floor of the left lateral ventricle, and had independent hemorrhages in both right and left corpora striata (Mitchell, *Edin. Med. Journal*, Nov., 1883).

† I have elsewhere recorded some cases illustrating the phenomenon, "On Sudden Paralysis in Cerebral Tumor," *Brain*, vol. i, 1879, p. 48.

But cerebral tumors are not invariably fatal. Syphilitic growths are more amenable to treatment than any other form of intracranial disease due to the same cause; the symptoms often pass away, and usually lessen, the degree of improvement depending on the extent to which they are due to interference with function by pressure, and not to actual destruction of nerve elements. Symptoms of irritation, such as convulsions, may, however, persist in spite of treatment, the nerve elements retaining the functional disposition resulting from the irritative damage, although the cause of this has been removed. Tubercular tumors may also cease to grow, and, as already stated, may become encapsuled. It is probable that they do not undergo actual reduction in size. Hence, in the best result, the symptoms cease to increase, and may even undergo some subsidence from the recovery of the nerve elements damaged only by irritation, but there is nothing like the amount of recovery seen in cases of syphilitic tumor. When arrest is once obtained, the symptoms may remain stationary for an indefinite period. It is certain that a similar arrest occurs in other tumors, although far less frequently. In such a case, degenerative changes, often calcareous, may take place, and an inert mass remains, giving rise to no symptoms except those which result from the damage caused during its active growth. For instance, I have recorded elsewhere * the case of a girl of fifteen who suffered from left hemiplegia, defect of sensation, left hemianopia, headache, and double optic neuritis, all of gradual onset in the course of three months. Under treatment, first the neuritis subsided, then the headache, then the leg improved, and lastly the arm, but with the development of a spastic state in the latter, which has continued with the hemianopia; otherwise she has been now for six years perfectly well. In this case it is probable that tubercular tumor existed in or near the optic thalamus. In another case,† a girl of twenty-three suffered from severe headache, double optic neuritis, slight bilateral weakness in the limbs, and paralysis of the upward movement of both eyes, cycloplegia, vomiting, and some opisthotonic convulsions. All the symptoms disappeared except the headache, and for three years there was no recurrence; she then died suddenly. A tumor in the corpora quadrigemina or anterior part of the middle lobe of the cerebellum was assumed to exist, and a small degenerated growth was found in the front of the valve of Vieussens.

Diagnosis.—The problem of the diagnosis of a case of cerebral tumor includes four questions. (1) Is there organic disease? (2) Is it a tumor? If so, what are (3) its seat, and (4) its nature? Of these questions, to the first an answer can almost always be given, to the second generally, to the third often, and to the fourth sometimes. (1) As evidence of organic disease the focal symptoms are of more importance than the "general" (diffuse) symptoms. Of the latter, optic neuritis is, alone, the most significant (although not, in itself, conclusive); next, causeless vomiting; while headache, giddiness, and general convulsions are of significance chiefly when combined with other symptoms, because they are so frequently due to other causes. Convulsions beginning locally are of the nature of "focal" symptoms, but are not alone conclusive of the existence of organic disease, since they may occur in idiopathic epilepsy. Both headache and giddiness are extremely common without organic disease; nevertheless, headache is a suspicious symptom if so severe as to keep the patient awake at night, and severe vertigo is also suspicious if there be no indication that it depends on disturbance of the labyrinth. As a rule, other symptoms exist to give significance to those which, alone, are equivocal. Two conditions are most likely to give rise to diagnostic error. One of these is the presence of diffuse symptoms, due to a blood state, which may simulate those produced by intracranial tumor. Headache and optic neuritis may be produced together by three general conditions—anæmia, kidney disease, and lead poisoning. In the first of these the poverty of blood is always extreme; other symptoms of anæmia are obtrusive; the patients are usually young girls; optic neuritis develops with a rapidity unusual in tumor; all focal symptoms are absent;

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* "Medical Ophthalmoscopy," Case 2, p. 282 (2d edit.).

† Loc. cit., Case 52, 2d ed.

‡ For the use of the word "organic disease," see p. 2.

and the headache and neuritis rapidly subside under the influence of iron, especially if the patient is kept at rest in bed, and is well fed with easily-digested food. In kidney disease, in which there is conspicuous neuritis, this is rarely very great in degree, and a sufficiently minute scrutiny of the retina will usually reveal some of the characteristic degenerative changes. The meaning of these is not open to doubt if the neuritis is slight. (Wide-spread and intense papillitis may leave, near its margin, indications of degeneration, in white spots, which then and there have no significance.*) The urine contains not only albumen (which may be present also in cerebral tumor) but also casts, while the tension of the pulse, and hypertrophy of the heart, indicate a profound systemic affection. Attention to these symptoms, and to the absence of signs of focal disease, will usually prevent an error as to the nature of the case. The cerebral symptoms which occur in lead poisoning—headache, optic neuritis, delirium, and convulsions—would be very liable to mislead if it were not that they are almost invariably preceded by other symptoms of saturnism, too conspicuous to be overlooked, and too distinctive to mislead.

The second condition likely to give rise to error is that in which a partial functional affection of the brain causes symptoms which simulate those of focal disease. Hysterical hemiplegia, spasm, contracture, or anæsthesia, may be thought to be due to an organic lesion; the more readily, if attended, as they usually are, by headache, or, as they sometimes are, by vomiting. The principles of the distinction of these *simulacra* from the diseases they imitate are considered at length in the chapter on hysteria. The unilateral affections possess characters of their own, which differ from those of organic disease; the more sudden onset after emotional disturbance or a hysteroid convulsion, and the absence of unilateral alteration of the reflexes and of optic neuritis, usually enable a confident diagnosis to be made. Hemianæsthesia, involving the special senses, is one of the rarest effects of cerebral tumor, and is absolutely unknown from this cause without associated loss of motor power. The greatest difficulty is presented by cases in which the imitative tendency of hysteria has been evoked by the witnessed symptoms of a case of actual cerebral tumor. This is sometimes seen in a child or girl who has watched the symptoms in a genuine case, and in whom analogous disturbances are developed under the influence of the mysterious mimetic tendency. But the nature of the case may usually be recognized by the absence of all symptoms of organic disease which are beyond the range of the will, the mode of development of the subjective symptoms, the difference they always present from their organic prototypes, and the causal influence which is conspicuous. A mistake is rarely made except by those unfamiliar with the mimetic characteristic of hysteria, or whose judgment is clouded by sorrow and alarm.

Much more frequent is the converse error; the symptoms of a tumor are ascribed to hysteria. Sometimes this is done without a shadow of excuse, merely because the patient is of the female sex. More often the fact that indications of hysteria *are* present is allowed to dominate the diagnosis. In young women, organic disease of every kind is apt to excite hysterical disturbance, especially convulsion, and this is eminently true of cerebral tumor. Error in diagnosis is readily avoided by attention to the simple rule, which, mentioned already more than once, cannot be too often repeated, that present symptoms of hysteria should never be allowed to influence the diagnosis until all indications of organic disease have been sought for and excluded. In cerebral tumor such symptoms are never absent.

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* Compare "Med. Oph.," plate viii, Fig. 2.

When convulsions are the chief symptom, the disease may be confounded with idiopathic epilepsy. The mistake is chiefly likely to occur in cases of slowly-growing tumor in or near the central convolutions, as in one recorded case, in which the fits recurred during fourteen years and there were long periods of freedom. The fits which thus recur almost always begin locally, but local commencement, while it suggests organic disease, does not prove its existence, since, as just stated, the discharge sometimes begins locally in idiopathic epilepsy. The most important indication is the presence of other symptoms besides the convulsion. Occasionally, in the absence of other symptoms, causal indications may be allowed weight; *e. g.*, a history of inherited tendency to epilepsy on the one hand or of a tumor-causing dyscrasia, such as syphilis, on the other.

It has been said that the existence of organic disease can *almost* always be recognized. In rare cases, the presence of a tumor has been unsuspected, until revealed by a post-mortem examination. In most of these it is probable that unequivocal symptoms would have been detected by sufficiently complete and repeated examination. For the most part, these unsuspected tumors have been found in patients who had been suffering from some other grave disease, in the presence of which the symptoms of the tumor were overlooked or misinterpreted.

(2) If the symptoms are such as to indicate the presence of organic disease, the second question presents itself—is the disease a tumor? Organic diseases may be divided into two great classes, those of sudden, and those of more or less gradual development. In each class the symptoms correspond, in their onset, with the character of the lesion. Cerebral tumor falls into the second class and all vascular lesions into the first. In the rare cases of tumor in which sudden symptoms occur, these are always preceded by others of gradual development. Hence the fact that the symptoms are never sudden in their onset, never attain a high degree in the course of a few hours, excludes at once the frequent vascular lesions, cerebral hemorrhage and acute softening of the brain. We have, therefore, only to consider the distinction from diseases in which the morbid process, and the accompanying symptoms, are developed more or less gradually. In some of these the process and symptoms are diffuse, as in general paralysis of the insane, and chronic cerebritis; in others they are focal, as in abscess and sclerosis, and local neural degeneration; in others again they are mixed, as in meningitis. Aneurism of a cerebral artery stands in a different relation, since in this there is actually an intracranial tumor.

Cases of general paralysis of the insane can only be confounded with the rare cases of cerebral tumor in which symptoms such as mental failure, general weakness, and slow speech exist alone. The absence of headache, optic neuritis, vomiting, and the presence of the tremor in the muscles of the lips and face, and of expansive delirium, will generally suffice for the diagnosis.

Chronic cerebritis is a rare disease, which causes diffuse symptoms identical with those that result from cerebral tumor—severe headache, vomiting and optic neuritis ; in the early stage these are indistinguishable from the diffuse symptoms of a tumor, but if months pass, and no focal symptoms are developed, the absence of a tumor may be suspected, but can never be a matter of certainty.

Of focal diseases, intracranial aneurism, which causes an intracranial "tumor," produces symptoms often closely resembling those of a growth, and the only absolute distinction is the presence of an audible aneurismal bruit, distinctly produced within the skull. But the existence of an aneurism may be suspected if the symptoms indicate a tumor in the situation of a vessel, and if a cause of aneurism can be traced. The question is considered fully in the chapter on intracranial aneurism.

Abscess of the brain sometimes causes symptoms which closly resemble those of tumor. Headache, vomiting, mental dullness, and optic neuritis are common to the two diseases, but the last is rather less common in abscess than in tumor. Focal symptoms are also comparatively rare. Slowly progressing symptoms, of uniform course, are characteristic of tumor, while in abscess there is usually a rapid development of acute and grave cerebral disturbance, after a "latent" period, in which symptoms are trifling or absent. The presence of a cause for abscess (previous injury, ear disease, or suppuration elsewhere) increases the probability of suppuration, but it must be remembered that injury may cause either a tumor or an abscess. The points of distinction between the two are described more fully in the chapter on abscess of the brain.

Acute meningitis can rarely be confounded with cerebral tumor. The acute onset and rapid course of the symptoms, and the presence of fever, are sufficient to distinguish acute inflammation from growth. The only difficulty arises in cases of tubercular meningitis in which tubercular growths in the brain coexist but cause few symptoms, before the onset of the meningitis. One important indication in such cases is the degree of optic neuritis. That which results from the meningitis alone is rarely considerable in degree, and if the swelling of the papilla is great, and hemorrhages exist—especially if such a change is found soon after the onset of the symptoms of meningitis—it is probable that there are tubercular growths, the more so if headache or vomiting had long preceded the onset of the symptoms of inflammation. When symptoms of any tumor develop rapidly, in the course of ten days or a fortnight, a doubt may be for a time unavoidable whether the disease is tumor, or meningitis running a subacute or pyrexial course.

Occasionally an acute increase (or even apparent onset) of the symptoms may be occasioned by exposure to some general or special morbid influence—cold, the heat of the sun, or a blow on the head—an influence which may conceivably excite meningitis. The nature of the case may often be decided by the indications just mentioned, preceding headache, or a considerable degree of optic neuritis. If these are absent, meningitis is more probable than tumor. In children, tubercular tumors may run an almost latent course for a time and may then cause symptoms so rapidly as to render the diagnosis a matter of great difficulty. In some cases a comparison of the relative degree and extent of the symptoms may help the decision. For instance, an ill-nourished child, five months old, came under treatment for right-sided weakness and rigidity, with complete paralysis of the left third nerve. These symptoms were said to have come on a week before, and the child was thought to be suffering from tubercular meningitis. But it was unlikely that so extensive a process as meningitis would completely paralyze one third nerve and leave the other cranial nerves unaffected. The symptoms pointed rather to a focal lesion of the left crus cerebri, probably a tubercular tumor, running at first a latent course; the diagnosis was verified a few days later. It must be remembered also that a tubercular tumor and meningitis may coexist. In such cases time alone can render the diagnosis approximately sure. If symptoms continue for four or six weeks there is almost certainly a tumor.

Chronic meningitis is exceedingly rare except as a result of syphilis or alcoholism. In the latter case the symptoms are diffuse—headache, mental dullness, and usually delirium, rarely slight optic neuritis. The headache may be inconspicuous, but the alcoholic history and marked alcoholic tremor are sufficiently distinctive. Syphilitic meningitis is often indistinguishable from tumor, but the absence of focal symptoms, except paralysis of cranial nerves, suggests meningitis.

Only those cases of cerebral tumor in which there is jerky incoördination can be confounded with disseminated cerebro-spinal sclerosis. A sufficient distinction is afforded by the fact that in sclerosis the incoördination is bilateral; in tumor it is always unilateral; in the former there is neither headache, vomiting, nor optic neuritis. In fact, the symptoms of tumor are generally so distinct that only ignorance of the fact that such disease may cause this form of incoördination can cause any doubt to be felt as to the nature of the case.

(3) If the disease is a tumor, the next question is, where is it? The answer depends on the symptoms present, the localizing significance of which has been already described, and need not be here recapitulated. It is important to remember that the diagnosis must depend, as a rule, on the grouping of symptoms rather than on the presence or absence of any one symptom. There is hardly any symptom which may not be absent, wherever a tumor is seated, and almost every individual symptom may be produced by disease in more than one position. It must also be remembered that, although the general region in which the growth is placed may be determined in the

majority of instances, it is not often that its exact situation can be confidently affirmed.

(4) The exact nature of the tumor can sometimes be determined with a high degree of probability, now and then with practical certainty. Much more frequently hardly more than a guess can be made—a guess that is almost as likely to be wrong as right. The most important indications are the following:—

1. The presence of morbid growths elsewhere, the nature of which can be determined. When symptoms of cerebral tumor are consecutive to an infecting growth elsewhere, *e. g.* cancer of the mamma, etc., it is practically certain that the intracranial tumor is of the same nature. In rare cases the presence of an hydatid tumor in another part indicates that the cerebral tumor is of the same nature.

2. The presence of a distinct general disease of which intracranial tumor is a common consequence. These diseases are tubercle and syphilis. In adults, the signs of phthisis are rarely absent when there is tubercular tumor of the brain, and their presence is strongly in favor of—their absence against—the tubercular nature of the cerebral growth. In children, however, signs of tubercular disease elsewhere are often wanting, and their absence is of much less significance than their presence. The same is true of a family history of phthisis and tubercular disease. A physical configuration, such as often coexists with a tubercular tendency, is also in favor of the tubercular nature of the growth. The symptoms or history of syphilis, congenital or acquired, renders it highly probable that the tumor is of syphilitic nature. The absence of a history of constitutional syphilis, if the patient has had a chancre, should not receive too much weight, because it is not uncommon for the secondary symptoms to have been absent or overlooked. Further, in an adult, the possibility of the syphilitic nature of the disease cannot be excluded, unless we can feel sure that the patient has never been exposed to the possibility of infection, that the patient (if a woman her husband also) has never had impure sexual intercourse. Undoubted syphilitic disease is occasionally met with, where there is no history or indication of primary or secondary disease, the former having been unnoticed, the latter absent or disregarded. But in both cases, of tubercular and syphilitic indications, the diagnosis is a matter of probability only, since a cerebral growth of different nature sometimes coexists with either diathesis.

3. The sex of the patient affords little indication of the probable nature of the tumor, and that afforded by age is very limited. If the patient is under fifteen, and presents no indication of inherited syphilis, the tumor is certainly not of syphilitic nature. If the patient is an adult, and presents no signs of phthisis, it is very unlikely to be tubercular.

4. The help afforded by the seat of the tumor is also small, and is practically limited to the following facts. *Cæteris paribus*, if the disease is within the cerebellum or pons there is some probability that it is tubercle or glioma; it is unlikely to be syphilitic if in the cerebellum, but it may be if in the pons. If in the cortex, the probability that it is syphilitic is consider-

able, but is small if it is situated in the deeper parts of the hemisphere. A tumor outside the brain tissue is probably sarcoma.

5. The course of the tumor is sometimes suggestive. A very slowly-growing tumor in the hemispheres is probably glioma, and if the development of the symptoms is extremely gradual, it is most unlikely that the growth is syphilitic. A tumor which grows rapidly at the onset and then becomes stationary is probably tubercular. The occurrence of a sudden apoplectic seizure of moderate severity in the course of the symptoms is rare except in glioma.

6. The effect of treatment is of diagnostic value only in the case of syphilitic and tubercular tumors. A disappearance of symptoms (especially headache, optic neuritis, and recent paralytic symptoms) under iodide of potassium and mercury, makes it highly probable that the tumor is syphilitic. Improvement under general tonics is some evidence that the tumor is tubercular, but the value of this indication is at present somewhat dubious, since the nature of most tumors which thus improve is uncertain, and it is possible that other than tubercular growths may be thus influenced.

7. Multiplicity of growth gives little indication of nature, since tubercular, syphilitic, gliomatous and cancerous tumors are all often multiple.

Prognosis.—Only when the tumor is of syphilitic nature can a reasonably good prognosis be given. Even in such a case it is only the disappearance of the diffuse symptoms, and of the recent paralytic symptoms, that can be confidently predicted. Paralysis of more than a few months' duration may persist, although the growth is removed; and convulsions sometimes continue. Happily it is by no means uncommon for all symptoms to pass away. In tubercular tumors arrest is occasionally obtained, and the patient may live on indefinitely; the symptoms in such a case may lessen slightly, but those that have attained a considerable degree usually persist. In all these cases, a diminution in the headache and subsidence of an optic neutritis which is moderate in degree (which has not, as it were, exhausted itself by its violence) usually precede other indications of improvement, and are therefore distinctly favorable indications. In other forms of tumor the prognosis is very grave. As a rule, they progress to a fatal termination, and the probable duration of life can only be estimated from the rate of the progress in the part. It is likely, however, to be longer in tumors of the cerebrum or cerebellum than in those of, or pressing on, the pons and medulla. Considerable mental dullness, obstinate vomiting, severe and frequent general convulsions, apoplectic seizures, and rapidly-developed and intense optic neuritis, are all indications which render the prognosis grave as to the near future. On the other hand, the absence of these symptoms indicates, other things being equal, that the danger is more remote. Very chronic optic neutritis is of especial significance as an indication that the course of the disease will be slow and prolonged. It is probable that, in most forms of tumor, arrest of growth now and then occurs, but these are exceptions too few and far between to justify, in any given case, more than the dimmest ray of hope.

Treatment.—The treatment of new growths, in such a position that they are beyond the reach of the knife, and of such a nature that they cannot be influenced by drugs, is always a sufficiently gloomy subject, and not least so when they are seated in an organ like the brain, in which they cause peculiar and varied suffering, and in which their development, even to a moderate degree, is rarely compatible with life. Too often all that can be done is to afford some slight relief to symptoms, and even the possible relief is often trifling in degree. The only case in which a considerable effect on the growth can be produced by drugs is that of syphilitic tumor. Whenever there is even a possibility that the tumor is syphilitic, iodide of potassium should be given in increasing doses up to half a drachm three times a day, followed, if the effect is inadequate, by mercury.

If it is probable or possible that the tumor is tubercular, general tonics are of paramount importance, and of these cod-liver oil, iron, and an inadequate supply of food, are more often distinctly beneficial than any other agency. Cod-liver oil is of especial importance. Fresh country air is very useful, but does not, alone, exert such an influence as do tonics. I have more than once known the symptoms of a cerebral tubercle, steadily increasing under country air and abundant food, become permanently arrested when the patient was taken into a London hospital and cod-liver oil and iron were given. A similar tonic treatment is indeed desirable in all cases, for arrest of symptoms occasionally occurs under its influence when there is no reason to think that the tumor is tubercular. It is quite possible that tonics may influence some other tumors in the same way. There is no evidence that any other drugs are capable of arresting the progress of morbid growths. Perhaps arsenic and phosphorus, from their marked influence on some nutritive processes, deserve further trial.

The problem, then, in too many cases, is the relief of the symptoms which we are powerless to arrest altogether. Those that depend on the destruction of nerve elements by pressure or invasion, the gradual palsies, no agent can prevent or modify. But those that depend on meningitis—which extend beyond the original range of mischief, and are characterized by subacute onset, with increased headache and vomiting, and often by fever—may sometimes be relieved by counter-irritation and by leeches, if the patient is in a condition to bear these. Of the other symptoms of tumor, headache is one which often calls urgently for attempts at its mitigation, since it often renders the patient's life one of almost continuous agony. Relief can usually be obtained by the hypodermic injection of morphia, but the ready habituation to the narcotic renders its use admissible only in the most acute paroxysms. Indian hemp is more often useful than other sedatives except morphia. Sometimes counter-irritation to the neck gives relief to occipital pain, but no application is so effective as ice, which often lessens not only the pain but the vomiting and convulsions. For the latter bromide of potassium is rarely useful. Chloral is more often effective, morphia occasionally, but the agent which seems to do good in one series of convulsions may fail entirely in the next. Although bromide has little influence over the convulsions which are due to active irritation, it often controls the consecutive fits, which recur, for instance, after active syphilitic mischief has been removed by iodide. In all cases as much brain rest as possible should be secured, active physical exertion and constipation should be avoided, since all these influences tend to increase the headache and other symptoms.

During the last few years some intracranial tumors have been successfully brought into the domain of surgery by MacEwen, Godlee, and especially by Horsley. With the help of antiseptic surgery, the removal of a tumor from the brain has been proved to be a less dangerous operation than some others that are performed. It is only, however, a small proportion of intracranial growths in which an operation can be undertaken with sufficient prospect of success to render it justifiable. The cases suitable for an operation are those in which the growth is in or just beneath the cortex of the brain. It is chiefly in tumors of the motor region that the symptoms are such as to indicate the seat of the disease with the necessary precision. Probably tumors of the occipital lobe, causing hemianopia, or of the first temporal, disturbing the auditory function, may also be successfully removed.

On the other hand, it is not probable that tumors of the central ganglia, crura, pons, or base can be successfully removed. A growth in one hemisphere of the cerebellum would no doubt be accessible, but such tumors seldom cause distinctive symptoms until they are of large size, and even then it is scarcely possible to ascertain their exact situation. The middle lobe of the cerebellum is so inaccessible and so close to important structures that it is not likely that the removal of a tumor could be survived.

The operation itself is a formidable one, and its mortality will probably be found to be considerably higher than was suggested by the earlier cases. There remains, of necessity, a considerable area of the skull from which bone is absent, and some protecting cap has afterward to be worn. There remains, also, a permanent loss of brain substance, often larger than that influenced by the growth, with some consequent permanent loss of function. Of the ultimate success of the operation it is too soon to speak. The entire removal of infiltrating tumors is a matter of considerable difficulty, and hence it is of great importance that there should be no unnecessary delay before the operation is undertaken, whenever the symptoms of a tumor are conclusive. But it is of great importance that antisyphilitic treatment should first be thoroughly tried in any case in which it is even possible that the disease may be amenable to such treatment. It must be remembered, also, that tubercular tumors often become latent and cease to trouble the patient. It is not often, however, that a tubercular tumor is in a situation from which its removal is practicable. Syphilitic growths, on the other hand, are often cortical, and in this case great caution is necessary.

INTRACRANIAL ANEURISM.

Both the larger arteries of the brain and their branches within the cerebral substance are occasionally the seat of aneurismal dilatation. Within the brain the aneurisms are minute, rarely exceeding a pin's head in size, and hence are called "miliary aneurisms." They are important chiefly as causing hemorrhage into the substance of the brain, in connection with which they have been described (p. 774). Only the aneurisms of the larger cerebral arteries will be considered in this section. Such aneurisms are more common in the vessels of the brain than in those of similar size elsewhere, partly on account of the considerable blood pressure to which these vessels are exposed, and partly because their walls are more frequently the seat of morbid changes.

CAUSES.—Males suffer more frequently than females, in the proportion of three to two. Practically unknown under ten years of age,* aneurisms occur in each decade of life, from ten to sixty, with nearly equal frequency; after sixty they become rare, but are met with occasionally up to extreme old age. About an equal number occur before and after forty, and about one-sixth in the second decade of life. Thus they are much more frequent during the first half of life than are aneurisms elsewhere. An explanation for this is found in their immediate causes.

There appears to be, in rare cases, a peculiar inherited tendency to the formation of aneurism. A distinguished physician died young, from the rupture of an intracranial aneurism; ten years later his brother, a medical student aged twenty, showed me a distinct aneurism on an artery of the hand.

The immediate cause of an intracranial aneurism is the same as that of aneurisms elsewhere, a change in the structure of the wall whereby the muscular and elastic tissue is replaced by simpler fibroid tissue. In consequence of this the elasticity and contractility of the wall are lost, it becomes distensible, and yields permanently under the blood pressure. An aneurismal bulging results, which goes on increasing as the walls, thinned by extension, lose still further their power of resistance. But the mechanism by which this structural change is produced varies in different cases. There may be a primary degeneration, or local damage to the wall may be caused by injury, by syphilitic disease, or by embolism. Primary degeneration and traumatic influences cause aneurism elsewhere more frequently than within the skull; but syphilitic disease and embolism cause aneurism much more frequently in this than in other situations.

(1) *Primary degeneration* is an occasional cause in the second half of life. It may be a fibroid change or simple atheroma. Often there is exten-

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* Although aneurisms are practically unknown in children it is probable that disease of the arteries due to inherited syphilis may cause aneurism, since it may certainly cause cerebral hemorrhage. An instance in a boy of eight is mentioned on p. 778. The occurrence of aneurism from embolism is also possible under ten, although I do not know of any recorded instance.

sive fatty and calcareous degeneration of the vessels at the base, and, on one of them, a distinct aneurism.

(2) *Injury* is a rare cause: an aneurism slowly develops after a blow or fall on the head, apparently in consequence of the change in the wall of the artery produced by inflammation spreading to it from adjacent structures. Possibly the wall of the vessel is sometimes directly damaged.

(3) *Syphilitic disease* affects, as is well known, the arteries of the brain more frequently than those of other parts. Its characters have been described at p. 811. A new growth infiltrates the wall, destroying the muscular tissue, and if it subsides after attaining a considerable degree, it may leave cicatricial damage, rendering the artery distensible. An aneurism has been frequently met with in cases of constitutional syphilis in young persons, in whom no other cause was discoverable.

(4) *Embolism* is probably the most frequent cause in the first half of life. There are few cases during this period, without indications of syphilis or injury, in which there is not evidence of past or present heart disease.* We know that the fragments of lymph which are washed from the valves in endocarditis have often an irritative character, bearing septic material or organisms, which may excite inflammation similar to that in the place from which they come. It is in the highest degree probable that the origin of aneurisms which occur in this association is the imperfect occlusion of an artery by such a fragment of lymph, which excites inflammation in the wall of the vessel, and, as a result of this inflammation, the wall becomes distensible. In these cases the arteries of the brain are free from any general disease. It is in harmony with this theory that the middle cerebral, embolic occlusion of which is the most frequent, is that vessel on which aneurisms are most common, and that in these cases there are often several aneurisms.†

General Pathology.—Aneurisms are twice as common in the system of the internal carotid as in that of the vertebrals. The middle cerebral branches are affected more often than any other, but only a little more frequently than the basilar, the two together making up more than half the number of cases. Next in frequency is the internal carotid itself, which is, however, diseased only half as frequently as the middle cerebral. In two-thirds of the cases of aneurism, one of these three vessels is the seat of the disease. The other arteries are affected in the following order of frequency: anterior cerebral, posterior communicating, anterior communicating, vertebral, posterior cerebral, inferior cerebellar.* Aneurism of the arteries of the central ganglia, within the substance of the brain, are very rare. A remarkable instance in which an aneurism in the lenticular nucleus attained the size of a small chestnut before it caused death by rupture, has been recorded by Bastian.† Other instances are on record in which an aneurism, although not found, was probable, because fatal hemorrhage occurred under the conditions in which aneurisms are produced.‡

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* The frequency of heart disease was noted by Lebert and other observers, but the significance of the association was first pointed out by Church.

† Recent medical literature abounds with illustrations of the association of intracranial aneurisms and endocarditis. One striking instance may be quoted. A boy, twelve years old, suffered from mitral disease after rheumatic fever, and after death there was found circumscribed deposits on the mitral valve, and numerous infarcts in the spleen and kidneys. At the origin of the left posterior cerebral artery from the basilar there was a small aneurism the size of a pea, and the right posterior cerebral was closed by an embolus, immediately behind which was another aneurism the size of a pea. The artery beyond was obliterated. Here we have a demonstration of the local association of the two processes (Rauchfuss, *St. Petersburg med. Wochenschrift*, Feb. 18th, 1878, and *Virchow's Jahresb.*, *1878*, vol. ii, p. 102.

Aneurisms are rather more common on the left side than on the right, in the proportion of four to three. In about a fifth of the cases there is more than one aneurism. In character, the aneurism is almost always a true sacculated aneurism; very rarely dissecting. Occasionally, there is a general dilatation of the vessel. The walls are usually thin, but often they are lined by laminated clot, and sometimes supported by thickened and adherent membranes. In size they commonly vary from a pea to a nut, sometimes attaining a larger size. They have been met with as large as a hen's egg on the internal carotid, anterior cerebral, basilar and posterior cerebral, and as large as a pigeon's egg on the middle cerebral and posterior communicating arteries. In shape they are usually round, sometimes oval. The surface is red and smooth, and the tumor often has the aspect of a small round mass of clot, the wall being so thin as to be invisible. When rupture has taken place, the aneurism is in the midst of a coagulum, and great care may be needed to discover it. It is best to wash away the clot slowly by a gentle stream of water.

An aneurism, except it be of very small size, necessarily obtains room by compressing adjacent structures. The cerebral substance may be thus damaged, the nerves at the base compressed, and even the bone eroded. It is by this means that the chief symptoms are produced.

Rupture of the aneurism occurs in rather more than half the cases, and in a still larger proportion in the young. Most frequently the blood escapes into the membranes at the base of the brain. The opening in the aneurism is usually small, and the escape of blood is gradual and sometimes intermittent. It may be forced from the base into the fourth ventricle by the openings through which liquid escapes out of the ventricle; this distention of the fourth ventricle is occasionally the immediate cause of death. In some cases, however, in which the aneurism is supported by thickened membranes on its outer side, it gives way where it is in contact with, and perhaps embedded in, the brain substance, and the hemorrhage occurs, not into the membranes, but into the brain. Thus the blood from an aneurism of the middle cerebral may escape into the hemisphere until it reaches the lateral ventricle, and I have known an aneurism of the commencement of the posterior cerebral to tear up and infiltrate the pons before it escaped externally. The slowness of the hemorrhage probably favors its extension into the brain, the blood slowly disintegrating the tissue before it, along the line of least resistance. In one case a narrow fistulous tract of blood extended from an aneurism of the middle cerebral to the lateral ventricle. An aneurism of the internal carotid has been known to burst into the cavernous sinus.

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* The following is the numerical frequency in 154 cases obtained by combining, in so far as they do not overlap, the statistics of Lebert (*Berlin. klin. Wochenschrift, 1866*), Durand ("Des aneurysms du Cerveau," Paris, 1868), and Bartholow (*Amer. Journ. Med. Sciences*, Oct. 1872), and the addition of ten cases recorded by others, Middle cerebral, 44 cases; basilar, 41; internal carotid, 23; anterior cerebral, 14; posterior communicating, 8; anterior communicating, 8; vertebral, 7; posterior cerebral, 6; inferior cerebellar, 3.

† *Trans. Clin. Soc., 1884*, p. 18.

‡ Another instance is the case of hemorrhage in a boy of eight with inherited syphilis and arterial disease, mentioned already. A very instructive case of hemorrhage into one corpus striatum, bursting into the ventricles, in a boy of fifteen, with slight mitral disease, has been recorded by Bastian (*Trans. Clin. Soc.*, xvii, 1884, p. 21). In each of these cases an aneurism of a cerebral artery was highly probable.

General Symptomatology.—A small aneurism, in any situation, may cause no symptoms, and its existence may be unsuspected until rupture occurs. Such latency is most common in aneurisms of the anterior communicating and the cerebellar arteries, and it is not infrequent in those of the middle cerebral and basilar. When symptoms occur, these are partly general, partly local. Headache is the most common; it is usually continuous, but sometimes paroxysmal. Its seat has little relation to the position of the aneurism, except that it is usually occipital when the basilar artery is diseased. Giddiness is also common, whatever be the seat of the aneurism. Mental dullness and irritability have been caused by aneurisms in various situations, but most frequently when the anterior cerebral was diseased, least frequently by aneurism of the internal carotid and middle cerebral. Convulsions are not frequent except in aneurism of the middle cerebral. Large aneurisms in any situation may cause paralysis of limbs, but this is most frequently due to those of the middle cerebral and basilar. The cranial nerves are often paralyzed, as they are adjacent to the vessels most frequently affected (see Fig. 311). Optic neuritis is not common; it has been occasionally met with, but only in aneurism of the internal carotid; when present it is double. Probably an ophthalmoscopic examination would have revealed it in a larger proportion of cases. A murmur, the most common symptom of aneurisms elsewhere, is very seldom produced by those within the skull. In a few recorded cases* of aneurism of the internal carotid, not only has the patient been conscious of a murmur, but this has been distinctly audible on auscultation of the skull, and has been arrested by pressure on the carotid. In one case of aneurism of the left vertebral artery, beside the medulla oblongata, a loud murmur could be heard on each side, between the mastoid process and the spinal column.†.

When embolism causes an aneurism, the obstruction is necessarily partial, and, as a rule, the causal process causes no symptoms. Nevertheless, it is possible that transient symptoms sometimes occur at the time of the embolism. In the case recorded by Bastian, mentioned in the note at p. 787, three years before the fatal apoplexy the boy was suddenly convulsed, and afterward screamed for two or three hours. A careful inquiry might elicit a history of transient symptoms in many of these cases.

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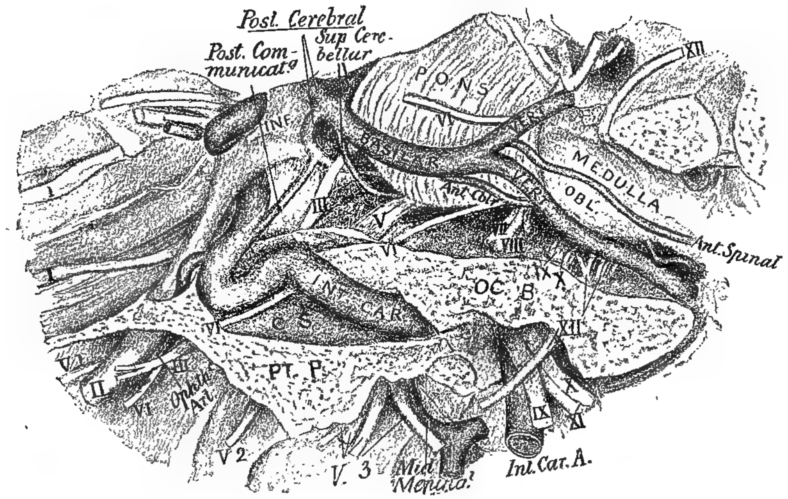
* Coe, Holmes of Chicago, Hutchinson, Humble, Jeaffreson.

† Moser, *Deut. Arch. f. kl. Med.*, Bd. 35, p. 418.

ANEURISMS OF SPECIAL ARTERIES.

The symptoms produced by aneurisms of special arteries depend on the structures adjacent to these vessels. Their relations are imperfectly presented in the brain after it has been removed. Fig. 311, representing the chief relations, is from a dissection, made at my suggestion by Mr. Shattock, for the purpose of showing the relative positions of the chief arteries, nerves, and other structures at the base of the brain. A reference to it will render clearer many points in the special symptomatology of the intracranial aneurisms.

FIG. 311.



Relations of the arteries and nerves at the base of the brain, from an actual dissection in which the bones, membranes, and wall of the cavernous sinus were removed from the left side. INF, infundibulum; C. S., cavernous sinus; PT. P., pterygoid process; OC. B., occipital bone. The cranial nerves are indicated by Roman numerals; the names of the arteries are marked upon them. (*After a drawing by Mr. C. E. Adams.*)

Internal Carotid.—An aneurism may be formed within the cavernous sinus, or after the artery has emerged from this, as it lies on the inner side of the anterior clinoid process and origin of the optic nerve, with the origin of the olfactory nerve in front of it. The aneurism may compress the adjacent frontal and temporo-sphenoidal lobes, but its chief symptoms are due to compression of the optic nerve on the inner side, and of the nerves in the wall of the cavernous sinus on the outer side. The sight of the eye on that side fails. The third nerve usually suffers early, and ptosis may be the first symptom, but all the muscles may ultimately become paralyzed, and the eyeball may lose sensibility in consequence of damage to the ophthalmic branch of the fifth. Smell also is often impaired. When the aneurism is large, and extends backward, it may cause hemiplegia by pressure on the crus. Generally the pressure effect is limited to the ocular nerves. Rupture has occurred in less than half the cases. Transient fullness of the retinal veins has been observed to result from the compression of the cavernous sinus by a carotid aneurism, but the pressure is quickly relieved by the free communication of the ophthalmic and facial veins; the enlarged angular vein may be conspicuous beneath the skin. Optic neuritis, when it exists, is apparently due, not to the pressure on the sinus or the nerve, but to the extension of inflammation in the vicinity of the aneurism (Michel).

Anterior Cerebral.—The aneurism is usually in front of the origin of the vessel, sometimes at a bifurcation. The artery runs between the optic and the olfactory nerves, and an aneurism of this part exerts compression on the same parts, and produces nearly the same symptoms, as one of the internal carotid, with the important exception that the nerves to the muscles of the eyeball generally escape. An aneurism further forward may cause no local symptoms. The sac usually has very thin walls, and rupture occurs in more than half the cases.

Anterior Communicating.—The tumor exerts pressure only on the inner surface of the frontal lobes. The aneurism is usually small, and, as a rule, no symptoms are produced. The wall is thin, and rupture occurs early in most cases.

Posterior Communicating.—The aneurism is usually situated near the origin of the artery from the internal carotid. It compresses the adjacent temporo-sphenoidal lobe, and the pressure may even reach the corpus striatum and optic thalamus. The third nerve is that which most frequently suffers. Affection of sight is also common, but it is usually incomplete, and we have no exact observations on its character. Probably there is hemianopia from pressure on the optic tract, which the artery crosses. Hemiplegia is rare, in spite of the proximity of the vessel to the crus cerebri, because the aneurism is thin walled, and usually bursts before it has attained considerable size or has exerted considerable pressure.

Middle Cerebral.—The aneurism is sometimes near the origin of the vessel, but more often within the fissure of Sylvius, sometimes on one of its branches. Occasionally there is more than one aneurism. The chief pressure is on the hemisphere, and when the aneurism is within the fissure of Sylvius, the pressure may reach the corpus striatum. Paralysis of cranial nerves is rare; on the other hand, hemiplegia and convulsions are common. Speech is occasionally affected when the disease is on the left side. Rupture occurs in two-thirds of the cases. Aneurisms on the central branches of the middle cerebral are small, and probably cause no symptoms until they burst.

Vertebral.—A distinct aneurism, such as is shown in Fig. 274, p. 662, is extremely rare. In most conditions thus described there has been only a general dilatation of the vessel, often determined by an original inequality in the size of the two arteries. Important as are the adjacent structures, symptoms have seldom been observed in the cases in which this dilatation was found. In the case figured, facial spasm was the result of the compression of the nerve trunk.

Basilar.—Although absolutely second in frequency of affection, if the short length of the vessel is taken into consideration, the basilar exhibits a greater tendency to suffer than any other artery. Occasionally there is general dilatation; more frequently the anterior extremity is enlarged; sometimes there is an aneurism on one side of the vessel in the middle of its course; rarely it is near the origin from the vertebral. The pressure is differently distributed according to the position of the aneurism. Usually the pons suffers chiefly, sometimes on one side only, and the pressure may even extend to the middle peduncle of the cerebellum and the adjacent hemisphere. Occasionally the aneurism extends into the pons so deeply as to reach the fourth ventricle. The compressed portion is often softened. An aneurism of the anterior part may compress the crus; one of the posterior part, the pyramids and olivary bodies of the medulla. The third nerves rarely suffer, but the cranial nerves from the fifth to the vagus are frequently compressed, according to the situation of the tumor. The hypoglossal usually escapes. Internal hydrocephalus is an occasional consequence of the obstruction to the escape of liquid from the lateral and third ventricles. Rupture occurs in about half the cases; it is usually sub-arachnoid, although the blood sometimes escapes into the substance of the pons. In about one-third of the cases there have been no symptoms, a fact which, as Lebert observed, is remarkable, considering the important adjacent structures. Headache is occipital in position, and may be severe. Giddiness is more pronounced than in aneurisms elsewhere. The other symptoms are those common to basal tumors in this situation; paralysis of the limbs may be bilateral or unilateral, and one or more cranial nerves may be paralyzed opposite to the side on which the limbs are chiefly affected. The fifth suffers most frequently, and sometimes there is neuro-paralytic ophthalmia. Difficulty in articulation and in swallowing are occasional symptoms. Convulsions are rare. Besides the terminal apoplexy which results from rupture, apoplectic attacks occasionally occur, and even end in death, without rupture, in consequence of softening adjacent to the aneurism, which is sometimes due to the closure of a branch.

Posterior Cerebral.—The aneurism is usually situated not far from the origin of the vessel, and may compress the upper part of pons, crus, temporo-sphenoidal lobe, and the third and sixth nerves. Hence, when symptoms have existed, there has been hemiplegia with alternate paralysis of these two nerves, especially of the third.

Cerebellar Arteries.—Both the superior and inferior anterior cerebellar arteries have been found affected with small aneurisms, but the pressure effects are too various, and usually also too slight, to permit any general statement.

COURSE.

The duration of an aneurism can only be inferred from the inadequate evidence afforded by the duration of the symptoms. It is certain, however, that this varies between wide limits, from a few weeks to five or six years. In some cases the sac becomes filled with clot, and a spontaneous cure is thus effected.* The majority of cases have ended by the rupture of the aneurism, which is, so far as past experience has gone, invariably fatal. Quickly deepening coma, and sometimes convulsions, result from the hemorrhage into the membranes, and death often results in a few hours, although sometimes the hemorrhage occurs so slowly that life is prolonged for days. Now and then one or more remissions occur, the cause of which can be traced, post-mortem, to be the temporary cessation of the hemorrhage, the blood having evidently been effused at different dates. When the hemorrhage occurs into the brain substance, the symptoms are similar to those in ordinary cerebral hemorrhage.

Diagnosis.—In a considerable number of cases the diagnosis of intracranial aneurism is impossible, because it produces no symptoms until its rupture. When symptoms are present, they are commonly those of a tumor at the base of the brain, pressing on the cranial nerves and motor tract. The aneurism *is* a tumor, and its distinction from a growth is often impossible; sometimes it may be made with greater or less probability, very rarely with certainty. It is certain only when a murmur exists, audible on auscultation of the skull. But such a murmur has only been heard in a few cases of aneurism of the internal carotid and vertebral. In other arteries the force of the blood current seems to be insufficient to produce it. The range of certain diagnosis is thus extremely limited. But the distinction may be made with some probability when the symptoms of a tumor, in the position of a vessel, develop in a patient who presents one of the two causes of aneurism, arterial degeneration and present or past heart disease. Syphilis and injury, two other causes, have little diagnostic significance, because each (and syphilis especially) is a cause of growth more frequently than of aneurism. It might be thought that aneurism elsewhere would occasionally aid the diagnosis, but, as a matter of fact, the coincidence is too rare to be of practical importance.

When the pressure is exerted only on cranial nerves, the distinction from local meningitis, such as results from syphilis, may be extremely difficult, especially if the patient has suffered from this constitutional disease. The most important indication is that treatment rarely fails to remove the symptoms in simple syphilitic disease, while it has little influence in cases of aneurism.

In all cases the fact that the tumor is in the position of an artery is of considerable diagnostic importance. The special symptoms which indicate this have been already described. The seat of pain is of significance only when it is occipital (basilar artery). Loss of sight in one eye, sometimes extending to the other; with, or especially without, optic neuritis; with or without loss of smell on the side first affected—occurs in aneurism of the internal carotid or anterior cerebral, and the distinction between the two depends on the presence or absence of paralysis of the other nerves to the eyeball first affected, which occurs especially when the aneurism is of the internal carotid. Paralysis of the third nerve without loss of sight in one eye is produced by aneurism of the posterior communicating, and associated with hemiplegia on the opposite side, by aneurism of the posterior cerebral. Hemianopia would increase the probability of an aneurism in this part of the base. Affection of the fifth alone is of little significance, but with bilateral weakness in the limbs, difficulty of articulation or of swallowing, it is produced by aneurism of the basilar, and so also is paralysis of the cranial nerves below the sixth, usually associated with weakness of the limbs on the opposite side. Hemiplegia is of little significance in itself, but if considerable it is unlikely that there is an aneurism of either the anterior cerebral or the posterior communicating arteries. Simple convulsions, epileptiform in character, are not common, but if they begin locally they suggest that the disease is in the middle cerebral, and if they are opisthotonic, that it is in the basilar. These symptoms are of diagnostic significance only in the presence of a cause of aneurism.

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* A case in which this occurred in an aneurism of the internal carotid has been recorded by Mr. Hutchinson (*Clin. Soc. Trans., 1875*).

The occurrence of rupture, with severe apoplectic symptoms, increases very much the probability of the diagnosis, although it is then a matter rather of scientific curiosity than of practical importance. Even if no previous symptoms have existed, it may be suspected that sudden apoplexy, steadily deepening in degree, is due to the rupture of an aneurism, if it occurs in a young person, especially if there is a history of heart disease or of syphilis, or of injury to the head. The characters of the apoplexy afford but limited indications of the probable seat of the aneurism. Those suggestive of meningeal hemorrhage are of no localizing significance. The symptoms of cerebral hemorrhage, at first unilateral and subsequently ventricular, suggest an aneurism of the middle cerebral, and this is the more probable if there are at first unilateral convulsions. The symptoms of hemorrhage into the pons suggest an aneurism of the basilar, or of the commencement of the posterior cerebral artery.

The difficulties of the diagnosis of aneurism do not always cease with life. The damage to the tissues and vessels, caused by the hemorrhage that follows rupture, may render it impossible to find the remains of a small aneurism. Hence, the occurrence of fatal cerebral hemorrhage in a person under forty who has no kidney disease, and who has suffered from valvular disease of the heart, syphilis acquired or inherited, or has had a blow on the head, justifies a strong suspicion of aneurism, even if no aneurism can be actually found.

Prognosis.—If the diagnosis of an intracranial aneurism is certain the prognosis is extremely grave. The probability that death will result is very great, and the duration of life is most uncertain, since it is impossible to say how near or how distant may be the fatal rupture. These conclusions are not invalidated by the fact that, in a considerable proportion of recorded cases, rupture has not occurred. In most of these the aneurisms were small, and caused no symptoms. The question of prognosis relates only to aneurisms which have caused symptoms, and in most of these cases rupture has occurred. But the end may not be near; in many cases the symptoms have continued for two, three, or five years, and if stationary, the chance of long duration is slightly greater. Nor is a fatal issue absolutely certain. A spontaneous cure of intracranial aneurism has been more than once observed. In aneurism of the internal carotid, the prognosis is distinctly better than in that of other arteries, because more can be done by treatment, or at least has been done, than when other vessels are the seat of the disease.

Treatment.—If an intracranial aneurism is suspected, it is important to avoid as far as possible all influences which tend to increase the arterial pressure, as exertion and stooping, or which tend to accelerate the movement of the blood, as alcohol. To these ends also the bowels should be kept loose, and the patient should sleep with head and shoulders well raised. If there is reason to suspect that the aneurism is of syphilitic origin, iodide of potassium should be given to prevent increase of the disease in the wall, although it is not to be anticipated that the removal of the syphilitic elements can influence an aneurism already developed. Ergotin has been recommended, but on doubtful grounds, as the wall of an aneurism contains no tissue on which the drug can act. More reasonable measures are those designed to favor coagulation in the sac. These consist, first, in the "starvation" treatment, the utility of which in intracranial aneurism has still to be proved. Secondly, iodide of potassium has been given, to promote coagulation, and with a result which certainly warrants its employment. In one case* the patient, a woman aged forty, suffered from right frontal headache, photophobia, impaired vision in the right eye, and paralysis of the external rectus; a systolic murmur could be heard, loudest at the right temple. Iodide of potassium was given in doses increasing to thirty-six grains daily. Four months later, after a violent attack of vomiting and purging, the murmur suddenly ceased, and did not recur. In five weeks the paralysis of the external rectus had disappeared. There can be no doubt that an aneurism existed, and that coagulation had occurred in the sac. The immediate cause of the clotting was probably the prostration from the vomiting and purging, but it is at least possible that the result may have been favored by the iodide of potassium, since this drug certainly promotes coagulation in aortic aneurisms. The third method of promoting coagulation in the sac is to ligature the artery from which the diseased vessel derives its supply of blood. Hitherto this has only been adopted in the case of aneurism of the internal carotid, for which the common carotid has been tied with success. It is only in aneurism of this artery that the diagnosis has been sufficiently sure to justify the surgeon's interference. If other aneurisms could be diagnosed with equal certainty, this measure would certainly be often justifiable. For basilar aneurism the vertebrals would have to be tied, but Dr. Alexander, of Liverpool, has shown that this operation is practicable, and, if not devoid of danger, is attended with less risk than is the aneurism itself. When rupture has occurred, no treatment has hitherto had any influence. The fact, however, that the escape of blood may be slow and intermittent suggests that a copious venesection might afford a very slender chance of arresting the hemorrhage.

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* Recorded by Dr. Humble, of Corfe Castle, *Lancet, 1875*, ii, pp. 490 and 874.

DEGENERATIONS OF THE BRAIN.

CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN.

The old view, that softening may occur as a primary process, although wrong as regards the common acute softening, seems to be justified by rare cases in which softening occurs without demonstrable association with vascular disease, and is so gradual in its onset and slowly progressive in its course, as to make such an association very improbable. Some cases of this kind have been collected by Wernicke,* and a few other instances are to be found in medical literature, old and recent. The affection is almost always seated in the white substance of the hemisphere, and consists of simple white softening, presenting, under the microscope, only the products of degeneration. The affected area is often sharply limited from the adjacent normal substance. The extent of the lesion varies much in different cases. The gray matter of the cortex appears never to be primarily affected, but it may suffer secondarily when the softening of the white substance is extensive. The diffluent tissue may then be limited externally by the pia mater and a thin layer of cortical gray matter below this. Chronic softening sometimes affects the cerebellum, but it there differs from the cerebral softening since the cortex suffers more than the white substance. I have seen the cortex of almost the whole of both hemispheres of the cerebellum uniformly softened and atrophied, while the white substance was unaffected. In this case there was also softening in the cerebral hemispheres.

The certain cases are too few to afford much information regarding the causes of chronic progressive softening. All that can be said is that it occurs in both sexes, and is a disease of the second half of life, most frequent between sixty and eighty. The chief symptoms are hemiplegia—motor and sensory—of gradual onset. Weakness commences in one part and gradually spreads to the whole of one side. Considerable sensory loss is rare. Slight irritation symptoms are common; rigidity often accompanies the commencing paralysis, but may cease when it is fully developed. Numbness, tingling, and formication in the limbs are also common, and in some cases these have been the seat of paroxysmal pain. Convulsions are very rare. There may be occasional attacks of giddiness, but apopletic seizures do not occur in the chronic form. Intellect may be little affected, or the patient may become dull and apathetic, and, toward the last, comatose. But it is not common for the cerebral symptoms to increase until death ; more often they reach a certain degree of severity and then remain stationary; death ultimately occurs from bedsores, pneumonia, or some other intercurrent senile malady, perhaps favored by the nervous state. The duration of recorded cases has usually varied between two months and two years.

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* "Lehrbuch der Gehirn-Krankheiten," Bd. ii, 149.

Although it is not probable that the condition is directly related to arterial disease, the latter is present in most senile cases, and may have an indirect influence in determining the lesion. The white substance is partly nourished by the central, partly by the cortical vessels, and in the district between the two regions of blood supply, small foci of softening are not uncommon, as Charcot has shown. More extensive softening in this region may be an occasional result of considerable diminution of the blood supply without any actual occlusion of vessels. In favor of this view is the fact that subacute and acute cases are met with which present every gradation of course between the chronic form and the ordinary sudden form of softening. In the chronic variety the softening has been regarded as inflammatory, but only on the ground of the initial symptoms of irritation—evidence that seems inadequate.

It will be seen that the chronic symptoms bear a considerable resemblance to those of cerebral tumor. A difference is in the fact that headache is more often absent than present, and very rarely reaches the degree of intensity that is so characteristic of tumor. The greatest diagnostic difficulty is presented by cases of very chronic course. If symptoms develop in a subacute manner, reach a considerable degree of intensity in six weeks or two months, and then remain stationary, the distinction from tumor is more easy, because a rapidly-growing new formation rarely undergoes arrest. The age of the patient is also of some significance; in the first half of life tumor is more probable than softening. The occurrence of convulsions is also strongly against softening. Of the ophthalmoscopic changes in chronic softening little is known, but it is probable that a very intense optic neuritis is in favor of tumor.

Little can be said regarding the treatment of these cases. Our knowledge of them depends only on the cases in which the nature of the disease has been ascertained by a post-mortem examination, and in these it does not appear that any treatment employed had an appreciable influence on the course of the disease. The next most suitable measures will probably be those recommended for the chronic stage of acute softening, rest, nutritious food, with general and cardiac tonics.

DISSEMINATED OR INSULAR SCLEROSIS.

The disease thus named consists in the formation of scattered islets of sclerosis in various parts of the brain and spinal cord, sometimes also in the cranial nerves. Our knowledge of the affection is recent. The lesion was, indeed, long since figured by Carswell and Cruveilhier, and was described thirty years ago by Frerichs, Rindfleisch, etc., but the malady was not generally recognized until re-investigated by Vulpian and by Charcot and his pupils, and described by Charcot in his widely-circulated "Lectures." It is called by the French, "Sclerose en Plaques Disseminées," and by the Germans, "Multiple Sclerosis." The apt designation "Insular Sclerosis" was proposed by the late Dr. Moxon, who first described the disease in this country.

The pathological relations of the morbid process are still, to a large extent, obscure, and much remains to be done in elucidating its clinical history, and in distinguishing it from allied diseases, whether it is to be separated from them or not. As regards the symptoms of the typical form, little can be added to the description given by Charcot, but there is much uncertainty regarding the history of untypical varieties.

Three forms of the disease have been distinguished by Charcot—cerebral, spinal, and cerebro-spinal, according to the situation of the islets of sclerosis, and the corresponding difference in the symptoms they produce. The cerebro-spinal variety must be regarded as the typical form, which gives rise to the complete series of symptoms.

ETIOLOGY.—Insular sclerosis occurs in both sexes with nearly the same frequency. It is met with at almost all periods of life between childhood and old age, but especially in the first half of adult life. The vast majority of the cases commence between twenty and thirty-five. In one verified case it commenced at seven;* in another as late as sixty, but in old age it is extremely rare. Direct heredity, or the affection of two brothers or sisters, has been noted in a few instances, but is quite exceptional. It is more common for the patient to come of a neuropathic stock, and to present a family history of epilepsy or of some form of chronic paralysis. Still more commonly, however, no hereditary or family predisposition can be traced. In about half the cases the disease develops without any apparent exciting cause. Among the morbid influences to which it has been immediately ascribed, the most frequent are exposure to cold, mental distress and overwork, some acute disease, an injury to the central nervous system, or some specific febrile disease. Among the latter, typhoid fever and smallpox are the most frequent antecedents; it has also been known to follow diphtheria and erysipelas. The preceding morbid processes in the nervous system have been apparently acute or subacute inflammation (myelitis, etc.), primary, or secondary to some severe concussion. The acute symptoms have subsided completely or partially, and after an interval, sometimes of a year or more, the indications of insular sclerosis have slowly developed.

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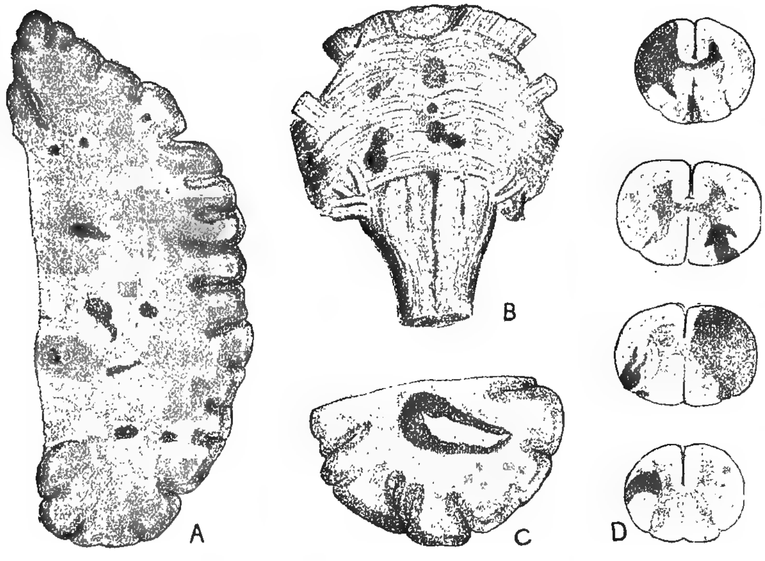
* Symptoms suggestive of insular sclerosis have been met with even in very young children.

Pathological Anatomy.—The islets of sclerosis that characterize the disease are always irregular in situation, scattered as it were at random through the central nervous system. The only law which can be traced in this distribution is that they generally commence in the white substance. In the brain they seldom invade the gray matter of the cortex, and never begin in it, although the white substance of the centrum ovale usually contains many areas of disease. They are irregular in shape, and vary in size from that of a pea to that of a walnut. In color they are reddish gray, a little darker than the normal gray substance of the cortex, and rather more translucent. Their section is usually on the level of that of the adjacent cerebral tissue, occasionally it is a little depressed, but on the surface they are sometimes slightly prominent. The sclerosis does not, however, increase perceptibly the volume of the part in which it occurs. To the naked eye the islets are sharply defined; the transition to the normal tissue is sudden. The consistence of the sclerosis is usually a little greater than that of the brain tissue, and they are sometimes distinctly firm; occasionally, in old cases, some may be almost cartilaginous in their firmness. Owing to the position of the islets in the cerebral hemisphere there is usually no indication of their presence on the surface of the brain, even when a large number are found in its interior. Although the cortex is seldom involved, the central ganglia are frequently invaded, and the sclerosis is sometimes abundant beneath the lining membrane of the lateral ventricle (Fig. 312, C) and in the corpus callosum. In the crus and pons the islets are often numerous; some may be conspicuous on the surface from their contrast with the white fibres which constitute the superficial layer of these parts, while others are scattered through the interior of the pons or medulla. They are met with also in the cerebellum, but are usually few, and here also are confined to the white substance. In the spinal cord, the areas of disease appear on the surface as gray patches; they are irregular in size, but usually of greater vertical than transverse extent, and on section they are seen to extend for a variable distance in the substance of the cord, sometimes through a considerable part of one-half (Fig. 312, D). Often not a single section of the cord is free from disease. Occasionally, in cases of long duration, the disease extends through the whole thickness of the cord, the surface of which is gray throughout at this part, and the sclerosis thus becomes diffuse. Such diffuse sclerosis of the lower part of the cord has been met with in association with characteristic insular sclerosis of the upper part of the cord and of the brain. Very rarely a diffuse slight sclerosis has united the nodules in the brain. The cranial nerves are often involved; for a certain distance the nerve may be gray in its entire thickness or in part. The olfactory, optic, third, fifth, and facial nerves are those that have been most frequently diseased. Less commonly the roots of some of the spinal nerves are affected.

In the diseased areas, there is an overgrowth of the neuroglia and a wasting of the nerve fibres. The tissue contains connective-tissue elements, glia cells and fibres, spider cells, and ultimately a mass of fine fibres, denser in aspect than the fibres of the normal neuroglia. Among these tissue elements there may be fatty granules and globules, the remains of the degenerated nerve elements. The nerve fibres that pass through are changed in various degrees. The chief alteration is a narrowing of the white substance, which becomes reduced (as seen in section) to a narrow, scarcely appreciable ring around the axis cylinder, and even this may cease to be recognizable. The axis cylinders persist longest, but ultimately they may disappear.* The walls of the vessels become thickened, and the increase of the connective-tissue elements may appear to start from them. Should the sclerosis involve the gray substance, the nerve cells become atrophied. Over the affected region there may also be a slight thickening of the pia mater, but rarely sufficient to be recognized with the naked eye.

FIG. 312.



Insular sclerosis.

A. Centrum ovale of right hemisphere.
B. Pons and medulla.
C. Peri-ependymal sclerosis around the descending cornu of the lateral ventricle. (A, B, and C are from an original case.)
D. Sections of the spinal cord to show the varying distribution of the sclerosis at different regions. (*After Leyden.*)

If an area of sclerosis occupies part of a tract of fibres that is liable to undergo secondary degeneration, such degeneration may or may not be found beyond the part diseased. The difference seems to depend on the condition of the axis cylinders; as long as the change is confined to the white substance of the nerve fibres there is no secondary degeneration, but as soon as the axis cylinders begin to suffer, degeneration follows.

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* The persistence of the axis cylinders was first pointed out by Charcot, and has been since confirmed by Schultze, Déjérine, Babinski, and others.

Symptoms.—The wide variations in the position of the islets of sclerosis entails a corresponding difference in the symptoms that are produced. Loss of muscular power in the limbs, variously distributed, and often at first partial, is the most constant manifestation of the disease. Since the morbid process usually involves the pons and spinal cord, as well as the brain, some islets can hardly fail to occupy the motor tract. The deficiency of power in the arms is associated with a peculiar symptom, a jerky incoördination, sometimes to be observed also in the legs, but in less characteristic form. Some of the cranial nerves also are often paralyzed. Among other symptoms, nystagmus is exceedingly common, and so also is a peculiar disturbance of articulation.

The variation in the character of the symptoms extends also to their order and to their mode of onset. In some cases the peculiar incoördination in the hands, or in one alone, is the first indication of the disease. In other and more frequent cases, the legs first become weak, or the patient is unsteady in standing or walking. Less commonly some cranial nerve palsy occurs at the onset, or speech is observed to be peculiar, but in the majority of cases the limb symptoms precede the others.

Of the individual symptoms, the jerky tremor is the most peculiar and characteristic. It is, as just observed, chiefly manifested in the arms. At rest the limb is still, and presents no spontaneous tremor, but as soon as a movement is attempted, in the endeavor, for instance, to take hold of some object, the arm is thrown about in a wildly irregular manner, and moves in sudden quick jerks, now in one direction, now in another, until at last, by a great effort, and sometimes with a sudden dash, the patient succeeds in grasping the object. If he tries to raise a glass of water, the movements are so wild that all the contents of the glass are probably spilled. In an attempt to write, the violent movements produce only irregular strokes upon the paper as the hand dashes to and fro, and often the pen is broken in the attempt. In some instances the movements are slighter in range and more regular. It is said that in rare instances the movements have been known to continue during rest, but there is some doubt as to the nature of such exceptional cases. The movements are, as a rule, increased by effort, by emotion, and by attention. The more the patient tries to overcome them, the more violent they are. The muscles of the neck often share the disorder of movement, and there is irregular oscillation of the head, and sometimes of the trunk, whenever the patient sits up.

Some weakness usually accompanies this irregular incoördination; it may be at first very slight, and, indeed, at the very onset it may be absent, even when the tremor is distinct. Once established, the loss of power gradually increases.

The jerky irregularity has been ascribed by Charcot to an irregular resistance to conduction in the nerve fibres at the sclerosed spot; it is supposed that the changes in the white substance, or in the axis cylinder, interfere with conduction along the latter, and as this change is unequal in degree, even in adjacent fibres, there is a various degree of retardation of the voluntary influence on the muscles, and hence the irregularity of the contractions. This hypothesis derives some support from the fact that a precisely similar symptom is present in some cases of tumor of the brain, in which, for instance, a tubercular growth compresses the motor path, as in the case of tubercle in the crus, shown at Fig. 310, p. 888. I have met with this symptom also in a case of tubercle of the pons. To some (Erb, etc.) the theory has appeared inadequate, and they incline to the alternative explanation, that the disorder is due to the occurrence of islets of sclerosis in some particular situation in which they derange the coördinating function. The symptom is said to coincide with sclerosis of the pons, and to be absent in cases in which the lesion is confined to the spinal cord.

While the characteristic tremor usually accompanies loss of power in the arms, the legs more often present either simple incoördination or simple weakness. The latter is the more common, and is often the earliest symptom of the disease. Weakness may commence in one leg before the other, or in both at the same time. It is usually accompanied by an excess of myotatic irritability (foot clonus, etc.) which goes on to spasm, so that a state of spastic paraplegia is established. This is indicative, as in other cases, of degeneration of the lower parts of the pyramidal tracts. Such degeneration in this disease is secondary; the consequence of the development of a patch of sclerosis somewhere in the dorsal region of the cord, involving the pyramidal tracts. In the rare cases in which the lesion is limited to the spinal cord, such spastic paraplegia may be the only symptom of its presence. The ataxy of the legs, that is present in other cases, varies in its character, and probably also in its mechanism. In some cases there is a jerky incoördination resembling that in the arms, and probably produced in the same manner, whatever that may be. In this condition also the myotatic irritability is increased, and as paralysis increases spasm comes on. In other cases the spastic palsy is accompanied by simple unsteadiness, more like cerebellar than spinal ataxy. It is possible that this is due to the interference with the upward conduction of the path in the posterior median columns, by which the sensory impressions from the muscles probably pass to and influence the cerebellum. In a third and much rarer group there is ataxy with loss of the knee jerk, a condition closely resembling that of true tabes. It is possible that this condition may be the consequence of the development of a patch of sclerosis in the posterior columns in the lumbar region, but it is also possible that the condition is true tabes, a system degeneration in one part of the central nervous system accompanying the insular sclerosis in another part. This association will be considered in the section on pathology.

The paralyzed muscles sometimes present rigidity, and even permanent contracture. The extensor rigidity of spastic paraplegia is often considerable; sometimes it gives place to flexor contracture, so that the legs are permanently drawn up. Occasionally a single group of muscles may become rigid and contracted in the arms as well as in the legs. Muscular wasting is rare, but local irregular atrophy may result from the invasion of the gray matter of the cord by the sclerosis, and the consequent damage to the nerve cells.

Cutaneous sensibility is normal in the early stage of the disease, but it may afterward be impaired. The loss varies much in its distribution, according to the situation of the sclerosis on which it depends. Rarely there is hemianæsthesia, from the development of an islet in the sensory path within the brain. The loss is more often partial, involving only a small area on a limb, and it may or may not correspond to the motor palsy. When there is diffuse sclerosis of the cord there may be extensive loss of sensibility on the legs. The condition of sensation is intelligible when we remember how slowly the axis cylinders of the nerve fibres suffer, and that a greater amount of damage is required to abolish sensory conduction than suffices to cause motor palsy. Irritative symptoms are occasionally present—formication, radiating pains, or a sense of constriction—but they are seldom severe, and it is remarkable how perfectly free from discomfort the patient may be, even when there is much spasmodic tremor.

Eye symptoms are frequent and important. The optic nerve may suffer in various ways. There may be impairment of sight in one eye or in both, irregular in character, sometimes with a segmental loss in the field of vision, and without at first any visible changes in the optic nerve to account for it. This condition depends upon the development of an islet of sclerosis in one or both optic nerves, or in the optic chiasma. It may progress to almost complete blindness of one eye. After a time, secondary atrophy supervenes, and can be seen with the ophthalmoscope. Occasionally the atrophy is preceded by slight neuritis, when the sclerosis is near the eyeball. In some cases, again, there is a primary atrophy of the optic nerves, exactly like that which occurs in tabes. There is the same failure of sight, proportioned to the visible alteration in the nerve. Both eyes usually suffer, but one is often affected earlier and more than the other. This complication, of which I have seen four or five instances, is of much pathological interest.

The internal ocular muscles are seldom involved, unless the third nerve is paralyzed. Loss of the reflex action of the iris is scarcely ever met with, but the pupils are sometimes unequal.

On the other hand, the external muscles often present some abnormality. The most frequent disturbance is nystagmus, which is an exceedingly common symptom of the disease. Why it should occur we are unable to say; although the symptom bears some resemblance to the jerky tremor of the limbs, its constancy precludes the same explanation, because it seems impossible to assume that an islet of sclerosis invariably develops in the path for the ocular movements, and the ocular tremor is not only far more frequent, but it is more regular, and occurs earlier, than the jerky incoördination in the limbs. It is, as a rule, present only on movement, and occurs on movements in each direction, the quick displacement of the globes being in the direction of the voluntary movement. It is usually more marked in the lateral than in the upward movement, and is least when the eyes are directed downward. Often it is most energetic when the eyes are directed to one side, either the right or the left. Very rarely there is rotatory nystagmus, or lateral nystagmus on movement in one direction, and rotatory on movement in another. The patient may or may not experience an apparent oscillation of the objects looked at. Occasionally some conjugate movement of the eyes is distinctly weakened, generally a lateral movement, very seldom convergence. In some cases, again, there is considerable and increasing palsy of one of the ocular nerves, due to the development of sclerosis in the nerve trunk. The third or sixth nerve may be thus affected, seldom the fourth. Such palsy necessarily causes strabismus, double vision, etc. Transient diplopia is also occasionally complained of, even in the early stage of the disease.

Other cranial nerves may also become paralyzed, especially the facial, fifth, or hypoglossal nerves. In the affection of the first of these, all parts of the face suffer, and the degenerative reaction is well marked. In the fifth nerve, either one or both parts may be involved. The implication of the sensory part causes much less pain than is produced by most other lesions of the nerve trunk. The sclerosis that thus damages these nerves, may be either in the nerve trunk, near the surface of the brain, or in the pons or medulla, at the deeper origin of the fibres. Difficulty in swallowing is present when the pons or medulla is the seat of the morbid process.

Another very frequent symptom is a peculiar change in articulation. Syllables are unduly separated and unduly accentuated, in what has been termed "staccato," or "syllabic," or "scanning" utterance. Sometimes there is an undue separation of words and not of syllables. With this scanning articulation there may be a tendency to elide the ends of words. The patient seems to speak with effort, of which, however, he may be apparently unconscious. This change in articulation, while not invariable, is very frequent, even more so than the characteristic tremor, and only less so than nystagmus. The movement of the tongue is sometimes jerky and irregular, but the face is seldom involved, and the peculiar affection of speech is independent of any visible disorder of the movement of the tongue. It has been met with when the pons and medulla were free from sclerosis, and seems thus to result, in some way, from the disease of the cerebral hemispheres.

Slight mental change is also common, and somewhat characteristic. There may be failure of memory, but the most frequent alteration is an undue complacency and contentment, which, under the increasing helplessness and disability, is distinctly unnatural. There is not often any mistaken idea of improvement, although the patient may seize on and exaggerate any distinct diminution in the symptoms; but he is happy and cheerful in spite of increasing and grave disability. I think that this morbid complacency is more frequent in women than in men. Very rarely there is a greater degree of mental disturbance, which may take the form of almost any variety of chronic insanity.

Among other symptoms that are frequently met with, are headache and giddiness; both may occur early in the disease. The vertigo is especially common, occurring, according to Charcot, in three-quarters of the cases. The attacks may be very severe, and come on apart from any apparent cause. As in other forms of severe giddiness, it may be accompanied by vomiting. The attacks usually cease after a time.

Other paroxysmal disturbances, perhaps allied to the vertigo, are sometimes met with. There may be attacks of vomiting without giddiness, of palpitation, and also attacks of apoplectiform character, similar to those that occur in general paralysis of the insane. There may be simple coma, with heat of skin (even to 104°), or there may be unilateral convulsions, or the attack may be followed by transient hemiplegic weakness or rigidity. Occasionally the patient dies in such an attack, and, post-mortem (as in general paralysis), no acute lesion can be found to explain the symptoms.

The general nutrition of the sufferer from this disease is often unimpaired. Sometimes, indeed, there is a considerable increase in the amount of subcutaneous fat. Trophic disturbances have been observed in exceedingly rare cases—local œdema, altered secretion of sweat, changes in the growth of hair or nails, chronic arthritis, and herpetic eruptions; the last is perhaps sometimes due to the arsenic so often given in the disease.

COURSE AND DURATION.—In some cases the course of the malady is uniformly progressive from the first to the last. More frequently periods of apparent arrest alternate with periods of progress. The duration varies between two and fifteen years, the average being between three and six years. The most rapid case I have known was fatal a year and three-quarters from the onset. The most common cause of early death is interference with the functions of the medulla oblongata, especially impairment of deglutition, with its various consequences. In a few cases, death occurs from failure of respiration. In the cases of longer duration, the patients die from weakness, bedsores, kidney disease, etc., and in not a few instances death results, not from any effect of the disease, but from some intercurrent malady, as phthisis.

PATHOLOGY.—Compared with many of the other forms of sclerosis in the central nervous system, this variety is sharply distinguished by its irregular distribution. The islets are, as it were, scattered at random, and they thus present a marked contrast to the "system degenerations," in which a tract of sclerosis extends through the whole of a group of fibres having a certain function, and is sharply limited to these. This fact suggests that, whereas the latter sclerosis commences by a wasting of the nerve elements, and the overgrowth of connective tissue is secondary (see p. 311), in disseminated sclerosis the primary change must be the increase in the interstitial tissue, and the damage to the nerve fibres must be secondary. The conclusion is supported by all we know of the pathology of the disease, by the gradual way in which the white substance of the fibres wastes before the overgrowth of tissue about them, and by the absence of secondary degenerations until the local process has attained a considerable degree of intensity. The immediate local determining causes of the overgrowth cannot yet be even guessed at. It has been conjectured that it proceeds from the walls of the vessels, but this opinion is based upon pathological appearances that are seen in equal degree in system degenerations, and even sometimes in those that are purely secondary in nature. Hence it must be regarded as unsupported by any valid evidence. Whenever there is an overgrowth of interstitial tissue there is always a special increase adjacent to the vessels, and these may take a secondary share in the process, even when there is no primary vascular change (see p. 312).

It must be observed, however, that these two forms of sclerosis, the random islets and the systematic affection, are sometimes combined. The most common instance of this is the occurrence of primary atrophy of the optic nerves in disseminated sclerosis. This is a true system degeneration of the nerves, similar to that which occurs in tabes. It is possible that in some cases there is also a system sclerosis of the posterior columns of the cord in the lumbar region, so that tabes and insular sclerosis are combined. Hence, different as the two processes are in their immediate pathology, they may possess some common etiological relations.

Processes of acute or subacute inflammation leave behind them a considerable increase of connective tissue, and these connective-tissue elements are similar in character, whatever be the mode in which their increase came about. This correspondence has led some authorities to consider the process essentially one of chronic inflammation. An important and very obscure point in its pathology is, however, the relation of the lesion to less chronic lesions which are certainly inflammatory. We have seen that it may supervene on acute inflammation, primary or traumatic, but the sclerosis, in these cases, is separate in time and seems to be rather a sequel than an effect of the inflammation. A more difficult problem is presented by some special forms of inflammation. Disseminated myelitis, subacute in course, reaching its height in six weeks or two months, is characterized by islets of disease which, in their size and distribution, bear a close resemblance to those of insular sclerosis. The diseased areas are gray, and their aspect differs from that of sclerosis only in their less sharp definition and slighter consistence. If the patient lives, the resemblance of these to islets of true sclerosis may become still closer, and it is doubtful whether the two can be distinguished. Is this condition to be regarded as the same disease as insular sclerosis of slow development? An answer to this question cannot yet be given. As a rule, the inflammation is confined to the spinal cord and gives rise to spastic paraplegia; the jerky incoördination is absent. It is probable that some cases described as insular sclerosis confined to the cord have been of this nature.

Another lesion that presents a close resemblance to insular sclerosis in seat and character is the disseminated sclerotic inflammation of syphilitic origin, affecting the brain and cord, figured at p. 851 and at pp. 241 and 242. In chronicity and distribution the two morbid processes closely correspond, and it is doubtful whether their histological characters afford any ground for their separation. But an important difference is the presence of cheesy nodules in the syphilitic lesion, which stamp it as specific in nature, and are always absent in disseminated sclerosis.

It thus appears that insular sclerosis does not stand alone among the morbid processes of the central nervous system. It seems to occupy an intermediate position between the subacute and syphilitic inflammations, on the one hand, and the system scleroses on the other. Unless this view is accepted we must separate from insular sclerosis many cases at present classed with it, especially those in which the spinal cord alone suffers; and much of the clinical history of the disease as at present accepted will need revision.

Diagnosis.—The great characteristic of the disease is the combination of the jerky incoördination and progressive weakness in the limbs with nystagmus. In such cases the diagnosis is easy. The incoördination is much more jerky than it is in locomotor ataxy, and resembles a very coarse irregular tremor, more than does the incoördination of tabes. The only disease in which this symptom exists in precisely the same form as in disseminated sclerosis is cerebral tumor (see p. 888). But these cases are distinguished by other indications of a tumor of the brain, and real difficulty in diagnosis can only arise from ignorance of the occurrence of the symptom in such cases.

It is seldom that the diagnosis from paralysis agitans has to be made. The tremor in the latter disease is always regular and rhythmical, while that in disseminated sclerosis is irregular. In paralysis agitans there is usually characteristic rigidity, a stiffness of aspect and slowness of movement. Articulation is quick instead of slow. The fact that the spontaneous movements in shaking palsy occur equally during rest and movement, while those of sclerosis cease during rest, is often given as the most important distinction; it is true of most of the cases, but it is not an invariable distinction, because the tremor of true paralysis agitans, in the early stage of the disease, may cease during rest.

The diagnosis in which the spontaneous movements give rise to most difficulty, is between insular sclerosis and some cases of general paralysis of the insane, in which the twitching contractions of the muscles occur in the limbs with unusual intensity. They may disorder voluntary movements in a manner very similar to that seen in disseminated sclerosis. The chief difference is that the spasm is most intense in the lips and tongue in general paralysis, and that the speech presents a drawling character, with hesitating phonation, and special difficulty in the articulation of certain consonants—features quite different from the syllabic articulation of sclerosis. Nevertheless, the speech in general paralysis does not always present the usual disorder, and too much weight must not be laid upon any single symptom. Considerable mental change is exceedingly rare in sclerosis, while it is common in general paralysis. Inequality of pupil, and loss of the iris reflex, are in favor of the disease being general paralysis. The difficulty of the diagnosis in some cases is very great.

The spastic paraplegia that results from sclerosis in the dorsal region of the spinal cord is not distinguishable from that met with in other chronic lesions of the same part. If the disease is limited to this region, its diagnosis is not possible. Especially the distinction from it of disseminated dorsal myelitis may not only be impossible during life, but extremely difficult after death, since the naked-eye characters of the lesion are almost the same. The microscopical distinction is by the presence of inflammatory products in the foci of disease, and the greater abundance of the products of degeneration of the nerve fibres. The most important difference in the course of the two diseases is the subacute development of the paralysis in the disseminated inflammation.

Cases have been described by Westphal,* Leyden, and Langer,† in which symptoms closely resembling those of insular sclerosis have existed during life without any post-mortem lesion to which they can be ascribed. There is much doubt as to the exact nature of these cases. It is possible that most of them have been cases of general paralysis of the insane, the symptoms of which vary widely. In some cases the amount of mental change is exceedingly slight, and, as already stated, the disorder of movement may closely resemble that of insular sclerosis. In some cases of "pseudo-sclerosis," as the simulating cases have been termed, there have been syllabic utterance and nystagmus, symptoms commonly absent in general paralysis. But these symptoms are met with in some forms of central degeneration other than insular sclerosis, and too much diagnostic weight must not be laid upon them.

It is needless to say that a disease which often occurs in young adult females is sometimes mistaken for hysteria. The error is especially easy in the cases in which actual symptoms of hysteria preceded the onset of the disease, or occur, as they may do, during its course. The opposite error is, as usual, much more rare, but I have known a case of characteristic hysterical tremor to be mistaken for this disease and a hopeless prognosis to be given, even by a physician of considerable experience. In most instances there is no excuse for the former error. Nystagmus is absolute evidence of more than hysteria, and this is one of the many maladies in which the symptom is of great diagnostic value. Real difficulty is sometimes occasioned by the fact that an irregular tremor, occurring on movement only, is sometimes due to hysteria. While resembling that of sclerosis in irregularity, it is much smaller in range, and it is always accompanied by a tardiness of muscular effort, if the patient is desired to exert force, and there is the contraction of antagonistic muscles described in the section on hysteria, which is of so much diagnostic importance.

PROGNOSIS.—The prognosis of insular sclerosis is exceedingly grave. In any case in which the diagnosis is certain, only a steady increase of the disease can be anticipated. The only indication of the probable duration of the malady is that afforded by its observed rate of progress. In cases in which there are periods of apparent arrest, the duration of life is likely to be longer than in those in which the progress of the symptoms is uniform.

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* *Archiv f. Psych.*, xiv, pp. 87 and 767.

† *Wien. Med. Presse, 1884*, p. 698.

In the latter, it is not probable that life will be prolonged for more than two years from the time at which the symptoms are sufficiently distinct to permit the diagnosis to be made with confidence. In the former, the patient may live for several years. The symptoms of greatest gravity are those of damage to the nerves of the medulla oblongata, especially difficulty in swallowing. The earlier such symptoms come on, the shorter is likely to be the duration of life.

TREATMENT.—Insular sclerosis is a malady for which even less can be done than for the other degenerative diseases of the nervous system. The primary overgrowth of the interstitial tissue resists therapeutical influences more than does the primary degeneration of the nerve elements which constitutes the initial change in the sclerosis of systematic distribution. The treatment for the latter is, however, that which most deserves a trial in the insular form, especially the nervine tonics, such as arsenic and nitrate of silver, quinine, etc. The hypodermic injection of arsenic has been recommended by Eulenberg, but the evidence that this method of administration has a special influence is insufficient. Occasionally these drugs seem to retard the progress of the disease, but any arrest of its progress is only temporary. Hydropathic treatment, and the use of electricity in various forms, have been advocated in Germany, but their influence is exceedingly doubtful. In the case of a peripheral palsy, such as that of the face, due to sclerosis of the nerve, the question presents itself whether the muscles should be subjected to electrical treatment. As a rule, it is not worth while to give the patient the pain and inconvenience of the applications. The disease is progressive, and no recovery of the nerve can be looked for; hence electrical treatment, however prolonged, can have no influence in modifying the condition. It is, of course, desirable to maintain the patient's general health in as good condition as possible, and to avoid all depressing influences. In the case of women, pregnancy is especially undesirable; the malady is apt to make more rapid progress during the later period of pregnancy and after delivery.

DIFFUSE SCLEROSIS.

When the connective-tissue elements are increased through a considerable area in any part of the brain, the sclerosis is said to be "diffuse" in distinction from the small islets of the disseminated form. Sclerosis is very common as a secondary change. Many forms of softening, which do not result in any extensive destruction of tissue, lead to cicatricial induration, and an area of sclerosis results, sometimes with one or more cavities in its interior. Sclerotic induration follows the damage to the cortex produced by the meningeal hemorrhage that occurs during birth (see p. 801), and also the cortical lesion, probably venous thrombosis, that is a frequent cause of infantile hemiplegia. An acute lesion in early life may have, as its consequence, a hindrance to the development of the whole hemisphere, throughout which there may be a relative increase in the neuroglia and consequent induration.

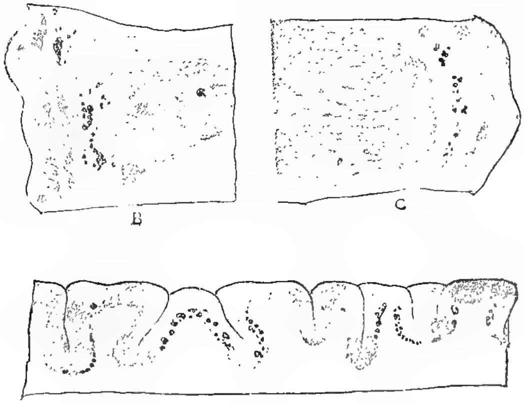
Apart from these forms of secondary sclerosis, there is sometimes a primary increase in the connective tissue through a more or less extensive tract in the brain, and after a time an increase in the consistence of the part. The change of color is much slighter than in insular sclerosis, and there may be no alteration ; so that the change can be recognized only by the touch. At the margin of the affected area there is no sharp demarcation, but the transition is gradual from the morbid to the healthy structure. It is said that the affected region may be at first increased in bulk and lessened in consistence, but it is rarely seen in this stage. Commonly there is induration, which may be slight or so considerable that the part is almost like cartilage. The change may affect the whole brain or one hemisphere, or only part of the brain. It may be limited to the cortex and subjacent white substance, or (as I have seen) may involve only a part of the white substance of the hemisphere. The causes of this condition are little known. Most cases have been congenital. I have once seen it in an adult, the subject of constitutional syphilis, in whose brain no ordinary syphilitic lesions were found. The recorded cases are at present very few, and the symptoms have varied much. In some cases there has been simple mental defect. In others there have been symptoms very similar to those of cerebral tumor, headache, vomiting, local palsy, convulsions, etc. It is doubtful whether the condition can be recognized during life. The separation of cases of general sclerosis from some forms of atrophy of the brain is uncertain.

MILIARY SCLEROSIS.

The term "miliary sclerosis" was first applied by Drs. Battey, Tuke and Rutherford to a lesion found by them in the white substance of an atrophied cerebellar hemisphere in an insane patient. The lesion was chiefly microscopic, and was only discovered after the brain had been hardened. It consisted of minute spots of degeneration, in which there was little evidence of a process deserving to be called "sclerosis." The term has been since used by others to designate another microscopic lesion, which was at first thought to be really pathological, but has since been abundantly proved to be of artificial origin, an "artefact," as the Americans say. The most common appearance is that in minute areas the tissue remains unstained by reagents, and presents a striking contrast to the well-stained vicinity. On close inspection, however, the tissue elements can be traced in the unstained area ; and it is evident that the sole difference between these spots and the adjacent tissue is in the absence of staining. This appearance is chiefly seen when spirit has been employed in hardening. The central organs ought never to be placed in spirit, at any rate after the first twenty-four hours. This is only one of many artificial changes produced by spirit, which are often very puzzling to the observer.

It is questionable whether the term "miliary" ought to be applied to a purely microscopic lesion ; it is more accurately used to designate changes resembling in size "miliary" tubercle. But that there is a form of sclerosis of the brain which is truly "miliary," is evident from one well-marked example that has come under my notice. In this case, throughout both hemispheres of the brain, and in all parts of them, the cortex contained minute reddish-gray spots at the junction of the gray and white substance. In tint they were a little darker than the darkest gray substance, and their aspect was exactly like that of the morbid tissue in insular sclerosis, and equally distinct in the recent state. In size they varied from that of a mustard seed, or a little larger, to the smallest point visible; large areas, one-eighth of an inch in length, were formed here and there by the coalescence of the smaller spots. Their size and distribution are shown in Fig. 313, A. None could be seen in the white substance of the hemisphere, but they were found in the central ganglia, especially in the outer part of the lenticular nucleus (B) and in the claustrum (C). None were found elsewhere in the brain, except two beneath the corpora quadrigemina. Under the microscope the centre of the larger areas consisted of a delicate fibrous tissue with abundant spider cells and small, round nucleated cells. In the fibrous tissue were small cavities, especially at the periphery of the larger spots and throughout the smaller ones, so that the tissue had a somewhat sponge-like aspect. No connection could be traced between most of these areas and vessels, but in places a similar change, of spongy aspect, could be seen along the side of a vessel, sometimes extending for a considerable distance. Thus, there was a wasting of the nerve elements, and an increase in the connective tissue in the affected areas.

FIG. 313.



Miliary sclerosis of the brain. Sections, A, of the cortex, B, of the left, and C, of the right lenticular nucleus and claustrum. The black dots represent the size and distribution of the minute foci of sclerosis.

I have not met with the record of any quite similar case, but an analogous lesion has been observed in a few cases of general paralysis of the insane, invisible, however, in the recent condition.*

In my case there was no mental derangement. The patient was a man, aged fifty, and the chief symptom was general weakness of the limbs, accompanied with some rigidity and a few left-sided convulsive attacks, beginning in the muscles of the shoulder. Then the speech became "mumbling" and unintelligible; coma set in; and after a few days of unconsciousness he died. He had also some cardiac weakness, enlargement of the liver and spleen, and general anasarca without albuminuria. The duration of the nervous symptoms was about ten weeks. No cause for the disease could be traced, but he had had constitutional syphilis many years before.*

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* See Grieff, *Archiv f. Psych.*, xiv, p. 287, and Simon, ib., Bd. ii.

ASSOCIATED PALSY OF THE BULBAR NERVES.

It has been mentioned that, among the cranial nerves, we may recognize two special groups, subserving associated functions and liable to associated disease. Each group is motor. One of these comprehends the nerves of the eyeball muscles, the associated palsy of which has been described at p. 615. The other group includes the nerves to the complex series of muscles of the upper part and orifice of the respiratory and alimentary canal—the mouth, fauces, pharynx, and larynx. These bulbar nerves arise from connected tracts of gray matter in the lowest part of the mesencephalon, or rather from the junction of this with the spinal cord. Their associated palsy has now to be considered. The most common form of this paralysis is due to a degenerative process in the nerve nuclei, analogous to that which, in the spinal cord, causes chronic muscular atrophy. Hence the pathological position of the disease is among the degenerations. But, as we have already seen, a palsy of similar seat sometimes results from an acute process in the same part of the brain. It will be convenient, therefore, briefly to describe the symptoms of the acute form in connection with that which is chronic, although the general relations of the acute lesions have been considered in the account of the similar morbid processes as they occur elsewhere in the brain.

The nerves, in the distribution of which the symptoms occur, are the fibres of the facial for the lower part of the face, especially for the orbicularis oris, the hypoglossal for the tongue, the spinal accessory for the larynx, and probably also for the palate, and the glosso-pharyngeal for the pharynx. To these is joined, in some cases, the pneumogastric. It deserves note that the facial occupies, as it were, an intermediate position between the two nerve groups above mentioned as liable to associated disease. The facial nerve supplies, on the one hand, the muscle that closes the eyelids, and on the other, the muscle that closes the mouth. But the orbicularis palpebrarum is not functionally associated with the eyeball muscles, and does not suffer with them in central disease, while the orbicularis oris, and the intrinsic muscles of the front of the tongue, have a functional association, closer, perhaps, than any other two muscles in the body. Neither can be put in action without the other (see p. 487, note). The orbicularis is always involved in degenerative disease of the hypoglossal nucleus, and escapes in disease of the nucleus proper of the facial, as in a case of acute atrophic ("infantile") paralysis in which the face was involved (see pp. 646 and 654).

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* Fuller details of this case will be found in the *Lancet, 1886*, January 23d, p. 145.

The central connection of the parts that are affected in the disease is well illustrated by the combination of paralysis of the tongue, palate and vocal cord that results from disease of the nerve roots on one side of the medulla (p. 707). The paralysis of the pharynx is only marked when the nerve roots on both sides are affected, and the paralysis is bilateral. In central disease of the medulla, the paralysis has the same distribution as in a lesion of the nerve roots, but it is almost always bilateral, and, in addition, the lips are paralyzed. They escape in disease of the roots, because the labial nerve fibres, although arising from cells in the medulla, ascend to the lower part of the pons, which they leave by the facial trunk. The most characteristic and complete distribution is seen in the cases of chronic degeneration in which the nerve cells suffer according to their functional nature. The chief centres concerned lie, however, so near together in the medulla, that random processes, such as softening or hemorrhage, may damage them on both sides, and cause palsy of the same distribution as that which results from primary degeneration. Hence all forms are included under the term "bulbar paralysis," and the varieties are distinguished as "chronic" and "acute." Only the chronic cases can be regarded as constituting a distinct malady; the acute forms depend on morbid processes that are frequent in other parts of the brain.

CHRONIC BULBAR PARALYSIS.

The chronic bulbar paralysis, the "labio-glosso-pharyngeal" paralysis of Duchenne, was first described in 1859 by Duménil, and systematically by Duchenne in 1860, while this, like so many of Duchenne's investigations, became widely known chiefly through the lectures of Trousseau. The awkwardness of the descriptive name given by Duchenne, led Wachsmuth, in 1864, to propose to call the disease "progressive bulbar paralysis," a designation that has come into general use, although it has not quite superseded the older term. The dependence of the symptoms on degeneration of the nuclei of the medulla was established especially by the labors of Charcot's pupils and of Leyden.

Causes.—The disease is essentially an affection of the second half of life, almost unknown in its pure form under forty. Most of the sufferers have been between fifty and seventy when the disease commenced. In exceptional cases it has been known to begin at thirty-six, thirty-two, and twenty-seven (Leyden), and even, after injury, as early as twenty.* Most cases of chronic bulbar palsy in early life are due to morbid growths and not to a primary degeneration. Males are rather more liable than females. Direct inheritance has not hitherto been observed, but indirect inheritance is probably a powerful predisposing cause, since other related neuroses may often be traced in the families of those who suffer. Thus, one patient had lost a brother from some form of slow progressive palsy affecting chiefly the limbs. It does not seem to be a consequence of syphilis. Immediate causes can be traced in only a minority of the cases; the most frequent are mental depression, exposure to cold, debilitating influences such as insufficient food, and injury, such as a blow upon the back of the neck. Over-use of the muscles has been supposed to be a rare cause; the symptoms developed rapidly in a man after playing on the clarionet for two whole nights, but the patient at the same time had been exposed to cold (Stein). In a considerable proportion of the cases, no cause, immediate or remote, can be traced.

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* In one case, symptoms suggestive of the degenerative disease, accompanied by palsy and rigidity of the limbs, developed slowly, after mental excitement, in a girl of twelve, but the nature of the lesion was not proved (Blumenthal, "Inaug. Diss.," Dorpat, 1884). A still earlier, but also irregular and incomplete case, is described by Stadthagen. After diphtheria, at four, the common paralysis of the palate was followed by wider symptoms of bulbar palsy;

The symptoms sometimes supervene on some other degenerative affection of the central nervous system, especially progressive muscular atrophy. The bulbar disease is indeed part of the spinal affection, the gray matter of the medulla undergoing the same degenerative change as that of the spinal cord.

SYMPTOMS.—The distribution of the symptoms, indicated by the name given to the disease by Duchenne, has been already mentioned. The lips, tongue, throat, and often the larynx, are paralyzed on both sides. The symptoms are, so to speak, grouped about the tongue as a centre, and it is in this organ that the earliest symptoms are usually manifested. In a few cases the distinct paralytic symptoms have been preceded by some discomfort or pain at the back of the head. The onset of definite symptoms is gradual; the cases in which there is a sudden or even acute onset are not examples of this disease. The first symptom is generally a trifling indistinctness of speech, due to imperfect articulation of those sounds in which the tongue is chiefly concerned, the lingual consonants *l*, *r*, *n*, and *t*, and afterward *s*. The commencing change in articulation is noticed only when the parts are fatigued by use, and the patient may be conscious that fatigue is produced with undue readiness. The tongue at first can still be protruded, although not quite so far as normal. The lips become weak with or soon after the tongue, and some difficulty in swallowing is added; the latter may, indeed, be the earliest symptom. In consequence of the weakness of the lips, the power of whistling is lost, and there develops a difficulty in the pronunciation of the sounds in which the lips are chiefly concerned, *o*, *u*, *p*, *b*, and *m*. The lips are not brought together so perfectly, or separated so promptly, as is necessary for the pronunciation of the labial explosives, and hence *b* and *p* become *m* and *v*. The power habitually exerted is generally less than the patient can exert if he tries. A word can often be articulated

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and when the patient came under observation, some years later, there was paralysis of the lips and palate, accompanied by weakness and contracture in the limbs (*Archiv f. Kinderheilk.*, Bd. v).

perfectly by a deliberate effort when the habitual articulation is very imperfect. The difficulty in speech is soon increased by the weakness of the palate, which ceases to shut off the nasal cavity. Swallowing gradually becomes difficult; the weakening of the tongue impairs the first part of the act of deglutition, that in which the food is rolled back into the pharynx by the application of the tongue to the roof of the mouth; the paralysis of the palate permits the regurgitation of liquids into the nose; and the weakening of the pharyngeal muscles further interferes with the movement of the food.

As the paralysis increases, closure of the mouth becomes impossible, the lower lip drops, and the muscles that move the angle of the mouth often become feeble. Hence the aspect of the lower part of the face changes, and indeed all expressional movement of the face below the eyes may cease. Saliva dribbles out of the open mouth, and the patient has to keep a handkerchief constantly in his hand.* The appearance of the patient is characteristic, and still more so when he attempts to speak. The tongue can no longer be protruded; only the tip can be projected over the lower teeth. Mastication is difficult, because the tongue can no longer guide and keep the food between the teeth. Articulate speech at last becomes impossible, and the only expression remaining to the patient is laryngeal phonation, slightly modulated, and broken into the rhythm of formless syllables, the meaning of which can be hardly more than guessed at. Swallowing becomes more and more difficult. Semi-solids are usually swallowed better than liquids, because there is less risk of their escape into the nose or larynx. The difficulty in swallowing the saliva increases the flow from the mouth. The muscles of the larynx, if not before, now usually become weak. The epiglottidean muscles fail to reflect the epiglottis during the act of deglutition, and particles of food readily enter the opening. The muscles of the vocal cords also suffer; the voice is low pitched, from the defective approximation of the cords, and the cough is imperfect, from the feebleness of closure. The laryngeal palsy rarely becomes complete, and it is still rarer for the power of abduction to be specially lost, common as abductor palsy is in some other forms of central degeneration.

There is no loss of sensibility in any of the affected parts, and taste is unimpaired, but the reflex action in the throat, so active in health, is usually lost. The palate or fauces, and even the larynx, can be touched or tickled without exciting the spasm that is normally produced. Sometimes this loss of reflex action precedes the other symptoms of the disease (Krishaber). It increases very much the risk of the entrance of food into the larynx. The impairment is apparently due to the affection of the motor section of the reflex arc. But reflex action is not invariably lost; Erb twice found it preserved in the palate and pharynx to a late stage of the disease.

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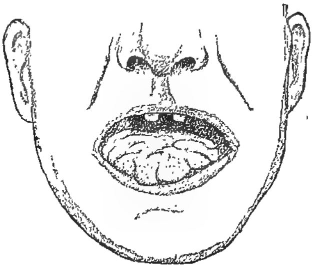
* It is often thought that there is an increase in the secretion of saliva, and "salivation" is said to exist, but it is doubtful whether the amount is larger than the normal. A pathological increase has been asserted very strongly by some authors; Schulz supposed that, in one case under his care, the secretion was increased to six times the normal quantity.

Alterations of nutrition are chiefly visible in the tongue, and its condition varies much in different cases. In some the organ is large and broad throughout, although soft and flabby to the touch. In other cases it is conspicuously wasted, and the surface is deeply furrowed and wrinkled, from the loss of the muscular substance (Fig. 314). In some cases the lips retain their normal size; in others they are conspicuously thin. Electric irritability is little changed; the muscles, even when greatly wasted, still react to faradaism with readiness. Erb has found an indication of the reaction of degeneration, an increased and altered reaction to voltaism, and this although the faradaic excitability was normal—the mixed (or "middle") form of reaction (see p. 45). Symptoms of motor irritation, such as convulsion or spasm, are never present.

FIG. 314.



Wasting of the tongue, and maximum' protrusion, in a case of chronic bulbar paralysis.

The intellectual faculties, as a rule, are unimpaired. The only alteration that is common is a curious emotional mobility; in consequence of this, laughter or tears are evoked by trifling causes, and the immobile face and unmodulated tone may render the laughter strangely grotesque. But the mental functions present no change; the unhappy sufferer is painfully conscious of his state; and his distress is increased by the inability to express it, except by the laryngeal gestures to which speech is reduced.

Progressive bulbar paralysis, already mentioned, is often associated with progressive muscular atrophy in the limbs and trunk, accompanied, it may be, with the symptoms of lateral sclerosis of the cord. Either the bulbar or spinal symptoms may lead the way, and dominate the aspect of the case. Toward the close of a general progressive atrophy the mouth and throat muscles may suffer, or when these are first and considerably affected, wasting may show itself in the muscles of the limbs. Less commonly, bulbar paralysis is associated with spastic paralysis of the limbs and slight wasting; lateral sclerosis is then found after death, with only slight changes in the anterior horns.

Even in cases not accompanied by general atrophy, the paralysis may spread beyond the muscles of the lips, tongue and throat. The muscles of the upper part of the face and of the eyeballs are scarcely ever involved.* Excessive frequency of the pulse (150–160) is sometimes observed toward the close, and attacks of dyspnœa have resulted, in rare cases, from the extension of the degeneration to the respiratory centre. It is rather strange that glycosuria seems never to result from the degenerative changes beneath the floor of the fourth ventricle that constitute this disease. This may be another instance of the way in which functional relation determines the distribution of the morbid process. Occasionally the muscles of mastication become weak.

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* Nevertheless, cases of progressive ophthalmoplegia may be ultimately complicated by bulbar paralysis.

Among the indirect effects of the disease, are the weakness and emaciation that result from the imperfect supply of nourishment, consequent on the difficulty of swallowing, and the bronchitis that is apt to be set up by the passsage of particles of food into the air passages.

The *Course* of the disease is always progressive, although not uniformly so. From time to time its progress is retarded, and it may seem, for weeks or even for months (seldom for a year or so), to be at a standstill. The hopes thus excited are usually baseless, for the disease again increases, often at a more rapid rate. An intercurrent malady, which lessens the patient's strength, accelerates the progress of the disease. Its duration is usually from one to three or four years. In one case it lasted for seven years (Leyden).

The *Causes of Death* are gradual weakness from inanition; bronchitis and broncho-pneumonia, from the entrance of food into the air passages; failure of respiratory power from associated atrophy of the muscles of the trunk, or from invasion of the respiratory centre in the medulla; or failure of the heart's action from the extension of the degeneration to the cardiac centre of the vagus.

PATHOLOGY.—In the affected muscles there are changes similar to those seen in progressive muscular atrophy of the limbs. The fibres may present extensive granular and fatty degeneration, or may be simply narrowed. Usually some fibres are much affected, and others but little, so that empty sarcolemma sheaths and normally striated fibres may lie side by side. There is an increase of the nuclei of the sheaths and of the interstitial tissue, and the latter may present an abnormal accumulation of fat. Masses of reddish pigment, the product of the degeneration of the fibres, also lie between them. The nerve endings in the muscle are degenerated. The motor nerve trunks are gray and soft, and the microscope shows degeneration of the nerve fibres and increase of the interstitial tissue.

The medulla oblongata itself generally appears normal to the naked eye; rarely there is slight diminution in bulk. The important morbid appearances are revealed only by microscopical examination. There may be distinct atrophy of the fibres of the hypoglossal and other nerves within the medulla. In the motor nuclei, changes are found quite similar to those presented by the gray matter of the cord in progressive muscular atrophy. There is wasting of the nerve cells, many of which lose their processes and become reduced to small angular bodies. The interstitial tissue becomes altered, and contains granule corpuscles and other products of the degeneration of the nerve elements; sometimes there is a conspicuous increase in the connective-tissue elements, spider cells, etc.; the walls of the vessels may be thickened. Degeneration is often found in the anterior pyramids, just as the pyramidal tracts are commonly degenerated in the corresponding affection of the anterior cornua of the spinal cord, which so often coexists with the bulbar degeneration. In such combined cases, the sclerosis of the pyramidal fibres may be traced through the pons and cerebral peduncles. When, as is commonly the case, there is muscular atrophy elsewhere, the spinal cord presents corresponding alterations in the gray and white substance. It is probable that, in some cases of bulbar paralysis, the disease is confined to the pyramidal fibres, as in cases of lateral sclerosis of the spinal cord causing spastic paralysis. The distribution of the degeneration in the medulla is fairly uniform; it is most considerable and most constant in the hypoglossal nucleus, next in the adjacent part of the nucleus of the spinal accessory nerve, and it is usually found, in less degree, in the nuclei of the vagus and glosso-pharyngeal, rarely in that of the motor part of the fifth nerve, and in the chief facial nucleus. Wasting of the nerve cells has also been observed in the "nucleus ambiguus" (X, Fig. 205, p. 484).* The degeneration is always bilateral, and corresponds very closely with the distribution of the symptoms. The grouping according to function makes it practically certain that the process begins in the nerve elements and follows their functional relations in its extension. The exact degeneration on which the paralysis of the orbicularis depends has not yet been clearly made out. Lockhart Clarke believed that he had traced a descending portion of the facial nucleus to the neighborhood of the hypoglossal, and that it was the disease of this part that caused the paralysis of the lips. Later researches have failed to establish the existence of this nucleus, but it is highly probable that the orbicular fibres do descend to the neighborhood of the hypoglossal (see p. 486), although it is impossible to trace them among the many horizontal fibres of the reticular formation in which they lie. The fibres of the anterior pyramids and pyramidal tracts in the pons and crura have also been found degenerated, but in the cases in which this has hitherto been traced, there has also been atrophy or palsy of the limbs, and then the degeneration of the pyramids is the rule (see p. 365).

The marked contrast presented by the tongue in different cases—in some small, wrinkled, and shrunken, in others large, broad, and flabby—led Duchenne to divide the cases into two varieties, the atrophic and the paralytic. Most later writers have rejected this distinction, on the ground that there may be considerable atrophy of the muscular fibres without shrinking, because an overgrowth of interstitial fat compensates for the lessened bulk of the muscular tissue. It is not certain, however, that the difference between the two sets of cases can be always thus explained. It is possible that in some of the cases without obvious wasting, the primary disease is not in the nerve nuclei but in the fibres that connect these with the cerebral hemispheres or in the lower extremities of these fibres, within the nuclei. We have seen that the pyramidal fibres may be degenerated, and these fibres are homologous with those that constitute the upward path from the nuclei to the cortex. In cases in which these fibres, and not the nuclei, are diseased, there should be no loss of reflex action, and we have seen that, in some cases, the reflex action is preserved. This feature would distinguish such cases from those of nuclear disease in which real wasting is obscured by overgrowth of fat.

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* Petersson, *Neur. Centralbl.*, *1886*, p. 376.

Atrophic bulbar paralysis must be regarded as an affection practically identical with the progressive muscular atrophy that is so often associated with it. The difference between them depends only on the seat of the morbid process, and not at all on its nature.* The disease is a degeneration of the lower segment of the motor path for the muscles affected (see pp. 130 and 367), and in some cases it appears to be a degeneration of the whole path, upper and lower segments. Although an affection of the cortical cells has not yet been found, it is highly probable from the analogy of progressive muscular atrophy (see p. 366). In other cases it is probable that the malady is the bulbar homologue of primary lateral sclerosis of the spinal cord, and that the degeneration is limited to the upper segment of the path.

Diagnosis.—The distribution of the palsy, its bilateral character, its gradual onset and progressive course, separate the disease with sufficient sharpness from most other maladies. The slow onset distinguishes it from acute lesions of the medulla, which may cause the symptoms of similar character and distribution described in the next section. The chief difficulty is presented by organic diseases of the medulla, which may cause "bulbar palsy" of slow onset. The most frequent of these is a tumor in or outside the medulla, damaging it by invasion or compression, or compressing the nerve roots, but the symptoms thus produced seldom present the perfect bilateral symmetry that characterizes the degenerative affection. One side is affected first or most, the difficulty of swallowing preponderates over the other symptoms, and the lips usually escape altogether. Moreover, headache is usual, and convulsions are occasionally met with.

Tumors within the medulla sometimes give rise to greater difficulty, but the cases in which a growth acts on both sides in such an equal manner as to cause perfectly symmetrical symptoms are excessively rare, and there are usually other indications to help the diagnosis—either there are other indications of a tumor, or the patient is so young as to render the degenerative disease highly improbable. Insular sclerosis, involving the medulla, is seldom so symmetrical as to give rise to real difficulty, and there are always indications in other parts of the morbid process. Chronic lesions in both cerebral hemispheres may cause symptoms resembling those of bulbar palsy, although such an effect is far more rarely caused by chronic than by acute lesions; the chief distinctions are afforded by the development of symptoms first on one side and then on the other—the affection of the limbs, as well as the face, in distinct double hemiplegia—and by other symptoms indicative of the nature of the morbid process. Reflex action is preserved in the parts paralyzed, and these are never wasted, but alone these points are not sufficient for the diagnosis, although they may be allowed weight in support of other indications.

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* The essential identity of the two diseases was first urged by Kussmaul (*Volkmann's Clin. Lect.*, No. 54, 1873).

Prognosis.—In every case of gradual onset the prognosis is most grave. The affection is progressive in its tendency, and the parts affected are so important for life, that the disease almost invariably leads to death. It is doubtful whether any chronic case has been cured or even permanently arrested.

Treatment.—Although experience shows that we can scarcely expect, in any case, that our treatment will have an appreciable influence on the disease, we may strive at least to retard its progress, and employ such measures as, from their action on the nervous system, are most likely to produce this effect. It is probable that cases will occasionally be met with in which the morbid tendency is less strong than it usually is, and in which treatment may have some influence. Nervine tonics, quinine, strychnine, arsenic, phosphorus, or nitrate of silver may be given, or hypodermic injections of strychnia ($\frac{1}{60}$ gr.) may be administered, as recommended for progressive muscular atrophy (p. 372). In advanced cases I have known transient improvement to follow the injection of strychnia combined with minute, stimulant doses of morphia ($\frac{1}{36}$–$\frac{1}{24}$ gr.). The power of swallowing has been increased by this treatment, but unfortunately the effect has not been permanent. To lessen the flow of saliva, belladonna or atropine may be given.

Electricity has been largely employed. With the view of influencing the morbid process in the medulla, the voltaic current has been passed from one mastoid process to the other. To the muscles, either faradaism or voltaism may be applied, for they respond to either in the majority of cases. It may be applied to the tongue, lips or pharynx, the latter by placing the positive pole at the back of the neck, and moving the other along the side of the pharynx. Electrization of the sympathetic has also been used as a method of treatment, but is probably as destitute of rational foundation as it certainly is of practical effect. Indeed, the result of all electrical treatment is very unsatisfactory. For an hour or two after each application there may be a little more power, but the effect does not last. I have never observed satisfactory evidence that electricity had the slightest influence on the course of the disease in any one of many cases in which I have seen it used.

In all cases the feeding of the patient demands much care. Semi-solid pulpy food can usually be swallowed better than liquids or solids. When deglutition is impossible, liquid food must be given by an œsophageal tube, or by a catheter or soft india-rubber tube passed through the nose into the upper part of the œsophagus. The food may be slowly poured into the tube through a small funnel. The only alternative is the less effectual method of rectal feeding with peptonized food. The larynx has been opened in one case, in which attacks of threatened suffocation were frequent and severe.

SUDDEN (APOPLECTIFORM) BULBAR PARALYSIS.

It has been already mentioned that various acute lesions of the medulla cause symptoms in the region of the nerves implicated in the degenerative form, and that these lesions generally differ from the latter in the irregularity of the grouping of the symptoms produced. Occasionally, however, a sudden lesion causes symptoms that are perfectly symmetrical, and correspond very closely to those of degeneration of the bulbar nuclei. Almost all the sufferers have been advanced in life—at the period in which arterial degeneration is common, but similar symptoms have been observed in younger persons as a result of blows and falls on the head or neck.* The onset is sudden and apoplectiform, sometimes with giddiness and vomiting, usually without loss of consciousness. The initial symptoms may be of wider range; there may be weakness of the limbs or affection of sensibility, usually in the form of subjective sensations, rarely actual anæsthesia. These wider symptoms pass away, and leave a permanent condition closely resembling that of the degenerative form, but differing in that there is no progressive tendency. For a time, indeed, there is improvement; the symptoms lessen, sometimes much, sometimes little; and then the condition remains stationary, it may be for many years. Occasionally, after a time degenerative changes may supervene on the original acute lesion, and then the case assumes a progressive character resembling the primarily degenerative form. Now and then the symptoms, although bilateral, are not perfectly symmetrical. Very rarely they are one-sided, as in a case recorded by Hirt, in which paralysis of one vocal cord and of the corresponding side of the tongue came on suddenly; there was wasting of the tongue with the reaction of degeneration.† In other cases, again, the symptoms, although bilateral, are more irregular in their distribution than in the typical form. The effects of random lesions of medulla necessarily vary considerably in different cases.

As an example of this sudden form may be mentioned the case of a man aged sixty-three. At fifty-five he had a slight attack of right hemiplegia of ordinary type, without affection of speech or of swallowing, from which he recovered perfectly in the course of a few months. Five weeks before being seen, he suddenly became unable to articulate and had great difficulty in swallowing. There was no loss of consciousness. No change in his condition had occurred when he came under observation. His condition then resembled perfectly that of the progressive degenerative form. His lower lip hung down; saliva constantly dribbled from the mouth. The tongue was broad, flabby and almost motionless, only the tip could be brought over the lower teeth (see Fig. 315). The palate was flaccid, but could be raised a little. Swallowing was very difficult and attacks of choking were frequent. The vocal cords could be brought together, although with little force, so that no explosive cough was possible. He could still phonate, although with little modulation. There was not the slightest power of articulation; attempts to speak resulted only in "ah-ah-ah." Expression by writing was unimpaired. This patient was seen from time to time for five years after the onset, and his condition remained essentially unaltered. His power of swallowing varied; sometimes it became less, and then under treatment it improved again, but on the whole the loss of articulation and of swallowing, the paralysis of lips, tongue and larynx, were neither better nor worse at the end of the five years than they were five weeks from the onset. There was no wasting and no loss of faradaic irritability. As in this case, the muscles involved are usually those supplied from the medulla oblongata only, but a curious case has been recorded by Dixon Mann, in which there was also paralysis of the muscles of mastication.*

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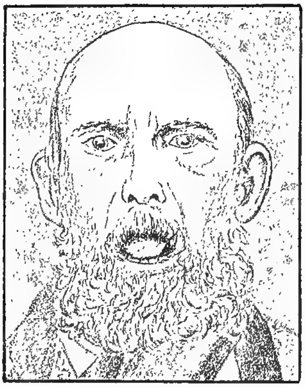
* As in a case in a boy, aged twelve, recorded by Schulz (*Neurol. Centralbl.*, *1883*, p. 99), in which bulbar paralysis followed immediately a blow on the back of the neck, and increased considerably a day or two afterward. It is assumed that there was traumatic hemorrhage, followed by secondary inflammation. Compare a case of fatal traumatic hemorrhage recorded by Bochefontaine, *Arch. de Phys.*, *1883*, p. 160.

† Hirt, *Berlin. klin. Wochenschrift*, *1885*, No. 26.

FIG. 315.



Bulbar paralysis of sudden onset; maximum protrusion of the tongue.

The pathology of these cases rests at present on little exact observation. It is probable that they depend on softening from vascular occlusion situated in or near the middle line, and, at any rate in the cases with muscular wasting, damaging the nuclei that are the seat of degeneration in the progressive form. The position of the lesion in cases such as that detailed above, in which there is no wasting and no loss of electrical irritability, *i. e.*, no evidence of damage to the nuclei themselves, is uncertain. It is possible that its seat is just above the nuclei, so as to damage the paths from the cerebral hemispheres, which probably decussate near the nuclei, and would therefore be liable to common damage from a single lesion in the middle line. Thus, in a case described by Leyden, there was an area of softening in the middle of the line at the level of the olivary bodies.† It would seem, however, that bilateral symptoms sometimes result from a one-sided lesion. The functional connection between the nuclei of the two sides is exceedingly close; they habitually act together in perfect equality, and destruction of those on one side may for a time interfere with the function of their fellows on the other side. In most cases of the kind the duration of life has been short, and it may be that the muscles of the other side would ultimately have recovered.

The *Diagnosis* of these cases presents little difficulty. They are sudden lesions of the medulla, and the cases in which the symptoms are symmetrical are not strictly separable from the others just mentioned, in which symptoms of similar character are of less regular and symmetrical arrangement. The distinction from the degenerative form rests on the mode of onset, sudden in the one case, gradual in the other. It is to be noted, however, that a sudden increase of the paralysis sometimes occurs in the slow form, but preceding symptoms distinguish these cases from the acute variety.

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* Dixon Mann, *Brain*, July, 1884, p. 244. It is very difficult to explain this combination of symptoms except on the hypothesis of a double lesion.

† Leyden, *Arch. f. Psych.*, vii, p. 44.

There is, however, one great diagnostic difficulty in connection with these cases. The "pseudo-bulbar paralysis" mentioned on p. 716, may simulate very closely the symptoms of a sudden lesion of the medulla.* In most cases of the kind the indications of a lesion in each cerebral hemisphere are clear. There are two distinct attacks, causing symptoms first on one side and then on the other, and it is the second attack that leaves the paralysis of the lips, tongue and pharynx that simulates a lesion in the medulla. In most cases of the kind the disease has been in the central ganglia, especially in the lenticular nucleus; less commonly it has been in the white substance, or in the lower part of the motor cortex, or in this part on one side, and in the central ganglia on the other. In cases of this character, the diagnosis is not difficult if the observer is aware of the occurrence of bulbar symptoms. If such symptoms follow the indications of a second lesion, and the case presents the negative indications presently to be mentioned, the case may be assumed to be one of "pseudo-bulbar paralysis." A much greater difficulty is presented by rare cases in which there are not two successive attacks, but the bulbar symptoms follow a single apoplectiform seizure. In some instances lesions occur simultaneously in both hemispheres; in others, a subsequent post-mortem examination has shown that only one hemisphere of the brain was diseased, and the lesion has sometimes been on one side, sometimes on the other. In several of these cases the disease was in the lenticular nucleus. It has even been assumed that this nucleus has a special relation to the processes of articulation and deglutition,† an assumption which is scarcely consistent with the frequency with which it has been found diseased without the occurrence of bulbar symptoms. The implication of the internal capsule can seldom be excluded in lesions of the nucleus. Another hypothesis is that in such cases of one-sided lesion there is an exclusive relation of these processes to one hemisphere, owing to some congenital structural peculiarities.‡ On the other hand, it has been held that undetected microscopical lesions of the medulla are really answerable for the bulbar symptoms in these and many other cases of pseudo-bulbar paralysis.§ It is certainly important to remember that disease, causing hemiplegia and death, sometimes escapes discovery, but we must admit that the mechanism by which the bulbar symptoms result from one-sided lesions is still practically unknown.

Besides the distinction often afforded by the mode of onset, these cases are characterized by the slighter degree of the symptoms, and by the facts that the nutrition of the tongue is unimpaired, reflex action is undiminished, and there is no change in electrical irritability. The larynx is seldom paralyzed. But, inasmuch as these negative characteristics are sometimes present when the lesion is in the medulla, their diagnostic value is not absolute. The fact that a second lesion may be in the medulla must also be borne in mind, and must occasionally lessen even the moderate amount of confidence with which the diagnosis of pseudo-bulbar paralysis can be made.

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* Cases of this character have been described by T. Barlow, Joffroy, Lépine, Berger, Jolly, Ross and others.

† Ross, *Brain*, July, 1882.

‡ O. Berger, *Breslauer Arztl. Zeitschr.*, *1884*.

§ Oppenheim and Siemerling, 59th *Versam. Deut. Aerzt.*, *1886*.

Prognosis.—Sudden apoplectiform bulbar paralysis involves much danger to life in the early stage of the disease, but the subsequent prognosis is less grave than in the chronic degenerative form. The tendency of the symptoms to progress is slight or absent. In many cases considerable improvement occurs ; some recover entirely. In others, as in that described above, little or no power returns in the paralyzed parts. The only guide as to the probable course of the symptoms is that afforded by the condition of the patient when the acute stage has passed. If at the end of a month there is no sign of improvement, and there is still a considerable degree of paralysis, it is improbable that much improvement will ensue.

The *treatment* of this form is essentially that of acute softening from vascular occlusion, and is described in detail at a previous page. The general management of the case in regard to feeding, etc., is the same as in the degenerative variety.

ACUTE (INFLAMMATORY) BULBAR PARALYSIS.

The term acute bulbar paralysis is commonly applied to the sudden form, described in the last section. But there is yet a third variety, exceedingly rare, in which the symptoms develop not suddenly, not in a few minutes, but acutely, in a few days. The mode of onset is that which suggests an acute inflammation, and that this is actually the lesion is proved by a case observed by Etter.* A boy, aged fifteen, was taken ill with headache and vomiting, discomfort in the throat, difficulty in swallowing, and fever. In the course of the first week there developed bilateral paralysis of the face, accompanied by palsy of the tongue, greatest on the left side, paralysis of the palate, and of the left sixth nerve. Death resulted from pneumonia on the tenth day. The post-mortem and microscopical examination revealed many myelitic foci in the medulla, symmetrical on the two sides, but greater on the left. They involved the left sixth nucleus, the left facial nerve within the pons, and the right facial nucleus, the left hypoglossal nucleus and the right hypoglossal fibres, and the accessory nucleus on each side, the motor part of the vagus nucleus on each side, and many cells and fibres in the tegmental region of the lower part of the pons. The foci of inflammation in the accessory nuclei could be traced down the cord as far as the fourth cervical nerves. Such a lesion is evidently closely analogous to that which, in the spinal cord, causes acute atrophic paralysis. We have seen (p. 616) that a similar inflammation may involve the nuclei of the ocular nerves in the upper part of the pons.

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* *Corresp.-bl. f. Schweizer Aerzte, 1882*, No. 24.

The treatment of such a case must be that of other forms of acute inflammation of the brain.

ATROPHY OF THE BRAIN.

The whole or part of the brain may be below the normal size. In general atrophy, the texture of the brain is normal; the whole brain is small, and the skull is also small (microcephaly). Little is known of the causes of this condition, whether the small size of the brain is the cause of the small size of the skull, or the reverse. The condition is generally associated with a high degree of mental defect.

In partial atrophy of the brain, one part is unduly small in proportion to the rest. The part so affected is usually changed in structure. It is firmer or softer than normal, and contains more connective tissue and fewer nerve elements. Almost any part of the brain may be thus affected—the whole of one cerebral hemisphere, or only part of it, the central ganglia on one side, the pons, or the cerebellum. Atrophy of the cerebellum may involve the whole or only one hemisphere, or both hemispheres—the middle lobe being normal. When the whole of one cerebral hemisphere is atrophied, one part is usually more affected than the rest. Several parts of the brain are frequently atrophied together, in a manner that shows a causal relation—that the atrophy of one entails that of the other. Thus atrophy of the whole of one cerebral hemisphere is usually associated with atrophy of the opposite cerebellar hemisphere. The latter may be associated with atrophy of the opposite corpus striatum without the rest of the hemisphere, and it is usually associated also with atrophy of the opposite olivary body of the medulla oblongata.

Partial atrophy of the brain is probably in most cases acquired. This is clear in many instances in which it is attended with symptoms that date from the first years of life. In some of these cases the lesion dates from birth, and the change in the brain is the result of its compression by local meningeal hemorrhage (see p. 801). In other instances the lesion occurs during infancy, and is accompanied by "infantile hemiplegia," in the account of which (p. 839) will be found some evidence as to the nature of the disease. In some cases, again, it is probable that an attack of meningitis was the cause. Both hemispheres may suffer from this cause, and also in the cases of meningeal hemorrhage during birth. In all cases the local atrophy is often accompanied by a general diminution in size of the hemisphere, no doubt due to the connection that exists between all parts, and to the effect of considerable damage to one part of the growing brain on the development of the rest.

Partial atrophy of the brain may also develop during intra-uterine life. It may be found present at the time of birth. In some cases it is, perhaps, due to intra-uterine disease similar in character to that which, after birth, has similar consequences. More frequently the atrophy is such that it can scarcely be thus explained. Atrophy of the cerebellum is more frequent in these cases than is atrophy of one cerebral hemisphere. Sometimes both cerebellar hemispheres are very small, and the middle lobe is of normal size. It is very difficult to explain this condition on the supposition of any intra-uterine disease. The condition is probably connected with the entire absence of the cerebellum sometimes observed, and is due to some perversion of the process of development, the cause of which is practically unknown.

The symptoms that attend atrophy of the brain vary much. Mental defect is the most constant, and usually amounts to idiocy. Hemiplegia and epileptic fits often accompany atrophy of one cerebral hemisphere. Bilateral weakness, with athetoid movements and incoördination, may attend bilateral atrophy such as is due to difficult birth. In many cases, however, it is scarcely correct to regard these symptoms as the consequences of the atrophy; they are the result of disease of the motor region, of which the general atrophy is also the result. In atrophy of the whole of the cerebellum, unsteadiness has been observed similar to that which results from disease of the middle lobe. When only the hemispheres have been affected, there have been no motor symptoms, but in some cases there was intellectual defect. Atrophy of one hemisphere of the cerebellum has been found when no symptoms, that could be ascribed to it, were present during life.

Senile Atrophy.—In old age the brain wastes, like many other organs, and becomes smaller and firmer. The amount of fluid in the ventricles and on the surface becomes increased in proportion to the lessened bulk of the brain. This natural increase of fluid was formerly regarded as pathological, and a mysterious death was ascribed to "serous apoplexy," a purely imaginary lesion. This wasting of the brain is commonly attended by no symptoms. Senile mental failure is often ascribed to it, but since it may exist in considerable degree without the slightest mental defect, caution should be observed in attributing to it any mental change that may coexist.

HYPERTROPHY OF THE BRAIN.

Under the name "hypertrophy of the brain," a condition has been described in which the brain is of abnormal size. Sometimes it has been found also to be of distinctly abnormal weight, but the wide variations in the weight of the brain under normal conditions render this criterion decisive only in extreme cases.* The size of the brain in relation to the size of the skull is the feature that has attracted most attention. If the condition develops before the bones have united, the skull is said to undergo enlargement similar to that of hydrocephalus. If the sutures have been closed, the convolutions are compressed and pale, and when the brain has been removed it cannot be replaced in the cranial cavity—a feature which has, since the days of Morgagni, attracted attention, perhaps more than it deserves. In all cases the ventricles are empty; their walls are pressed together, and the vessels of the brain contain little blood.

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* The weight and size of the brain differ according to age and stature, but even thus tested the variations are great. Proportion to absolute body weight has also been taken as a guide, but it is not to be relied on, since the body weight varies in each direction, that of obesity and of emaciation, irrespective of stature. If the body weight is taken as a term of comparison it should be the average for each age and stature.

Decisive examples of this state are extremely rare; there are few satisfactory observations on the minute structure of the enlarged organ, and we do not therefore know whether this is the same in all cases. In some an increase in the neuroglia has been found, and has been regarded as the cause of the enlargement (Virchow; Tuke, in an unusual case in which the enlargement was confined to one hemisphere). In other cases no increase in the connective tissue could be found.

Such enlargement of the brain has been met with chiefly under three conditions: (1) In very young children soon after birth. It is in these that the hydrocephaloid enlargement of the head has been described. (2) Toward the end of the first year of life, in association with rickets. It may be that the closure of the skull being retarded, the brain attains an abnormal size in consequence of the slightness of the mechanical restraint that should be supplied at a certain age by the closure of the skull. It is doubtful whether, in these cases, the enlargement in the brain increases the size of the skull. The skull does become large, but it has the form characteristic of rickets. It may be that this is due not only to the abnormal process of ossification, but also to the influence of the brain upon the soft skull. (3) Enlargement of the brain has been described occasionally in older children and adults, but of its nature very little is known.

Of the causes of this condition only two facts can be regarded as established: first, that it has been occasionally observed in families; secondly, the relation to rickets already described.

The symptoms that it produces are very uncertain. The form that occurs soon after birth and causes enlargement of the head, is said to be attended by nervous symptoms almost identical with those of chronic hydrocephalus, so that the distinction of the two is practically impossible. It is doubtful whether the rickety enlargement causes any symptoms. We are not justified in referring to it the laryngeal spasm and general convulsions and mental backwardness of rickety children, since these may be present when there is no enlargement of the brain.

Even more doubt exists as to the symptoms of the form that has been met with at a later age. In some cases it has apparently caused none. In others, acute cerebral symptoms, resembling meningitis, have occurred, and have run a rapid course, ending in death. The evidence of a connection between the enlargement of the brain, which was regarded as chronic, and the acute symptoms is not satisfactory, and no explanation of the supposed connection has been attempted. The condition seems never to have been recognized during life, and nothing is known of its treatment.

The so-called hypertrophy of a small part of the brain, as of certain convolutions, or of the pons Varolii, have probably been always cases of an infiltrating growth (see p. 876).

From what has been said, it is clear that the pathology of enlargement of the brain needs reconsideration in the light of fresh investigation. To describe enlargement from overgrowth of connective tissue as "hypertrophy of the brain" is inconsistent with the proper use of the word "hypertrophy," which, when applied to an organ without qualification, should designate only an overgrowth in which the elements on which the function of the organ directly depends bear their proper proportion to the interstitial tissue.

HYDROCEPHALUS.

Hydrocephalus, or dropsy of the brain, consists in an accumulation of fluid within the skull, either in the subdural space (external hydrocephalus) or within the ventricles (internal hydrocephalus). The process may be acute or chronic, may be the result of other morbid processes that are conspicuous (secondary form), or may occur in consequence of a process that is difficult to trace except by this effect (so-called primary form).

ACUTE HYDROCEPHALUS.

The only known cause of acute hydrocephalus is meningitis. This, in all forms, may be attended by effusion of fluid in the subdural space or into the ventricles. The latter, for instance, occurs in four-fifths of the cases of tubercular meningitis. Hence the term "acute hydrocephalus" was for a long time the common designation for meningitis. The external effusion is the direct result of the inflammation of the pia mater, and the internal effusion is probably also the result of inflammation of the choroid plexuses and velum interpositum, perhaps also of the lining membrane of the ventricle.

In some cases, however, the effusion into the ventricles is the only pathological change in an affection of acute and febrile cause. The external meninges are healthy; the choroid plexuses may be the seat of distinct inflammatory changes, and the lining membrane of the ventricles may be finely granular; the adjacent brain tissue is softened, and the brain substance and convolutions are compressed. There is no indication of any processes of which the effusion could be a mechanical consequence, such as is an occasional cause of chronic hydrocephalus. The symptoms during life are those of an acute inflammatory disease, and resemble very closely those of tubercular meningitis. Hence it is commonly assumed that they are due to a ventricular meningitis, affecting chiefly the choroid plexuses, and causing an abundant effusion of serum. Although the pathology of the cases is obscure, no better explanation has yet been given.

CHRONIC HYDROCEPHALUS.

External Chronic Hydrocephalus.—Whenever there is wasting of the brain, there is increase of the subarachnoid fluid to occupy the space left. This is a common senile condition. Of the same character is the accumulation of fluid met with in some cases of arrested development, in which the brain is small and does not fill the cranial cavity.

In other cases, without the brain being smaller than normal, there is a congenital excess in the amount of fluid in the subdural space, and this expands the skull. The expansion may be so great as to prevent the child being born alive. If birth is survived, the head rapidly increases in size, and is of the same shape as in the internal hydrocephalus, to be immediately described. Post-mortem, the excess of fluid is the only morbid condition. There is no change in the membranes, and the actual pathology of these cases is obscure. The condition is usually attended by the same symptoms, and runs the same course, as internal effusion. Caries of the bone of the skull in such cases has occasionally permitted the escape of the effusion.

External hydrocephalus is sometimes "sacculated," *i. e.*, limited to a certain region by adhesions between the dura and pia mater. This condition is, in effect, a meningeal cyst. Its position may be over part of one cerebral hemisphere or beneath the tentorium. If there is enlargement of the skull, this is correspondingly unsymmetrical. The brain may be compressed opposite the seat of the effusion, and corresponding local symptoms may result. These cases doubtless arise from inflammation, and their course may be varied by intercurrent attacks of a meningitic character.

Chronic Internal Hydrocephalus may be either congenital or acquired.

Congenital Internal Hydrocephalus develops *in utero*, and may cause such enlargement of the head as to prevent birth until the fluid is let out, or it may be moderate at the time of birth, and afterward rapidly increase. The causes and pathological mechanism are practically unknown. It has been ascribed to maternal grief or other emotion, but on no reasonable grounds, and also to injury of the fœtus by falls, etc.—an influence which is at least intelligible. A connection has been supposed to exist between uterine disease and hydrocephalus, and has been explained hypothetically, but the causal significance of the association needs further proof. One etiological fact is, however, certain—that a tendency to the occurrence of fœtal hydrocephalus sometimes runs in families, and that more than one child may be affected in succession.

Sometimes all the ventricles are distended; more often the fourth ventricle suffers little, even though there is no obstruction in the aqueduct of Sylvius. In such a case the aqueduct may be funnel-shaped, dilated toward the enlarged third ventricle. If the distention is confined to one or both lateral ventricles, there is some obstruction at the foramen of Monro (see further, under Acquired Hydrocephalus). The fluid is usually clear, of low specific gravity, 1001–1009, and contains a small and variable amount of albumen, some chloride of sodium, and sometimes urea, cholesterin, and other substances in small quantity. The amount of liquid varies according to the degree of the disease, and has been as much as twenty-seven pounds. In proportion to its quantity, the substance of the cerebral hemispheres is compressed, and thinned by stretching. The corpus callosum is displaced upward, and if the cranium is enlarged, the hemisphere extends further forward, backward, upward and outward than normal. The cerebral substance may be reduced to a layer only a few millimetres thick, constituting the thin wall of a vast cavity. In such a case all traces of the convolutions and sulci may have disappeared, and even the basal ganglia may be almost unrecognizable. More often indications of the sulci can be traced, and the compressed basal ganglia lie at the bottom of the sac. The cranium is enlarged in proportion to the amount of effusion, and the bones of the skull are thin. The falx is necessarily stretched, and its edge forms a larger curve than normal; the extension of its edge raises the anterior part of the tentorium. Hence the sub-tentorial space is increased in size, and is not completely filled by the cerebellum, the interval being occupied by liquid, and sometimes by loose connective tissue. Otherwise the membranes are normal. The choroid plexus may be thickened, as if from old inflammation. The lining membrane of the ventricles is often finely granular on the surface and is sometimes thickened.

The external enlargement of the skull is very conspicuous and characteristic, and rapidly increases after birth. The fontanelles become very large, and bulge; at the sutures the bones are widely separated. The cranium has a rounded shape, and becomes disproportionately large in comparison with the face. The disparity is increased by the projection of the frontal portion of the skull. The orbital plates have an oblique direction, and the eyes are directed downward and partially covered by the lower eyelids. If the child lives, the head may attain an enormous size; in one case, at sixteen months old, the circumference was 107.6 cm. (Klein). The symptoms vary much. There is usually considerable mental defect, often amounting to idiocy. The limbs are weak; convulsions and various contractures are frequent; occasionally febrile attacks occur, attended by vomiting. The head is supported with difficulty, both on account of its weight and of the muscular weakness. The eyeballs are often rolled from side to side, and their axes may not correspond. The skin of the head is thin and the hair scanty. In extreme cases blindness occurs, and the ophthalmoscope shows optic-nerve atrophy, produced by the stretching of the nerve or by the compression of the chiasma. In congenital cases, the rapid increase of the disease usually causes death in the second or third month of life, by marasmus, convulsions, or coma. Occasionally the morbid process becomes arrested, and the patient may live on to adult life, and even to old age. It is said that the fluid is sometimes gradually absorbed. Ossification of the bones may progress, and is usually completed by the development of "Wormian bones" within the sutures. If the disease is considerable in degree, the mental and motor symptoms usually continue in some degree through life—mental weakness, often with irritability of temper, epileptic fits, muscular weakness, and contractures.

Acquired Chronic Internal Hydrocephalus may be (1) secondary to a lesion that produces the effusion mechanically; (2) consecutive to an attack of acute meningitis; (3) of apparently primary origin.

Secondary Mechanical Form.—As Whytt pointed out in the last century, any obstruction of the veins of Galen, hindering the return of blood from the intra-ventricular vessels, causes effusion into the ventricles, which may reach an extreme degree. The most common cause is an adjacent tumor. The fluid normally passes out of the ventricles by the openings in the membrane closing the fourth ventricle, the foramen of Magendie in the middle line, and the openings, one on each side, behind the roots of the glosso-pharyngeal nerve, first described by Mierzejewski. These may be occluded by meningitis, and then all the ventricles become distended (as Hilton first pointed out in his classical lectures on "Rest and Pain.") Or the passage from the third to the fourth ventricles may be closed, as a result of inflammation or of adjacent pressure, and then only the ventricles above are distended. Or the foramen of Monro may become closed, and the effusion be confined to the lateral ventricles. These accidents may occur at any age. Chronic internal hydrocephalus is sometimes the sequel to an acute attack of meningitis, but progressive effusion is probably in most cases the result of one of the two mechanical processes just mentioned, chiefly of the closure of the openings into the fourth ventricle by the inflammation.

Primary Hydrocephalus.—Internal effusion sometimes occurs without any of the above causes being traceable. In children with yielding skulls, it is supposed the mechanical congestion of frequent cough, or the tendency to transudation present in anæmia, may lead to some effusion, but it is not probable that the amount from these causes is ever considerable. At any age the condition has been known to develop and attain a considerable degree, without, it is said, any other pathological condition being discoverable than the slight traces of ventricular inflammation that are met with in the congenital cases. Dean Swift died from this disease at seventy-eight, after it had existed for three years. These cases are extremely rare, and their pathology needs re-investigation.

The symptoms in the acquired form are, on the whole, similar to those in the congenital variety, due allowance being made for differences of age. There are mental weakness, somnolence, coma, muscular weakness, convulsion, contractures, and loss of sight from pressure of the distended third ventricle on the optic chiasma. Temporal hemianopia may therefore precede the complete blindness. In young children with incomplete closure of the sutures, the head readily enlarges, although rarely to the same extent as in the congenital form. In the adult, enlargement of the head is less uncommon, although it has been known to occur, and the sutures may, as I have seen, become separated. This does not involve such great mechanical force as might be supposed, because the separation of the sutures is probably always preceded by another change, the gradual thinning of the cranial bones described in the section on tumors of the brain (p. 883). When the bones are reduced to the thickness of parchment, it is evident that a comparatively slight force may separate the sutures. Still, the fact that life may continue during this process is a remarkable example of the tolerance by the brain of slowly induced pressure. It might be supposed that the intraocular circulation would show the effects of the increased pressure within the skull, but this is seldom the case, on account of the anastomoses of the ophthalmic vein. Even during the process of separation of the sutures, I have been unable to observe any marked increase in the size of the retinal veins. The course of the acquired form varies much. Death usually occurs at the end of a few months or years. The progress sometimes ceases, and if the affection is moderate in degree, recovery may occur. It is only in children, in whom the ready enlargement of the head enables a certain diagnosis to be made when the disease is still in an early stage, that recovery can be proved. The sac has been known, in very rare instances, to rupture into the subdural space.

Diagnosis.—As just intimated, hydrocephalus can only be diagnosed with certainty when there is distinct progressive enlargement of the head. When the bones are united, internal effusion may be suspected if its indications slowly follow an attack of meningitic character, or accompany the symptoms of a tumor of the cerebellum. But primary hydrocephalus causes only symptoms that are not distinctive, and are much more frequently produced by other morbid processes. Hence, as a matter of fact, the existence of the disease, when it leads to no enlargement of the head, and when the causal indications just mentioned are absent, cannot be recognized.

In slight degree the enlargement of the head may be confounded with that produced by two other causes, rickets and thickening of the bone. In the former, the head has a somewhat square form, and not the globular shape characteristic of hydrocephalus. Although the fontanelle may be large, it is not bulged. The other signs of rickets are present in high degree. Thickening of the cranial bones may simulate hydrocephalus at almost any age. I have seen it in a boy of ten, the subject of inherited syphilis, and in a man of fifty. In the latter, a slow progressive enlargement of the head during several years led to a diagnosis of hydrocephalus, but at the post-mortem examination the cranial bones were found to be three-quarters of an inch in thickness, and the cavity was of normal size. It is doubtful whether the nature of these rare cases can be ascertained during life. A distinction of internal from external hydrocephalus can only be made by paracentesis, and not always with certainty, even by this means, since a thin-walled sac may be readily opened by a comparatively superficial puncture.

The Prognosis of hydrocephalus of any form is usually grave and always uncertain, unless the occurrence of arrest can be distinctly recognized.

Treatment.—Whatever lessens the volume of the blood, diminishes for a time the amount of the effusion. Thus an attack of diarrhœa lessens the prominence of the fontanelle. But purgatives are inadmissible : to be effective they must be more vigorous than a hydrocephalic child can bear. Diuresis constitutes a safer, but unfortunately less effective, mode of attaining the same end. In no case in which these measures have been used has a permanent effect been produced. Agents that are supposed to promote absorption, as iodide of potassium and mercury, have been extensively tried, but are, as a rule, powerless, and are sometimes harmful. The most direct treatment, which is unfortunately the most dangerous, is evacuation by puncture, a small quantity being let out each time, and compression of the skull by elastic bandages kept up during and after the operation. This procedure is, of course, most suitable to external effusion, but it has been employed in ventricular effusion, occasionally without ill effects, but with absolute success only in rare instances. The best place for puncture is at the outer angle of the anterior fontanelle. Not more than an ounce should be removed at a time, or collapse and convulsions are produced. The first puncture has generally been well borne, but in many cases the second or third has been fatal ; in some instances apparently because too little time has been allowed to elapse between the operations. Good has been observed to follow simple compression, a mode of treatment first advocated and energetically employed fifty years ago by Barnard, of Bath. Trousseau employed strips of diachylon plaster, a third of an inch broad, in the following manner :* (1) From each mastoid process to the outer part of the orbit on the opposite side ; (2) From the hair at the back of the neck, along the sagittal suture, to the root of the nose ; (3) Over the whole head in such a manner that the different strips shall cross each other at the vertex ; (4) A long strip around the head three times, taken first above the ears and eyebrows and a little below the occipital protuberance, so that the ends of all the other strips shall project below the circular strip ; these ends are next to be doubled up on the circular strip, and its remaining two turns passed over them in the same direction as the first turn. It is necessary to watch the effect, and loosen the strips if there are any symptoms of compression. Dr. West advises a broad elastic band as safer and more manageable than the plasters. His experience of pressure in severe cases has not been encouraging, since it has not hindered the accumulation of fluid, and has increased the symptoms of compression of the brain.

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* *Journal de Médecine*, April, 1843, quoted by West, "Dis. of Infancy and Childhood," Seventh Edition, 1884, p. 136.

PART V.

GENERAL AND FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

The diseases that remain for consideration are those in which there are no constant changes to be seen with the naked eye. It was formerly the custom to include them all under the term "functional diseases," but microscopical changes have been discovered in some of them with sufficient frequency to make it certain that there is far more than a mere disturbance of function, and it cannot be doubted that most of these maladies depend upon alterations in the nutrition of the nerve elements, although these may not yet have been found, and perhaps cannot be detected without more means of investigation than we at present possess. The diseases themselves are so different in character and so various in seat, that their classification into groups is alike difficult and useless. Anything like a scientific classification, based upon our present knowledge, would result in little more than enumeration, and is therefore not attempted. The order in which they are described is based only on convenience.

CHOREA.

Chorea is a disease that occurs chiefly in young persons, is usually of limited duration, and is characterized by irregular spasmodic movements, by incoördination of voluntary movement, and often by muscular and mental weakness. The proportion of these elements varies in different cases.

The term "Chorea Sancti Viti" (*χορεια* = dancing), or St. Vitus's dance, was first applied at Strasburg to the epidemic dancing mania prevalent in the fourteenth and fifteenth centuries, when the sufferers were taken, by order of the Strasburg magistrate, to the chapel of St. Vitus, to be cured by the influence of the saint. The term became restricted, chiefly through the influence of Sydenham, to the disease now known by the name, but it is even now used more widely in Germany, and the affection here described is distinguished, as *chorea minor*, from *chorea major*, a form of hysterical disorder distinct from ordinary chorea, and more closely allied to the epidemic malady of the Middle Ages.

Etiology.—The relation of chorea to *race* is a subject that has been but little studied. According to Weir Mitchell, in the United States the disease is less frequent among the negro than among the white population, and in some places in which there is a mixed population (*e. g.* in the island of Cuba), where chorea is not uncommon among the white children, it is unknown among the colored children. Urban residence is said by the same writer to favor the occurrence of the disease, and it is probable that the influence could be traced also in this country.

Hereditary influences can be found in many cases of chorea, and the double relation of the disease can often be traced in these influences; its relation to acute rheumatism on the one hand, and to other nervous diseases on the other. Rheumatism is, however, so common a disease that it is only when the family tendency to it is strong or close that weight can be placed on its occurrence.* Some neuropathic heredity is to be traced in one-sixth of the cases; epilepsy, insanity, or chorea itself. Although the total proportion is not great, the family tendency in some cases is very marked. For example, three sisters suffered from true chorea; a sister also suffered from chorea, and the mother from epilepsy; the mother of one patient was insane, and her sister's child also had chorea; the father of another had suffered from chorea in early life and so had two children of his brother; five relations of another had been insane. Many other similar facts have been met with in the investigation of the clinical history of epilepsy. For instance, a man was epileptic, his sister insane, and two of her children had chorea. Two sisters of an epileptic girl had had chorea. Two children suffered from chorea; their mother's sister being epileptic and insane. In other cases, again, there is a family history of both rheumatism and nervous diseases.†

Age.—Chorea is essentially a disease of the later period of childhood. Nine-tenths of the cases occur between 5 and 20, and four-fifths between 5 and 15. A larger number of first attacks (nearly half the whole) occur between 5 and 10 than between 10 and 15, but according to my own observations, if relapses are included, more cases occur between 10 and 15, and the thirteenth year of life is that which presents the largest number of attacks.* Under 5 the disease is extremely rare; a few cases have been recorded at 4, and I have seen one case at the age of 3½.† Not more than 5 per cent. of the total number of attacks occur over 20. The disease is, however, occasionally met with in later life, even up to extreme old age. Between 30 and 40 the disease is scarcely ever met with. Many cases of senile chorea probably depend on a morbid process, different in nature from that which causes the juvenile form of the disease, although similar in effect.

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* In the British Medical Association's Collection (see note on next page) there was a family history of rheumatism in 45 per cent. of the 439 cases.

† Many illustrations of these family tendencies are mentioned by Money in his analysis of 236 cases of chorea contained in the case books of University College Hospital and of the Hospital for Sick Children (*Brain, 1882*, vol. v, p. 513). In two cases the patient's father had suffered from rheumatic fever and chorea; in another both the father and the mother had had rheumatic fever, and a brother chorea; in another the mother had had rheumatic fever, and mother, father and brother had had chorea.

A remarkable form of hereditary chorea (affecting many generations) has been described by Huntingdon as existing in Long Island, New York (a favorite haunt of tetanus), but it is evidently a different disease from ordinary chorea, since it begins between thirty and forty, is accompanied by great mental failure, and is invariably fatal. A family in which six cases of a somewhat similar affection can be traced has been recorded by Mr. West, of Stoke-on-Trent. All the sufferers became affected in adult life, and the chorea was accompanied by some mental change (*Brit. Med. Journal*, Feb. 26th, 1887, p. 435).

Sex.—Chorea affects girls nearly, but not quite three times as frequently as boys. A combination of recorded statistics yields 365 boys to 1000 girls.‡ The preponderance of girls is least in childhood and increases after puberty. The disease is rare in lads over 16. Between 20 and 30 it is practically confined to females. During the second half of life, however, the rare cases of chorea occur in both sexes.

Climate has little influence. Chorea occurs in warm as well as in temperate countries. The question of its relation to *season* has recently attracted attention, especially in America. At Philadelphia, Morris Lewis found an apparently distinct relation to time of year, most cases occurring in the spring months;§ in Boston, however, Putnam failed to trace this influence, and in this country the influence of season does not seem to be great, the numbers (of 100 attacks) commencing in each quarter being 1st thirty-three, 2d twenty-five, 3d twenty, 4th twenty-seven. The minimum was in July and August. A more distinct relation to season is, however, occasionally to be traced in the recurrences of the disease (see p. 970). Dr. Morris Lewis|| has lately instituted an elaborate inquiry with the object of ascertaining whether there is any relation between temperature, humidity, or barometric variations and the occurrence of chorea (compared over a period of ten years), but he could find none. He found, however, that there is a slight correspondence with the average number of cloudy days per month and also with the actual number of rainy days, and a still closer connection with the num-

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* The 439 cases tabulated by Dr. S. Mackenzie for the Brit. Med. Assoc. Collective Investigation Committee (*Brit. Med. Journal*, February 26th, 1887) give 34 per cent. (one-third) between 5 and 10, 43 per cent. between 10 and 15, and 16 per cent. between 15 and 20; but unfortunately it is not stated how many were first attacks. In this list are cases at 40, 63, 68, 73, 78 and 86. I may add that the statistical conclusions of the "Report" agree very closely with those given in this chapter, most of which was written some years ago.

† The patient was a girl in the Children's Hospital under the care of Dr. Sturges. The chorea, although slight, was quite characteristic, and the heart presented a mitral regurgitant murmur and recent pericardial friction.

‡ The statements in the text are based on the cases collected by Hughes, Sée, Pye-Smith, Rufz, Steiner, Wilkinson, Sturges, and on 100 original cases. Correct facts regarding the influence of age can only be ascertained by combining statistics, since they are not fairly represented either by the figures obtained from children's hospitals or by those from general hospitals in towns in which children's hospitals exist.

§ The percentage varied from 4.1 per cent. in October, and 4.3 per cent, in November, to 8.2 per cent. in January, 15.3 in March, 8.6 in April, 10.7 in May, and 10.5 in July, and then fell gradually to its lowest point in October.

|| *The Polyclinic*, January, 1887, p. 205.

ber of storm centres passing over Philadelphia; and that the correspondence became closer the wider the range of country included in the meteorological observations, till an area of a radius of 400 miles was reached, and then the correspondence between the storm curve and the chorea curve became still more close. This curious fact has not yet been confirmed by observations elsewhere.*

The only immediate cause of chorea that can be traced with any frequency is emotion, usually fright, rarely mental distress. The proportion of cases in which mental emotion can be traced has varied in different collections, between a fifth and a fourth. I have found the frequency to be nearly the same in each sex, but it is relatively more common in boys under twelve, and is rare in boys over fourteen, whereas it is a not uncommon excitant of chorea in girls up to twenty. The interval between the fright and the first symptoms of the chorea rarely exceeds a week;† it is about as frequent for it to be one week or to be less than a week (three or five days). Very rarely the interval is only one day, and still more rarely there is no interval—the chorea immediately succeeds the fright. This was the case in a lad in whom the movements commenced immediately after a pistol had been unexpectedly discharged close to his ear. Another boy was discovered in an apple tree, and fell in his hasty descent; he immediately began to shake; the tremor of alarm assumed a choreic character, and persisted as chorea.

Occasionally the fright which was the cause of the chorea in girls occurred at the menstrual period, but such instances are too rare for any special significance to be attached to the coincidence. Nor is any disorder of menstruation to be recognized with sufficient frequency to justify the assumption of a causal influence. Imitation is commonly given as a cause of chorea, and the hysterical variety often arises thus, but I have not known ordinary chorea to be distinctly so produced. It should be noted, however, that the connection between some cases of apparently hysterical chorea and the common form is exceedingly close.

Chorea sometimes results from traumatic influences, but it is probable that their efficiency depends on coincident emotion. Thus chorea has resulted from a fall or a blow on the head and from the extraction of a tooth, but it has also followed an attempt at tooth extraction which was unsuccessful.

Intestinal worms are occasionally present in choreic patients, and have been supposed to cause the disease. In extremely rare cases chorea has been ascribed to the irritation of a peripheral spinal nerve. The influence of these reflex causes is doubtful.

Chorea is so rarely met with in association with acute specific diseases, that any such association is probably accidental; or, at most, the general disease only serves to excite the chorea in an indirect way. A girl, sixteen years old, had a well-marked attack of chorea during the course of typhoid, but she had suffered from acute articular rheumatism a year before.*

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* Comparing the average number of attacks per month of chorea and rheumatism, the curious fact was ascertained that the variations in the occurrence of rheumatism corresponded with those of chorea, but were uniformly a month later.

† In the cases in which many weeks elapse between the fright and the first symptoms, a causal relation is extremely doubtful.

Three important etiological influences remain for discussion, two of which have given rise to much controversy—acute rheumatism, organic heart disease, and pregnancy.

Acute Rheumatism.—The fact that a considerable number of patients with chorea have had acute rheumatism has been recognized by all observers since it was first pointed out by Hughes, although the extent and significance of the association have been variously estimated. The largest recorded proportion of cases with preceding rheumatism is one-fourth.† In my own cases the proportion was about one-fourth (24 of 100 cases). The proportion varies, however, at different ages. Among my cases, in only one under nine was there a history of rheumatism, while of fifty-three cases which occurred between ten and fifteen years, sixteen, considerably more than one-fourth, had suffered from rheumatic fever. This fact shows clearly that the influence of rheumatism will be under-represented in statistics collected at hospitals for children. The proportion, in later childhood at least, is too large to be ascribed to accidental coincidence, and the close relationship between the two diseases is shown, in an emphatic manner, by the facts that not very rarely chorea immediately follows acute rheumatism, that the latter occasionally comes on in the course of chorea, and also by the mysterious but unquestionable relation next to be mentioned between chorea and disease of the cardiac valves.

Heart disease may be developed during the course of chorea, or may precede it. The latter only concerns us now. Published statistics afford no information on this point, for no attempt has been made to discriminate between the two forms.‡ When the cardiac disease is considerable in amount, and the chorea of short duration, and especially if the walls of the heart present evidence of consecutive changes, we are justified in assuming that the heart disease preceded the chorea, and this assumption is rendered the more probable if there has been a preceding attack of rheumatic fever. Of forty cases in which I found evidence of organic heart disease there was strong reason to believe that this preceded the chorea in eighteen. From the nature of the investigation this proportion is probably below the actual facts (because only conclusive evidence was admitted), and it is therefore within the truth to say that in one-half of the cases in which organic disease of the heart exists, this preceded the chorea.

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* Peiper, *Deut. med. Wochenschrift, 1885*, No. 8.

† Hughes, Sée, and the "B. M. A. Coll. Investigation" (loc. cit., p. 428). In the latter the percentage was 26, and about the same in each sex; but when to these cases are added those in which acute rheumatism occurred during or after the chorea, the percentage rises to 32, or one-third, and another 14 per cent. had suffered from vague rheumatic pains.

‡ To this statement, however, the "B. M. A. Collect. Inv. Report" constitutes an exception.

Regarding the combinations of the three conditions, rheumatism, organic heart disease, and fright, I have found that cardiac disease was present (either before or during the chorea) in three-quarters of the cases in which there was a history of rheumatic fever, while in those without such history there was heart disease in only a third. Fright was the apparent cause of the chorea in a rather larger proportion of the patients who had had rheumatic fever, or preceding heart disease, than of those who had not.

Pregnancy.—Chorea may occur as a complication and consequence of pregnancy. Some of the patients who thus suffer have had chorea or rheumatic fever in earlier life, or an attack of rheumatic fever immediately precedes the chorea. The occasional relation to rheumatic fever is well shown by the case of one patient who had no chorea during the first pregnancy, but in the interval between the first and the second she had an attack of rheumatic fever, and during the second she suffered from chorea (Mosler). But in the majority there is no predisposing influence, except the pregnancy, to which the disease can be ascribed. Fright, or some other distressing emotion, is the apparent exciting cause of the disease in about the same proportion in gestational chorea as in the chorea of earlier life. If the disease has occurred during one pregnancy it may or may not occur during one or more succeeding pregnancies. It scarcely ever occurs for the first time over twenty-five years of age. The number of (28) cases occurring at different ages is as follows: 17, three; 18, three; 19, three; 20, eight; 21, two; 22, two; 23, six; 24, one.*

In the recorded cases of distinct chorea occurring during pregnancy after twenty-four years of age, the patients have suffered from chorea during a previous pregnancy. The disease is most common during the first pregnancy, and is very rare after the second, except as a recurrence. Of thirty-eight recorded attacks, twenty-five were during the first, and ten were during the second pregnancy, the first having been free. In two attacks in the third, and one during the fourth pregnancy, the patients had previously suffered from chorea during a similar condition. In the only recorded case† in which a patient is said to have suffered for the first time during the fourth pregnancy, it is doubtful whether the disease was really chorea, since the movements were confined to the legs.

The chorea may commence at any period of pregnancy, from the beginning until near the close, but it begins more frequently in the third than in any other month, and very rarely in the ninth month. Of thirty-six attacks‡ the numbers commencing in each month were: 1st, four; 2d, three; 3d, nine; 4th, five; 5th, four; 6th, four; 7th, three; 8th, three; 9th, one. Thus two-thirds of the attacks commence before the end of the fifth month, and one-quarter of the total number commence in the third month. If chorea recurs during several pregnancies, there is no uniformity in the date of its commencement; for instance, in one remarkable example of recurrence, the patient (who had had rheumatic fever, followed by chorea, at sixteen) became choreic in the fourth month of her first pregnancy (at twenty-two), in the third month of the second, at the commencement of the third, and in the fourth month of the fourth pregnancy (Lawson Tait). The cases apparently excited by fright show no difference in the date of their commencement, but a rather larger proportion do not exceed twenty years of age than the cases in which no exciting cause can be traced. In one instance, albuminuria existed, left (with a cardiac murmur) by an attack of scarlet fever in the fourth month of pregnancy; the chorea commenced in the eighth month (Woodman).

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* The figures are based on an analysis of recorded and original cases; most of the former have been collected by Barnes (*Obstetrical Trans., 1869*), but some of his cases have not been included, either because insufficiently reported, or because their nature is open to question.

† Levick, *Amer. Journal of Med. Sciences, 1862.*

‡ In the statement in the text those cases which have been recorded as commencing when the pregnancy had existed for a certain number of months are considered as commencing in the subsequent "month" of gestation.

In extremely rare cases, chorea (of ordinary form) has been observed to commence after delivery or after abortion, and the subjects of this post-puerperal chorea have, for the most part, been beyond the age to which the chorea of gestation is chiefly limited.

SYMPTOMS.—The characteristic symptoms of chorea, the spontaneous movement, incoördination, and muscular weakness, are all present in severe cases, and are often associated with some mental failure. In slight cases the symptoms may be combined in various degrees.

The first thing to attract attention is sometimes the spontaneous twitching, sometimes the interference with voluntary action. The spontaneous movements are first noticed in the hands or face, rarely in the legs, and so closely resemble those involuntary actions which "nervous" children present under emotion that they are often regarded as such. The interference with movement is rarely at first a conspicuous incoördination; a sudden purposed movement is more considerable than was intended, or a persistent action is suddenly disturbed by an unwilled movement. The insubordination of the motor centres is seen also in the occasional unintended relaxation of muscles or in a delay of intended relaxation. Thus, objects which are being carried are suddenly dropped, and in one lad the first indication of the commencing disease was that in throwing a cricket ball the fingers relaxed their grasp too late, and the ball simply fell to the ground. The spontaneous movements may at first be recognized only on close observation, but as the disease advances they become conspicuous enough. They are quick and irregular, sometimes complex in character, and each movement is brief in duration. The mouth is drawn to one side with an associated movement of the lips, the eyes are closed for a moment. The fingers are irregularly extended, the hand is pronated or supinated, or the whole arm is suddenly extended or rotated, and these movements are often combined. In the legs the movements are usually more simple, momentary, muscular contractions; they may cause merely a slight jerk of the body, but, if considerable, walking and even standing may be seriously interfered with.

The spasmodic movements are always irregular in time as well as in character and degree. At first they are occasional only, but their frequency increases with their severity, until at last they may be so continuous and violent that the limbs are in constant movement. The spasm in the muscles of the neck may cause frequent movement of the head to one side, and the eyes may move with the head. In many cases the spasm of the eyeball muscles is not quite equal and momentary diplopia results. The muscles of the trunk may also be involved. When their affection is slight, the patient from time to time sways from side to side while sitting or standing; when considerable, standing or sitting is impossible, and the patient may even be thrown out of bed by some violent contortion. The limbs may be thrown about with such force that serious bruises result from their contact with adjacent objects, and in one recorded case, the choreic spasm of the muscles of the jaw was so violent that several of the teeth were broken (Tuckwell). The movements are always increased by excitement, and by attempts at voluntary movement, and are lessened by repose, physical and mental. They almost invariably cease during sleep, natural or induced, but their severity may prevent sleep for days together.

Voluntary movements are executed rapidly and in a spasmodic manner. This character seems to be in part impressed upon them by the tendency to spasm, in part to be the result of an attempt to escape the disturbing influence by rapidity of movement. Thus the tongue is protruded suddenly and jerked back again, or the hand makes a dash at any object which it attempts to grasp. Usually, and always in severe cases, the resulting movement is irregular, partly from disturbing spasm, partly from an incoördination which is not always related in degree to the amount of spontaneous movement. In some cases the latter may be slight while incoördination is great, and, conversely, voluntary movement may be almost steady although there is much spontaneous spasm. Sustained muscular action is often impossible; an object may be grasped with firmness, but first one finger and then another relaxes in spite of the will.

The muscles of respiration, especially the diaphragm, frequently share the irregularity observed in the action of the more strictly voluntary muscles; the respirations are unequal in depth—a sigh alternates with shallow breaths. Irregularity of the heart's action may sometimes be observed, but is almost always produced by, and distinctly consequent on, the irregularity of the respiratory movements.

Besides the interference with movement occasioned by spasm and incoördination, there is usually distinct deficiency of muscular power. The degree of loss of power may not be proportioned to the spasm; it may be considerable when the latter is barely recognizable. This is frequently noticeable at the onset, but sometimes there is much weakness and scarcely any spasm, a form which I have proposed to call "paralytic chorea."* There is never, however, anything like complete loss of power, and often the loss of use of a limb is out of proportion to the actual muscular weakness.

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* *Brit. Med. Journal*, April 23d, 1881.

These motor symptoms may be from the first general, and throughout may involve both sides equally. The arms are almost always affected earlier and in greater degree than the legs, and the movements in the arms are wider in range and more irregular in character. The difference that exists between the physiological movements of the upper and the lower limbs is thus reproduced in the spasm. In at least half the cases, however, the affection of the two sides is not equal. It may be throughout limited to one side (hemichorea), or it may affect one side first, and afterward the other. In the latter cases it may continue on the side first affected when the other is involved, unilateral chorea thus becoming general, or it may cease on the first when it passes to the other. The cases in which it is limited to one side present considerable variety in the area affected; the movements may involve the arm only, the leg escaping; they may affect the arm and leg on the same side, or one arm and both legs, the other arm remaining free and the leg on the side of the unaffected arm being usually less involved than the other. These variations in the distribution of partial chorea correspond closely to the distribution of partial convulsive seizures. There seems to be no difference in the frequency with which the right or the left side is the seat of hemichorea, or is affected most or first in general chorea. No relation can be traced between the presence of heart disease, or the influence of fright, in causing the disease, and the side affected. Of sixty-four cases I find the total numbers nearly equal for each side:—

	Right.	Left.	
One side only	11	13	
One side first	10	10	
One side most	10	10	
	—	—	
	31	33	= 64

The *electric irritability* of the muscles and nerves can only be thoroughly studied in cases of hemichorea in which the unaffected limbs are available for comparison. Sometimes no alteration of irritability can be discovered. But in other cases a distinct increase of irritability may be found in nerve and muscle to both faradaism and voltaism (Benedikt, Rosenthal, myself). Muscular contraction can be obtained with a weaker current on the affected than on the unaffected side, the difference amounting to one or two centimetres of the secondary coil of a faradaic apparatus, or two to four cells of a voltaic battery. I have observed this increase of irritability to be absent at the commencement, to come on during the course of the disease, and to subside with recovery. A "qualitative" change in the mode of response to voltaism has also been described, instead of the contraction on closure of the circuit, occurring at the negative pole with a weaker current than at the positive; the latter may occur as readily as the former (*i. e.* instead of 1 K.C.C., 2 A.C.C., we have K.C.C. = A.C.C., see p. 67). I have not myself been able to observe this.

Speech is often impaired in chorea. The movements of the muscles of articulation are disturbed, just as those of other voluntary muscles. Irregularity of movement of the vocal cords has been observed with the laryngoscope, while the disturbance of respiration further deranges utterance (v. Ziemssen). Words are uttered quickly, just as voluntary movements are performed quickly; speech is interrupted by sudden, deep inspirations, which often cut off the last syllables of a word. Sometimes the words are jerked out in separated parts. Actual stammering is rare, but I have met with one case in which it preceded the other symptoms. In severe cases the interference with articulation may be very great, the spasm in the muscles of the mouth and tongue on any attempt at articulation may render it impossible, for weeks, for the patient to utter a single word. There is often a disinclination to speak (due to the conscious difficulty, and partly perhaps to the mental state presently to be described) in addition to the actual interference with articulation.

Sensibility is, as a rule, unaffected. In extremely rare cases, unilateral diminution or increase of sensibility, sometimes involving the special senses, has been observed. Such disturbance is probably allied to that met with in hysteria, and is certainly no part of the ordinary symptoms of chorea. Tender points along the spine or along the course of the nerves (especially where these emerge from deeper structures) have rarely been observed (Cartier).

Chorea is, as a rule, a painless disease. The muscular spasm may cause fatigue, but occasions no sensation of pain. I have met with only two cases in which there was pain in the limbs at the onset. One patient, a girl, complained of pain in the left hand, which gradually, in the course of a few hours, passed up the arm to the side of the head, and three days after was felt in the leg. The choreic movements commenced in each limb at the same time as the pain. The disease afterward became general without any pain in the other side. There was no affection of the heart. In the other case, also a girl, intermittent neuralgic pains in face, arm and leg occurred some weeks before the onset of right-sided chorea. The latter was accompanied by some hysterical hemianæsthesia.

The pupils are often large, but it is doubtful whether this character is connected with the disease. Once, I observed inequality of the pupils, the larger being on the side opposite to the limbs most affected.

The mental state, in slight cases of chorea, may be normal, but in severe cases there is usually some irritability and often distinct mental dullness. The degree of change does not bear any relation to the severity of the other symptoms. The mental dullness is usually conspicuous in the patient's aspect and may amount to practical dementia, so that stools and urine may be passed into the bed, not from paralysis of the sphincters, but from mental apathy. In other cases the irritability may pass into excitement, the mind seems to share the disturbance so conspicuous in the muscles, and there is excited delirium, which, in its restlessness and delusions, may resemble mania, and in some cases must be regarded as such. In some of these cases the physical symptoms are trifling compared with the mental disturbance.

Symptoms Outside the Nervous System.—The temperature in slight cases is normal throughout, but in severe forms it may be raised one or two degrees. Even in these it rarely reaches, and still more rarely exceeds, 102° F. I have once observed occasional elevation of temperature, from time to time, during a prolonged attack, without any complications or endocarditis to which it could be referred.* Hyperpyrexia is probably always a rheumatic complication. The patients are often anæmic, and if not so at the onset, they become pale and lose weight rapidly during the course of the disease, especially if this is so severe as to interfere with sleep.

Heart.—The cardiac symptoms are of great importance. The pulse, as already stated, may be irregular in consequence of the irregularity of the breathing. It is usually increased in frequency. I have several times noted that the effect of posture on the pulse is less than in health; the pulse rate may be nearly or quite the same in the upright and the recumbent postures. In some cases the sounds and impulse of the heart are normal. More frequently there is some abnormality: (1) There may be murmurs, due to the anæmia, in the pulmonary artery and aorta, often associated with a venous hum in the jugular vein. Sometimes the systolic murmur at the base is continuous with one heard over both ventricles, having its maximum at the inner end of the fourth intercostal space, heard up to, but not beyond the left apex, and probably produced in the ventricles, in consequence of the blood state. With this there may be such a change in the character of the impulse as indicates slight dilatation of the heart, consequent on the anæmia. (2) There may be a faint systolic murmur at the apex of the heart, heard with some beats and not with others, which is commonly said to be due to irregular contraction of the papillary muscles, which sometimes fail to compensate for the shortening of the ventricle, and thus permit occasional regurgitation—a mechanism that can neither be proved nor disproved. (3) In a considerable number of cases there is distinct evidence of an organic valvular lesion. The frequency of organic disease is less in childhood than in youth,† and hence has been variously estimated by different observers, according to the class of cases which have come under their notice.

In some cases with organic disease there is good reason to believe that the heart disease preceded the chorea (see *ante*, p. 959); in other cases the true relation between the two is doubtful; in other cases, again, the organic murmur may be observed to develop during the course of the disease, an indication of endocarditis which, as will be seen, pathology amply confirms. Hence it is of great importance that the heart should be examined repeatedly during the course of the disease, and also after its termination. Preceding valvular disease is most frequently mitral regurgitation; occasionally, there is mitral constriction alone or combined with regurgitation. Aortic dise is much less common ; I have only met with two instances of aortic regur tation among about 250 cases of chorea that have come under my obser tion. In one case there was aortic obstructive murmur, musical in to combined with mitral regurgitation.* In the cases in which the relation the disease to the chorea is doubtful, or in which the heart is affected duri the chorea, the lesion is almost always mitral regurgitation. The frequen of a hæmic murmur at the aortic orifice renders it almost impossible recognize the development of an organic obstructive murmur during chor but its occurrence was probable in a case in which I found, at a second atta of chorea, unquestionable evidence of aortic obstruction which was abse in the first attack, the patient having had in the interval no rheumatic fev Post-mortem, endocarditis has been found at the aortic as well as at t mitral orifice.

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* In the "Collect. Invest." cases, pyrexia was noted in only twelve per cent., but it is probable that this only represents the frequency of such considerable and prolonged pyrexia as would attract attention.

† This statement is at variance with the opinion of others, by whom, for the most part, "heart symptoms" of hæmic, dynamic, and organic origin have been lumped together.

The *urine* in chorea contains an excess of urea and of phosphates (Walsh Handfield Jones), but it is in other respects normal. Albumen is prese only from independent kidney disease.

Complications.—Endocarditis is so common in chorea that it can scarce be regarded as a complication. It is usually of a benign form and caus no subjective symptoms, although it may lead to permanent valvular diseas Occasionally, its existence is emphasized by the occurrence of embolis I have seen hemiplegia from embolic softening of the brain occur during t course of chorea, and a similar consequence of endocarditis distinctly orig nating during an attack of chorea has been recorded by others. It is excee ingly rare for the endocarditis to assume the more malignant type of t "ulcerative" form. In the only instance of this dangerous complicati that has come under my notice, the patient was also suffering from Bright disease, but died from the septic embolic processes.

Acute articular rheumatism may not only immediately precede chorea b may come on during the course of the disease without any distinct excitir cause. It is usually attended with moderate elevation of temperature, trifling in severity, and brief in duration. Evidence of fresh endocardit may sometimes be detected during the course of the rheumatism. In son patients there may be found the small subcutaneous nodules, varying in si from that of a pin's head to that of a pea, met with also in those who a liable to rheumatism.† They may be felt not only beneath the skir especially of the arms, but also on the tendons, especially of the flexors of the fingers, and the peroneal muscles of the hands. They have been observed to come on with the chorea and pass away with it.*

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* In the "Brit. Med. Ass. Collect. Inv." there were 116 cases of pure mitral disease an only six of pure aortic disease. Unfortunately, the facts regarding heart disease and chor are given in so condensed a form in the Report that it is difficult to draw any general concl sion from them. Distinct organic disease, either preceding or secondary, was found in thirt two per cent. of the cases. But the difficulty of deciding whether there is or is not organ disease is often very great, and the large number of observers entails considerable uncertain as to the value of the statistics on this point.

† They were first observed by Meynet (*Lyons Médical, 1875*, No. 49). The most impor ant articles on the subject are by Barlow and Warner, *Trans. International Med. Congres 1881*, vol. iv, p. 116, and by Hirschsprung, *Jahrb. für Kinderheilk.*, March, 1881.

In most cases of chorea the ophthalmoscopic appearances are those of health. In a few there is optic neuritis, usually slight in degree, just enough to be unequivocal. It passes away when the chorea is over. In only one case have I seen considerable neuritis, comparable to that seen in a case of tumor; the inflammation passed entirely away with the chorea. It is probable that the neuritis is related to the cause of the chorea rather than directly to the morbid process in the brain. Many of the patients had considerable hypermetropia, and it is known that this condition disposes to slight neuritic changes in the discs, and may aid other influences in leading to the change.

Convulsive attacks of any form are more rare than might have been anticipated from the spasmodic nature of the affection. In one case, in which the choreic movements were most severe, occasional attacks occurred, in which there were apparent loss of consciousness and peculiar convulsions, partly choreic in form. In one, for instance, the head turned to the right, the right arm and leg presented violent choreic movements, while the left was stretched out, the fingers extended, the thumb inverted and the whole limb in a state of cataleptic rigidity, so that it remained in whatever position it was placed. The attack lasted two minutes. In another attack there was some arching of the back and tonic flexor spasm in the arms, followed by violent choreic movement in the right arm. Such attacks occurred daily for three weeks.† Epileptiform convulsions are very rare. In one case of chorea in a boy of twelve, greatest on the right side, four convulsive attacks, limited to this side, occurred during the fortnight after the cessation of the chorea, and similar attacks afterward persisted as chronic epilepsy. There was no cardiac murmur. I have met with a few other cases in which epilepsy dated from an attack of chorea.

Persistent spasm is also occasionally met with. In a young child there was continuous spasm in the arm during the attack, which was one of true chorea. The elbow- and wrist-joints were flexed, and the aspect of the limb was similar to that seen after infantile hemiplegia. The choreic movements were present in the arm, although in much less degree than in the other limbs. As the child recovered, the persistent spasm lessened and the movements in the limb became greater. Similar spasm had been present in a previous attack.

The cases in which the mental disturbance is very great have been termed *maniacal chorea*. The form occurs chiefly in females, at or soon after puberty, or during pregnancy. The mental disturbance may not come on until the disease is well developed, or it may precede the chorea. The latter may remain slight, and may even, as I have seen, quickly cease while the mental disturbance continues in intense form. There are often delusions, and sometimes wild, violent excitement, but rarely the intense garrulity so common in ordinary mania. The excitement usually subsides in the course of a week or two, and leaves dullness and apathy, with a depressed, listless look, disinclination to speak, and sometimes persistent hallucinations. Food has often to be administered by the rectum or by force, and stools and urine are commonly passed unnoticed. The condition usually slowly passes away, but occasionally persists for weeks or even for months after the chorea has ceased, and it may even be permanent. In one case, recorded by Golgi, there was no improvement, but slowly increasing mental failure, until death ten years later. A similar sequel has been observed to maniacal chorea occurring during pregnancy.

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† Sheele, *Deut. med. Wochenschrift, 1885*, No. 41, who observed that the development of the nodules was accompanied by transient contracture of the flexors.

* I have given fuller details of these attacks in "Epilepsy, etc.," 1881, p. 181.

The cases in which loss of use of a limb is the only conspicuous symptom, "*paralytic chorea,*" occur only in childhood and affect only the arms, one always more than the other. Slight twitchings may occur at the onset and cease, the weakness remaining. More commonly the loss of power is the first symptom, and it may persist or even lessen as the movements develop. This form is chiefly of importance in respect to diagnosis.

Duration.—Few diseases are so variable in duration as chorea. It usually lasts from six weeks to six months. It rarely falls short of the earlier limit, but I have known the disease in one or two instances to last only three weeks. On the other hand, the limit of six months is frequently exceeded. The average duration has been found by more than one observer to be ten weeks.[*] This is, however, true only of cases which are admitted into hospitals before they have lasted for a considerable time. At the National Hospital for the Paralyzed and Epileptic, to which choreic patients are often brought as a last resort, I have met with no less than fifteen cases in which the duration exceeded six months, and seven in which it exceeded a year. Occasionally a slight degree of chorea persists for a much longer time, and I have seen cases in which it has continued for two, three, four, and even six years, without any complete intermission. In most of these cases of very long duration, however, there are occasional remissions of the disease, so that the prolonged course is rather a series of relapses, with imperfect recovery in the intervals. In extremely rare instances the disease never ceases. A patient was attacked with chorea in youth, and the disease continued until death at the age of sixty-six.[†] A girl was attacked with chorea at the age of nine, and the movements continued and were still present at the age of fifty.[‡]

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[*] From the tables given in the "Coll. Invest. Report," I have calculated the following percentages of the duration of 396 cases: Not exceeding one month, 18 per cent.; between one and three months, 57 per cent.; over three months, 25 per cent.; only 6 per cent. exceeded three months. The percentage is nearly the same for each sex. (The calculation is made on the assumption that the cases in which the duration is (for instance) three months are given in the column headed "under three months," which is probable from the way the table runs although inconsistent with its wording).

[†] Meldner, *Wochenbl. der Gesellsch. der Wiener Aerzte, 1869*, No. 19.

[‡] Macdougal, *Lancet, 1885*. No. xvii.

As a rule, the more severe an attack the longer is its duration, but the influences which determine either severity or duration are very obscure. I have been unable, on a comparison of the details of cases, to discover any relation between duration and age, sex, state of heart, preceding rheumatic fever, or exciting cause.

Recurrence.—Chorea is a disease prone to recur. In how many cases it again attacks an individual can only be approximately determined, since the subsequent history of many patients is unknown. Of the series of original cases that I have analyzed, one-third suffered from more than one attack. Fifteen patients had more than two attacks, viz., nine, three attacks; one, four; three, five; one, six; and one no less than nine separate attacks. Females are more liable to relapse than males, just as they are more prone to chorea; the proportion of the sexes in second attacks, however, is nearly the same as in first attacks, but in the cases in which more than two occur the predisposing influence of sex is very strong, the proportion of males being much below the average, while almost all patients who have more than three attacks are of the female sex. The disease very seldom recurs between eighteen and thirty, except in connection with pregnancy.

Preceding rheumatic fever seems to have no influence on recurrence. I have found a history of it in precisely the same proportion of the cases that recurred as in the whole series. Organic heart disease is unquestionably more frequently present in recurrences than in first attacks. It was distinct in one-half of the cases in which the attack was the second or the third, and in all the cases in which the number of attacks exceeded three. This might, indeed, be expected from the fact that endocarditis often occurs in chorea without other discoverable cause; the more attacks of chorea a patient has had, the greater will be the probability of consecutive heart disease. The same conclusion, that the organic heart disease is, for the most part, the result of the repeated attacks, is also shown by the fact, mentioned above, that a history of rheumatic fever is not more frequent in recurrences, and also by the not infrequent cases in which, during a first attack there is no distinct evidence of valvular disease, but in which, at the second attack such disease is unmistakable, although there has been no rheumatic fever in the interval.

The interval between the termination of the first and commencement of the second attack of chorea varies from a few weeks to two or three years. It is rarely less than two months or more than two years. The average interval is about one year. In only one-third of my own cases was the interval more than a year, and in only two did it exceed a year and a half. If, therefore, a patient has remained well for eighteen months after the chorea is over, it is improbable that another attack will occur. The intervals between subsequent recurrences present similar variations. In one case, however, a third attack commenced only two weeks after the termination of the second. The intervals usually vary much in the same patient, and, as a rule, there is no uniformity in the time of year at which the recurrence occurs. Remarkable exceptions, however, are occasionally met with, which are of considerable interest in connection with the relation of chorea to season. One girl first suffered from chorea at nine years old. The attack commenced in October, and lasted four months. Every autumn, for the next six years, she became choreic, and remained so until the middle or end of winter. Her eighth attack, however, began in May, and in it (at the age of seventeen) she came under my care, and presented the characteristic signs of mitral constriction, although she had never had rheumatism: the attack lasted four months. The following year a ninth attack commenced in June; it was the shortest of all, lasting only a month, and has, I believe, been followed by no recurrence. Another girl was attacked with chorea at seven, after distress at the death of her mother, and in each of the next five years chorea came on in late spring or early summer, and lasted for three or four months. I saw the sixth attack, which commenced in May. There was a loud mitral regurgitant murmur, and she also had never had rheumatic fever.

Fright is to be traced as a cause of recurrence in at least as large a proportion as of first attacks. One girl, for instance, had attacks at 15, 17, 17½ and 18, all of which were apparently excited by fright. The attack at 17½ commenced the morning after she had been frightened by a severe thunderstorm; the last attack began a week after she had been much startled by the touch of an unseen person when she thought she was alone. On the other hand, in the case in which there were nine attacks, not one of them was excited by fright. In the girl who had six attacks, the first five occurred without discoverable cause; the sixth commenced a few hours after she had been alarmed by the upsetting of a lamp.

The course of the affection in successive recurrences, and the side affected first or chiefly, present no uniformity. In exceptional cases the commencement is on the same side in every attack. Thus, in one patient four successive attacks began, and were more severe, on the left side. In the patient who had nine attacks, the first seven, which occurred in winter, affected the right side chiefly, but in the eighth and ninth attacks, which occurred in summer, the left side suffered most. The symptoms of the recurrent disease are similar to those of first attacks, but its degree is often slighter and its duration shorter. In this respect, as in most others, chorea shows its characteristic variability; relapses are occasionally most severe. A lad had attacks of chorea at five and seven, each lasting three months, and at ten, without exciting cause, a third attack came on, of great severity, which lasted, varying in intensity, for more than twelve months, in spite of most careful treatment. In this case there was no organic heart disease, but I have more than once observed distinct signs of endocarditis to come on during a recurrence, at the onset of which the heart was healthy.

Terminations and Sequelæ.—The vast majority of the cases terminate in recovery. The movements gradually lessen in severity, until at last they are to be observed only in moments of emotional excitement, and finally they disappear. Usually incoördination ceases, and any weakness passes away, before the spasm has entirely disappeared. The final disappearance is often extremely gradual, and slight occasional movements may be discerned, if carefully looked for, long after the patient is practically well.

In very rare cases considerable muscular weakness, general or local, may succeed chorea. It is a post-choreic paralysis. The loss of power is sometimes very great. There is no change in muscular irritability, but there is, in some cases, impairment of sensibility. It is, perhaps, analogous to the mental weakness which sometimes succeeds chorea, and, like it, always passes away in the course of a few weeks.

Occasionally the disease leaves behind it, for a time, a liability to sudden starts of the limbs, similar to those which many persons experience on going to sleep. In one child they were troublesome for many months. A sudden start in the legs would throw her down, and once the start was such as to make her jump into an open cellar, at the edge of which she was standing. The fact that epilepsy is a rare sequel to chorea has been already mentioned.

When the chorea is attended by considerable mental disturbance, this may continue, as dullness or delusions, for some weeks after the motor phenomena are at an end. Rarely the mental derangement persists as a chronic disease for months or years, but it gradually passes away.

We have seen already that juvenile chorea, in extremely rare cases, instead of ceasing, persists, in more or less severity, as a chronic disease, in spite of all treatment. In the case mentioned, in which the chorea commenced immediately after the discharge of a pistol near the ear, the disease had lasted for three years when the patient came under treatment, and it was still present when he passed from observation six months later. Adults are sometimes seen in the streets presenting all the characteristics of severe chorea, but following their avocations in a way that shows that the condition is one to which they have become habituated. This persistent form, while closely resembling ordinary chorea, does not prevent locomotion. Most of the subjects have been males. It has been pointed out that the proportion of males is unusually large in cases of prolonged duration.

The chorea of childhood rarely terminates in death. The average mortality of a disease so rarely fatal is not easy to ascertain, but it is certainly less than 3 per cent.,* and most of the fatal cases occur at the time of puberty. The proportion of deaths in the two sexes at this time corresponds nearly with their liability to chorea (Sturges), but in earlier childhood the mortality, compared with the number of cases, is relatively lower in boys than in girls. The immediate cause of death is sometimes exhaustion from the severity of the disease and the loss of sleep, sometimes associated rheumatism or rheumatic hyperpyrexia, sometimes intercurrent maladies, as pyæmia from local injuries. Most of the fatal cases are first attacks; death in recurrent chorea is very rare. The chorea of pregnancy is much more fatal, the mortality being 20 or 25 per cent.; but even in these cases death seldom results directly from the chorea; it has usually been due to the effects of delivery or abortion in the cachectic state of the system.

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* Nine deaths occurred among the 439 cases of the B. M. A. Coll. Inv.—a mortality of 2 per cent.

VARIETIES.—*Hysterical Chorea.*—Hysterical patients occasionally suffer from general spasmodic movements closely resembling those of true chorea. Sometimes, indeed, the resemblance is so close that it is impossible to say, from the character of the symptoms, whether the malady is or is not true chorea. The mere existence of symptoms of hysteria has often been taken as proof that the chorea is hysterical in nature, but it is obvious that a patient suffering from hysteria may also suffer from true chorea, and it is certain also that, in a predisposed person, hysterical disturbance may develop during an attack of true chorea, and may even be evoked by it. The only circumstance that would justify the diagnosis of the hysterical nature of such an attack would be its origin by imitation, by witnessing the symptoms of chorea in another patient. In true hysterical chorea the movements generally possess some peculiarity that is not present, or is present but rarely, in true chorea. The most frequent is that the muscular contractions are more sudden and shock-like, resembling those of the so-called electrical chorea. This is a rare feature of true chorea, and although it is not absolutely diagnostic of the hysterical character of the attack, it always affords a strong presumption of this. The second occasional feature, of still greater significance, is a rhythmical character of the spasm. The movements are more or less regular, instead of being irregular. The rhythmical character is especially seen in the movements of the fingers. The spasm is usually of a moderate degree of severity, never so intense as in the severer forms of the genuine malady, but nevertheless the hysterical form is often a very troublesome affection. It may last for many months; its average duration is considerably greater than that of the ordinary form. It should be noted that the term "hysterical chorea" is sometimes applied to other forms of hysterical spasm which bear little resemblance to true chorea; these are described in the chapter on that disease.

Adult and Senile Chorea.—The rare cases of chorea that occur in adult life and old age present some peculiarities. The cases may be placed in two classes according to their course, although we do not yet know whether there is any corresponding difference in the nature of the disease, or whether this differs essentially from the similar disease of childhood. In some cases, chorea in the second half of life runs a course like that of the juvenile form, and, after it has lasted for a few months, passes away. In other cases, instead of subsiding, it persists, and continues, it may be for many years, until the death of the patient. The cases in which the disease persists are unfortunately the more common. The difference does not depend on the age of the sufferer; in one case, which commenced at forty, the disease was permanent,* while in another, in which the symptoms commenced at seventy-seven, the patient recovered after a few months.† Nor is it related to the character of the symptoms, although in the persistent form the affection is usually general, and in the cases that have recovered the movements have often been partial.

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* McLearn, *Lancet*, *1885*, Feb. 21st.

† Russell, *Med. Times and Gaz.*, *1878*, i, p. 459. Another case (of hemichorea), in a

The spasm in the second half of life is generally identical in character with that of the juvenile form. There are the same irregular movements, and the same incoördination of voluntary movement. The spasm is often most severe, and may render intended actions almost impossible. The affection of the face and tongue may be so extreme as to greatly derange articulation, and make it almost impossible to understand what the patient says. The spasm is usually increased by emotion, and the patient, who is thrown into the most violent physical agitation by the visit of a stranger, may be comparatively still when free from excitement. It ceases, as a rule, during sleep, but has been known sometimes to continue. The affection of the legs is slighter than that of the arms, but may be sufficient to render standing impossible. There may be some loss of muscular power, but it is seldom considerable, so far as the spasm permits the strength to be ascertained.

The malady may commence at any period of adult life, even up to eighty years of age. Both sexes suffer, males in a somewhat larger proportion than in earlier life. The subjects of the disease often belong to families in which other neuroses prevail, but the cases are, as a rule, isolated. In a few recorded instances more than one member of a family has suffered from chorea, but these cases have usually differed somewhat in character from the ordinary senile form. Two sisters and a brother constituted the group described by Macleod, but in two of them organic disease was found in the brain. In another instance described by Harbinson three members of a family suffered from a malady resembling that under consideration. In a case recorded by West,* a man, aged forty-five, had lost a brother from chorea at the age of fifty; in another (Ewald†), a mother and two daughters suffered. The most remarkable instances of inheritance, however, are that mentioned on p. 956 (note) and one recorded by Peretti. Of four families descended from a choreic woman, two were healthy, but in the other two, excluding all doubtful cases, twelve persons became choreic in the second half of life (after thirty).‡ When an exciting cause can be traced it is generally some depressing emotion, especially grief, which is so potent a cause of neuroses in late life. A fright has excited the disease only in those who were still in middle life. Thus in a case described by Berkeley,§

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woman of 74, ending in recovery, is recorded by Ferguson, *Lancet*, July 11th, 1885. Other cases of adult and senile chorea have been recorded by Charcot, *Med. Times and Gaz.*, March 9th, 1878; Macleod, *Journal of Mental Science*, July, 1881; McLearn, *Lancet*, *1885*, No. viii; Saundby, *Lancet*, Nov. 29th, 1884. In the last paper references will be found to other cases recorded by Graves, Sinkler and others.

* S. West, *Brit. Med. Journal*, *1884*, Jan. 5th.

† *Zeitschr. f. klin. Med.*, *1884*, Bd. vii.

‡ Peretti, *Berlin, klin. Wochenschr.*, *1885*, No. 50–52.

§ *Med. News*, Aug. 25th, 1883.

the patient, a woman, became choreic a few hours after a severe fright, at the age of thirty-four; the disease continued till her death, seven years later. A case of similar causation has been recorded by West.*

The malady in the second half of life does not seem to have any special relation to heart disease or rheumatism. Valvular disease, when present, has been apparently due to chronic degeneration, and any rheumatism from which the patients had suffered has also, as a rule, been chronic.

In many cases the mind is unaffected, but in some there has been progressive chronic dementia, and in a few there has been some maniacal excitement. Hence many cases of senile chorea have been reported from asylums for the insane. The duration of the disease, in cases that have recovered, has been from a few months to one or two years. That of the persistent form is often long, for the disease has but little tendency to shorten life. Charcot mentions two cases at the age of seventy-one; in one of which the disease commenced at fifty-nine, and in the other at sixty. In a case recorded by McLearn the disease commenced at forty, and had already lasted fifteen years when the patient came under observation.

The malady termed "electrical chorea," and some other diseases allied to chorea, are described at the end of this chapter.

PATHOLOGICAL ANATOMY.—As a rule, the nerve centres in chorea present no abnormal appearances on naked-eye examination, and the lesions occasionally found have for the most part been clearly coincident or consecutive. This is true, also, of the minute changes that the microscope has more frequently revealed. Cerebral hemorrhage, evidently secondary, has been found in rare cases. Somewhat less rarely spots of softening have been found (Todd, Tuckwell, and others), usually small, occasionally more extensive. Sometimes there is embolic plugging of a large vessel, an associated consequence of the endocarditis. In one case (Gray) the basilar, vertebrals, and middle cerebrals were all thus closed. Similar obstruction of minute vessels, especially in the corpora striata and cortex, have been found on microscopical examination (Tuckwell, Bastian, Long, Fox, Elischer, etc.), and have usually been regarded as embolic, but as thrombotic by Bastian. They are not invariable, for they have often been carefully looked for without success (by Dickinson, myself, and many others.) Effusion into the cerebral ventricles has been very rarely observed, and it is only in cases of extremely chronic course, which have lasted for years, that any trace of meningitis has been seen. Changes in the nerve cells have been found by some recent observers, hyaline swelling and degeneration in the central ganglia (Meynert), and in the claustrum and island of Reil (Elischer). The connective tissue of the brain has been found increased (Meynert, Rokitansky) and the specific gravity of the central ganglia lowered (Aitken). In one chronic case Golgi saw what he considered to be calcification of the cells of Purkinje in the cerebellum. No special significance can be attached to the fatty degeneration of the perivascular sheaths described by some observers, or to the erosions around vessels observed by Dickinson, since both these changes are common at all ages.

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* S. West, *Brit. Med. Journ.*, *1884*, Jan. 5th.

In the spinal cord local softening has rarely been found, chiefly in the cervical region (Hutchinson, Lockhart Clarke). Extravasation of blood around minute vessels is frequent—very rarely there is more extensive hemorrhage. Corpuscular elements have been found aggregated in the walls of the vessels (Elischer) and fibrinous plugs within them (Ross). The nuclei of the interstitial connective tissue have been increased. The large nerve cells were found by Elischer to present signs of degeneration, somewhat questionable in significance (pigmentation and indistinctness of nuclei). Inflammation of the spinal dura mater was found in one case by Frerichs, but was doubtless an accidental complication, for, as a rule, the membranes are free from any other morbid state than passive hyperæmia.

In the peripheral nerves, Elischer found an increase of connective tissue, and changes in the axis cylinders, which presented hyaline swelling, and stained less readily than in health.

Such of the above changes as are not accidental concomitants of chorea are probably the result of the excessive and perverted functional activity of the nerve elements. It is important to remember that tissue changes set up by such functional disturbance may develop to some extent independently of their cause. This is well illustrated by the pathological anatomy of chorea in the dog, an affection which differs from the ordinary form of chorea in man, in the shock-like character of the muscular spasm, resembling in this the so-called "electrical chorea." Of two typical cases examined by Sankey and myself, one presented only slight degenerative changes (granular swelling) in the motor nerve cells of the spinal cord; in the other these degenerative changes were more intense, the cells were extensively vacuolated, and the spinal cord, medulla and cerebellum presented areas and foci of intense nuclear (leucocytal?) infiltration, situated in different parts of the gray or white substance, and apparently of secondary origin, and of independent course. Similar changes have also been found by Horsley.

In the persistent chorea of the old, changes have been occasionally found in the brain; more frequently the examination has failed to reveal any alterations that can be regarded as the cause of the disease. Slight degenerative changes, such as were found extensively distributed through the central nervous system by Berkeley, in the case of seven years' duration (see p. 974), are probably in part of secondary origin. In one of the cases recorded by Harbinson, there was evidence throughout the brain of degeneration of the nerve cells, together with an increase of the lymphoid elements, which were aggregated around the vessels.* In all the three cases recorded by Macleod (see p. 973), in which, however, there was also motor palsy, disease of the motor region of the cortex was found; in two, the convolutions were compressed by cystic thickening of the membranes, in the other there were small tumors.*

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* *Path. Trans.*, *1882*, p. 33. The changes were examined for the Society by Dr. Savage and myself. Similar changes seem to have been found by him in two other cases in the same family (*Med. Press and Circular*, *1880*, Feb. 18), but the existence of actual sclerosis is doubtful (see report to the Path. Soc., loc. cit.).

Outside the nervous system, changes are usually found only in the heart. Of eighty fatal cases collected by Sturges, the heart was healthy in only five. The most frequent change is endocarditis, usually of the mitral valves, which present delicate vegetations, especially on the auricular aspect. Similar vegetations are sometimes found also on the aortic valves. Occasionally, more extensive valvular disease, of older date, is present. Recent endocarditis may be found when there has been no antecedent rheumatism. Although very frequent, endocarditis is not invariable, and it has even been absent in a case in which the disease followed acute rheumatism. The changes met with in the chorea of pregnancy are similar; endocarditis is usually present, but in rare cases is absent.

PATHOLOGY.—The problem of the pathology of chorea resolves itself into three questions, (1) What is the primary seat of the disease? (2) What is the nature of the change in the nerve elements? (3) What is the cause of that change?

(1) The changes that have been found after death afford no clear indication of the part primarily deranged. They have been found widely distributed, in the brain, spinal cord, and peripheral nerves, and are, moreover, inconstant. They are certainly, for the most part, either merely concomitant or secondary to the functional disturbance of the nerve elements. Organic disease has been found only in cases that differ in some respects from the common, juvenile form, and the significance of these lesions will be considered presently. The uncertain indications of pathological anatomy must therefore be interpreted by the help of the symptoms themselves. These symptoms supply strong evidence that the primary seat of the disease is in the brain. The fact that the movements cease during sleep, is opposed to their origin in the spinal cord, the functions of which are in a state of increased activity during sleep. The frequency with which chorea is unilateral or commences unilaterally, suggests that it depends on an affection of the motor elements, where those related to the various parts of one side have more in common than have those for the upper or lower limbs of both sides; *i. e.* that it depends on an affection in the cerebral hemispheres. A closer study of the distribution of unilateral chorea affords further support to this opinion, since the greater affection of the arm than the leg points to a region in which the more complex movements of the arm are proportionately represented, and agrees with the distribution of unilateral paralysis and convulsion of cerebral origin. Since chorea, beginning unilaterally, often becomes general, we are justified in believing that the latter depends on an affection, in both hemispheres, of those structures, the disturbance of which in one hemisphere causes hemichorea. Choreic movements have been produced by organic disease in so many parts of the brain that no conclusion can be

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* *Journal of Mental Science*, July, 1881.

drawn from such cases as to the probable seat of the morbid process that causes ordinary chorea. Such movements have been caused by lesions of the cortex, central ganglia, and crus cerebri.*

The motor impulses that excite the muscles pass to the spinal cord direct from the motor region of the cortex. It is here that movements are arranged, and it is certain that if movements are disordered, as they are in chorea, and the disorder proceeds from the brain, it must depend directly on a disordered action of the cells of the motor cortex. We must assume, therefore, that there is such disorder in chorea. Is this derangement primary? Is this the actual seat of the disease, or is the disorder here merely secondary to a morbid process elsewhere? To this question no definite answer can at present be given. It is, however, doubtful whether there are at present any facts to justify us in going beyond the cortex in our search for the primary disturbance. At the same time we know very little of the mechanisms that guide the cortex in arranging movements. It does not seem that there is any coördination of movement in the brain after the motor impulse leaves the convolutions. The impulse passes directly to the spinal cord. The influences that guide the cortex in arranging movements, the influence of the cerebellum, for instance, however exerted, must be exerted on the cortex, or on nerve processes anterior in time to those in the cortex. It is possible that the derangement of such a process may be the cause of chorea, but we have no evidence of the fact.

It was formerly thought that the corpus striatum is the part primarily diseased, but this opinion was based upon the theory that the movements are arranged in the central ganglia after the impulses leave the cortex. We now know that this is impossible, and therefore the ground on which this theory was based has disappeared. Moreover, we know of no anatomical arrangement by which the gray matter of the corpus striatum can influence the cortex. The optic thalamus, however, stands on a different footing. Choreoid movements have been caused by disease of the thalamus, and it is connected with all parts of the brain. It has been conjectured that it is a centre through which peripheral impressions, which do not affect consciousness, influence the action of the cortex, and it is possible that a primary derangement of the thalamus may be the cause of chorea. But of this we have no direct evidence, and the fact that its disease may cause choreoid movements, does not prove that ordinary chorea is due to its disease. The difference between the two conditions is great, and it is quite possible that a disorder of the action of the cortical cells, which may be secondary to the disease of the thalamus in the one case, may also occur as a primary change in the other.

It seems probable, moreover, that the nerve cells of the cortex are the seat, not only of disturbed function, but also of disturbed nutrition. The change in the electrical irritability of the nerve fibres of the limbs means an alteration in their molecular nutrition, and this, from all analogy, must pend on a similar change in the nutrition of the motor cells of the spinal rd. But if the disease is not a primary one of the cord, it is reasonable assume that, just as the change descends to the peripheral nerve fibres m the spinal cells, so it must descend to the spinal cells from the brain, at is, from the cells of the cortex, and that in these it exists in even greater gree than in the spinal cells.

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* Cases from disease of the crus have been described by Magnan (hemorrhage), and by Canfield and Putnam (softening).

It may be considered that the absence of the peculiar epileptiform convul- ns which so often result from organic disease of the motor-cortex is a fficulty in assuming that this part of the brain is the region primarily seased. The weight of the objection may, however, be over-estimated. hatever be the seat of the disease, the morbid process of chorea is evi- ntly one of peculiar character, and the absence of such symptoms as are oduced by organic disease may be due solely to the nature of the morbid ange. Moreover, epileptiform fits, such as are characteristic of cortical sease, are not absolutely unknown in chorea, as the case mentioned on p. 7 shows.

There is other evidence that the functions of the cortex are impaired in orea. We must refer to this cause the mental dullness which is so common, d to this, also, we must refer the maniacal disturbance that occasionally companies chorea. Perhaps, indeed, ordinary acute mania presents a ore close analogy to chorea than does any other malady. The two are mparable in course and in duration, and they are not altogether dissim- r in their characters. Chorea has been called "insanity of the muscles," d mania might also be called "chorea of the mind." Finally, the facts at symptoms resembling chorea may result from organic disease of the rtex (Macleod), and from experimental lesions in the monkey (Couty), ay be admitted as affording some support to the opinion that this is the rt primarily deranged in the ordinary form.

The intense over-action of the nerve elements affords an explanation of the ght and variable morbid appearances occasionally found—the indications the vascular disturbance, and the increase in connective tissue in cases of ng duration. The distention of the vessels, and the perivascular hemor- ages, are doubtless due chiefly to the mechanical distention which usually ecedes death.

Does the spinal cord merely act as a conductor or does it intensify the asm? The peculiar choreic spasms of the dog persist after the separation the spinal cord from the brain; but this disease differs from the chorea of an too much to permit us to draw any inference from the fact. It has been njectured that some of the spasm depends on the cord, but there is, as t, no direct evidence of this, although its possibility is clearly shown by e fact that unilateral choreoid spasms in the monkey, produced by a lesion the surface of the brain, may persist after division of the spinal cord outy).

(2) The affection of the nerve elements, whatever its primary seat, must one of peculiar character, with a definite tendency to increase, extend, d subside; it must consist of changes that can pass away entirely. The

symptoms show that there must be a tendency to spontaneous action of nerve cells, a tendency for this action to spread unduly among the cells, and a diminution in the possible energy of action. In the language of modern physiology, we may say that the "resistance" is lowered alike in the cells themselves, and in the connections between the cells by which the character and degree of their associated action is determined. This is, of course, only a statement of the nervous phenomena that correspond to the muscular phenomena; it is not an explanation of the nature of the change in the nerve centres. In this connection, and in relation to the theory that the cortex is the part primarily disturbed, the tendency to extension of the functional disturbance deserves special note. It spreads from one side to the other, just as does a convulsion due to organic disease. The latter is certainly due to the spread of "discharge" from the motor cortex on one side to that on the other side: the over-action of the cells has a tendency to excite the over-action of other cells that are connected with the first, and a similar tendency to the extension of functional action may be the mechanism by which the disorder of movement extends in chorea.

(3) By what morbid influence is the derangement of function of the nerve cells produced? This is the essential problem of the pathology of chorea. Morbid anatomy throws no light upon it; no change has been found with sufficient frequency to justify the assumption that it indicates the nature of the primary lesion. A theory of the nature of the disease must therefore be based, not only on the meagre facts revealed by the scalpel and the microscope, but also on those which have been ascertained regarding the causation and the pathological relations of the disease.

The first important etiological fact is the distinctly predisposing influence of age and sex. Chorea is especially a disease of later childhood. That is, it occurs when the functional education of the nerve centres has been to a large extent effected, but is not yet complete; when the directions of functional activity and the lines of resistance have been developed, but have not yet received that stability which only long-continued action, with its underlying nutrition, can achieve.

The age and sex most liable are also those in which emotion is most active and exerts the greatest influence on the motor nervous system—in which, indeed, as Sturges has insisted, the physiological expression of emotion is often by movements bearing no distant resemblance to the slighter spasms of chorea. With this predisposition is to be associated the important fact that an emotion has been recognized, by almost all observers, as the one exciting cause that can be traced with frequency. Were these the only facts of etiology, it would be natural to regard chorea as a purely functional disease, in the strictest sense of the term, a disturbance of the functions of the motor centres, conditioned by their state of functional development, and owning no other cause than an activity in constant or occasional excess of the stability of the nervous mechanism. This theory, as an exclusive explanation of chorea, has been indeed put forward by Sturges.

But the association of chorea with rheumatism and with endocardial lesions is too frequent, and often too close, to be explained away as mere coincidence, and too important to be disregarded. Although the proportion of cases of chorea in which there is a history of acute rheumatism (about one-fourth) is not large, it is too large to be the result of accident; and this explanation is rendered entirely inadmissible by the evidence of the close relation between the diseases afforded by the cases in which the two are immediately associated, in which an attack of chorea succeeds acute rheumatism or the latter occurs during the course of the chorea. Moreover, their close relation is emphasized by the lesion which is common in each, even when dissociated, endocarditis, and which is so rare in other maladies, nervous or general. Yet it is impossible to regard chorea as the result of acute rheumatism, since each disease occurs so frequently without the other, and no relation has been traced between chorea and the common cause of rheumatism, exposure to cold.*

The question of the association of chorea with rheumatism cannot be separated from that of its relation to the cardiac disease which is common to both. The great frequency with which endocarditis is found post-mortem in fatal chorea has given rise to the theory (originally advanced by Kirkes and advocated by Hughlings-Jackson, Broadbent, and others) that chorea is due (some say occasionally, others always) to minute fragments being washed off the cardiac valves, and obstructing the small vessels of the motor centres in the brain. The supporters of this theory appeal to the frequency of endocarditis, and to the delicacy of the vegetations on the valves (which are such as to supply minute plugs), to the evidence of such capillary obstruction which has occasionally been found post-mortem, and to the proof which larger infarction has (rarely) supplied of the reality of the occurrence of embolic processes. They assume that rheumatism causes chorea by producing endocarditis.

On this theory, endocardial disease must precede the symptoms of chorea. But it is only in a very small proportion of the cases that this precedence can be traced. Even in severe and fatal cases, endocarditis has been found absent after death. In the large number of cases which recover, and in which no sign of endocarditis can be detected, we are not justified in assuming its existence. In other cases, again, the signs of endocarditis come on during the course of the chorea. In these two classes, which comprise the majority of the cases of chorea, the embolic theory is inapplicable, except on the assumption that in two-thirds of the cases in which endocarditis exists it causes no symptom—an assumption which is rendered quite unjustifiable by the fact that the absence of endocarditis, even in severe cases, has been proved post-mortem.

The actual obstruction of cerebral vessels, large or small, which has occasionally been observed, may easily be over-rated as evidence of the embolic origin of chorea. A careful search has so frequently failed to discover emboli that we must regard them as occasional only, even when there is endocarditis. Occasional embolism does not do more than emphasize the fact that endocarditis exists; it does not prove that the chorea is due to vascular obstruction. As an explanation of the relation of rheumatism to chorea the embolic theory also fails, since in a fatal case of chorea after rheumatism, recorded by Sawyer, no trace of endocarditis could be found. Fright is effective in causing chorea in cases with preceding heart disease, as in others which present no sign of endocardial mischief throughout their course. It is exceedingly difficult to reconcile the influence of fright with the embolic theory.*

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* The facts ascertained by Morris Lewis, even if generally true, invalidate this conclusion.

If the endocarditis is not the cause of chorea it must be either the result of the chorea, or the two must be consequences of a common cause. This conclusion is supported by the fact that, if we are to trust clinical evidence, the endocarditis is often secondary to the chorea in point of time. Pathology at present knows nothing of the direct dependence of such a lesion on an altered state of the nervous system. All known facts regarding the etiology of endocarditis point to its dependence on morbid states of the blood. That excessive muscular action changes the composition of the blood is practically certain, but the endocarditis can scarcely be ascribed to this cause, since it does not occur in other states of muscular over-action; it may be present even in slight cases of chorea, and it may precede the chorea in its development. The hypothesis which seems best to embrace the facts is the old theory that the common cause of the endocarditis and the chorea is a blood state, or at least a condition of the system, including the blood, which may be most conveniently spoken of as a blood state. This state may be allied to, but not identical with, that which causes acute rheumatism. According to the precise nature of the blood change, chorea or acute rheumatism or both, with or without endocarditis, may be produced. Of the mechanism by which the assumed blood changes cause chorea, we are still ignorant. Bastian, because the minute vessels of the corpus striatum have been found plugged with white corpuscles, has suggested that the mechanism is by spontaneous thrombosis in these vessels. But against this theory is the fact that ordinary cases of chorea exhibit no tendency to vascular thrombosis, such as might reasonably be expected to be observed, at least occasionally, and also the fact that such capillary thrombosis has seldom been found. The hypothesis (accepted by Leube) that the blood change is a chemical one, having a specific action on a certain part of the nerve centres, is more probable, since the facts of toxicology show a large number of chemical substances have an action limited in a remarkable way to certain parts of the nervous system.*

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* It has been suggested that the disturbance of the heart's action produced by fright may be the means of detaching embolic fragments. But this explanation cannot be accepted for the cases of chorea due to fright in which there is no endocardial mischief, or for the influence of other depressing emotions, and we are driven to admit for these cases a direct influence of the emotion on the nerve centre. But a theory which must assume that the influence of rheumatism and fright in the causation of chorea is sometimes exerted in one way, and sometimes in a totally different mechanism, can scarcely be regarded as admissible. That extensive capillary embolism may give rise to movements resembling those of chorea has recently been proved by Dr. Money (*Med.-Chir. Trans.*, vol. lxviii); but the fact has little bearing on the question of the pathology of human chorea. The facts of disease show that many morbid processes in the brain may give rise to choreoid movements.

Occasionally chorea is accompanied by such severe general symptoms as to suggest the influence of a very intense blood disturbance. The concurrence of rheumatic hyperpyrexia may be regarded as a complication, but it is a very significant one. In the case mentioned already, as fatal by ulcerative endocarditis, the symptoms of this coincided in onset with those of the chorea. In another recorded case the symptoms of chorea in a child of seven were quickly followed by those of an intense blood disease, which was quickly fatal.†

We must not, however, regard the blood change as the exclusive, or even the dominant element in the causation of chorea. Important facts already considered demonstrate the influence of functional development as a predisposition and of functional disturbance as an excitant of the disease. No theory is complete that does not take cognizance of all three factors. The ascertained facts of etiology seem to show that the part played by the three influences varies in different cases. Always, probably, the functional predisposition is essential; it may in some instances attain such an intensity that the disease occurs without other causes; in other cases the change in the blood, or the influence of emotion, or both, are combined with the predisposition in various degrees.

The nature of the influence which pregnancy exerts on the occurrence of chorea is still unknown. It is commonly regarded as a reflex influence, analogous to that which causes vomiting, but it is to be observed that the period of development of chorea is usually later, and is much more variable, than that of the morning sickness, and often does not cease immediately on the removal, by parturition, of the source of reflex irritation. We know, also, very little of the occurrence of chorea from other forms of reflex irritation.

The influences which determine the precise form that the chorea assumes are also unknown. Maniacal chorea is not met with until the period of puberty, and is very rare in the male sex. It occurs in girls between fourteen and twenty, and not unfrequently during pregnancy—conditions of age and sex in which emotional disturbance is readily produced.

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* If the conception of a blood state allied to, although not the same as, that which causes acute rheumatism, seems difficult, we may remember that the varieties in the manifestations of rheumatism must be evidence of a corresponding variety in the constitutional condition that predisposes to it. Of a number of persons who get their feet wet, for instance, one will have a cold in the head, a second acute tonsillitis, a third pericarditis, a fourth acute general arthritis, and a fifth arthritis with endocarditis. The cause is the same in all, the effect must be due to the previous condition of the individual. It does not seem inconceivable that an allied constitutional condition should have an influence on certain nerve centres, rendering their function and nutrition susceptible of derangement.

† Nauwerck, "Beiträge zur Path. Anat. u. Phys.," by Ziegler and Nauwerck, Jena, 1886, p. 407. Foci of inflammation (mycotic?) were found in the brain and medulla, and there were also pericarditis, endocarditis, and pneumonia.

Diagnosis.—No disease is more easily recognized than chorea in its common form. The peculiar movements at once attract attention; their character is unmistakable; and their recent onset distinguishes the case from the only condition in which similar movements occur—that of congenital disease of the brain. The expression of countenance, with its listless, somewhat vacant aspect, often by itself suggests the nature of the malady. It is only in some special varieties of the disease that any diagnostic difficulty arises. In the rare cases in which the legs suffer more than the arms, the interference with standing and walking may cause the condition of the patient to resemble that of paraplegia, but the spasmodic movements are always conspicuous, and show the nature of the case. A greater difficulty is sometimes presented by the cases of "paralytic chorea," in which spontaneous spasm may be difficult to detect, and the loss of power alone attracts the attention of the friends, and sometimes also of the medical attendant. The distinction rests on the fact that the loss of power is confined to one arm, and does not involve the face or leg, that it comes on gradually (in the course of a few weeks) in later childhood. Close observation will always detect slight occasional choreic movements in the weak arm and often elsewhere, or there may be distinct incoördination, sometimes most manifest on sustained exertion, as in holding the hand over the head—an action that often reveals the existence of choreic movements more distinctly than any other. In other cases sustained muscular action is impossible; the grasping fingers, for instance, relax involuntarily, or desired relaxation is for a moment delayed. Once aware of the possible nature of the case and the diagnosis is not difficult. As a rule, when a child between seven and twelve years of age is said to have gradually lost the use of one arm, the disease is chorea.*

Maniacal chorea, when the muscular spasm is slight, may be mistaken for simple mania. There is, however, less continuous garrulity than in simple mania; usually there is sufficient spasmodic movement to show the nature of the case; and the patients are younger than most subjects of acute mania. The greatest difficulty arises in the rare cases in which the muscular spasm ceases when the mental disturbance becomes considerable, and there is only the history of the spasm to guide the diagnosis.

The diagnosis of hysterical from ordinary chorea rests on the suddenness and isolation of the muscular contractions, on their occasional rhythmical character, on the age and sex of the patient, and on the frequent origin of the disease in imitation. The sudden shock-like character of the contractions is characteristic also of the disease termed "electrical chorea," which is described in a separate section.

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* This statement may seem too absolute, but it is literally true. Organic disease either weakens the leg as well as the arm, or else it causes convulsions or some other symptom that attracts attention, and is mentioned at the outset.

Chorea in the adult is recognized by the resemblance of the movements to those seen in earlier life. An occasional difficulty is due to the rarity of the disease, in consequence of which many practitioners are ignorant of its occurrence, and they do not think of actual chorea when the symptoms present themselves in an old person. A greater difficulty is due to the fact that there are many varieties of spasmodic disorder bearing more or less resemblance to chorea, so that the question may arise in a given case, "Should this disease be called chorea or not?" The difficulty is chiefly one of name. It has been customary to call by this name most cases of general clonic spasm, irregular in character, not distinctly forming part of some other disorder. What separable affections are thus included, future investigations must decide.

PROGNOSIS.—In the vast majority of cases, even of severe chorea, the prognosis is favorable. As long as the disease is free from complications, and the patient obtains a fair amount of sleep, no anxiety need be felt. The chief source of danger is exhaustion, from the violence of the movements, and from the deficiency of sleep. The endocarditis has little influence on the immediate prognosis, the only risk to life that it entails is through the danger of embolic processes, and this is extremely small. Intercurrent rheumatism is also commonly of a mild type, and the gravest complications of rheumatism—pericarditis and hyperpyrexia—are very rare in chorea. Preceding heart disease renders the prognosis worse in proportion to the gravity of the cardiac lesion. It is also worse in the maniacal than in the ordinary form, and considerably worse in the chorea of pregnancy than in that of youth. It is somewhat better in a relapse than in a first attack, but to this rule the chorea of pregnancy is an exception.

The more severe the disease, the longer will be its probable duration. Relapses will probably terminate sooner than first attacks. Etiological conditions of age, sex, cause, preceding rheumatism or heart disease, and present endocarditis, afford no indications of the probable duration of the disease. This conclusion is drawn from a careful analysis of the facts of the cases which have come under my notice. In hysterical chorea the prognosis as to life is absolutely favorable, but the duration of the disease is often long.

Of the sequelæ of ordinary chorea, mental and muscular weakness always pass away. It is only when there has been severe mania that there is any risk of the persistence of dementia. Convulsive attacks, if of epileptoid type, may continue to recur, and, as we have seen, may develop into epilepsy.

TREATMENT.—The derangement of motor and psychical functions in chorea, its frequent origin in emotion, and the distinct increase of the symptoms, that may often be observed to result from emotional disturbance and from mental and physical exertion—all teach the lesson, which experience confirms, that the first important element in treatment is to secure the patient from causes of mental and physical fatigue and emotional excitement. Lessons should be discontinued, and, even in slight cases, the periods of physical rest should be increased. If the disease is not severe, a certain amount of muscular exercise does not appear to do harm, but it must be carefully regulated. In severe cases there is only too much spontaneous muscular action, and all voluntary exertion should be avoided by absolute rest in bed during the height of the disease. Even in moderate cases the beneficial effect of a few days' absolute rest is most conspicuous, and is desirable at the commencement of treatment. Often, in such cases, prolonged rest is not followed by a continued improvement, and the patient may be allowed to be up during the greater part of the day, provided the movements are not thereby increased in intensity. If the severity of the disease does not make prolonged rest in bed absolutely necessary, the mental depression produced thereby often more than counteracts the good effected by the rest. All sources of mental irritation should be avoided ; a cheerful room and cheerful companionship are most important.

When the movements are severe, extreme care must be taken to obviate the chance of injury from the contact of the limbs with hard objects. These should be removed out of the range of the limbs, or covered with soft padding. The importance of this measure cannot be over-stated. One cause of death in chorea is the effect of slight mechanical injuries, which, in the cachectic state of the patient, often run an unfavorable course, and may lead to blood poisoning. The risk that the patient may be thrown out of bed by the spasm must not be forgotten. In severe cases it is well to place the patient's bed on the floor in a corner of the room, and to cover the adjacent walls with cushions or mattresses. If bedsores are threatened, a water bed should be obtained. The influence of the spasm must be borne in mind in all proceedings that are adopted. The temperature, for instance, should never be taken in the mouth. I have known a choreic patient to bite off and swallow the bulb of a thermometer placed in the mouth ; fortunately, the accident was followed by no more serious consequence than an increase in the chorea from the alarm occasioned ; the thermometer bulb was safely passed next day, per rectum.

It is needless to insist on the importance of an adequate supply of easily-digested food, and of absolute cleanliness. The latter is of great importance when the patient's mental apathy causes unnoticed evacuations, for at such a stage the spasm often lessens, and comparative stillness increases the risk of bedsores from pressure. The bowels should be kept open, but purgation is useless.

In many severe cases of chorea the skin is dry and harsh, and in these a free diaphoresis has often the most striking effect in lessening the intensity of the disease. The old method was to give a small dose of antimonial wine, and follow this with a hot-air bath, and I have seen excellent results from this treatment. But it is only when the strength is good that the antimony is admissible ; the hot-air bath may always be employed if the state of the skin indicates its use.

The influence of drugs on chorea is a subject on which the most diverse opinions have been expressed. Some physicians, drawing their conclusions ɔm the observation of individual cases, are confident that its severity may : lessened and its duration shortened by certain medicines. Others have ımpared the course and duration of cases which have been thus treated th those of cases in which no drug has been given, and finding no marked fference between the two sets, have inferred that the natural tendency to covery has influenced the conclusions of others, and that chorea is not pable of being influenced by drugs. My own belief is that the facts on ɪich each of these conclusions, apparently contradictory, is based, are in e main correct. The majority of cases of chorea, if they are admitted at ı early stage into a well-arranged hospital, and if kept at perfect rest, seem get well as speedily as the nature of the disease will permit, and it is :ceedingly difficult, in such cases, by isolated or collective observation, to ɔtain satisfactory evidence of the influence of drugs on the disease. On e other hand, in cases that are treated under less favorable conditions, in ose that are seen as hospital out-patients, or in which the disease has lasted r a long time, the duration of the symptoms is much longer, the tendency spontaneous cessation is much less marked, recovery is often exceedingly ɔw, and the influence of drugs is often so distinct, as to justify a doubt ɪether the cases which have given rise to the first conclusion are altogether ɜe from fallacy, and to suggest the desirability of employing those agents ɪich seem most useful, even when their effect is difficult to trace.

Sedatives and tonics have both been largely employed in the treatment of ıorea. Of the former bromide of potassium is of singularly little value. hloral hydrate is more useful. In severe cases it is often of great service producing sleep, but in cases of moderate severity its chief use is to crease the tranquillity obtained by a period of absolute rest in bed at the ıset of the treatment. Ten or fifteen grains may be given every four or ɛ hours. Some physicians have advised that the patients should be kept nstantly asleep by chloral, but this method of treatment is scarcely comɜnded by a case recorded by Bouchut, in which continuous sleep had to be aintained for a month before the movements ceased. In other cases thus ɜated it has seemed that the period of sleep was merely interpolated in the sease, the course of this being not otherwise modified. Large doses of loral sometimes succeed where small quantities fail, as in one case (Gairdr) in which an accidental dose of sixty grains had a striking effect. Inha:ions of chloroform have been employed with advantage to secure rest in ɾere cases, but they have been practically superseded by chloral, which oduces the same effect. Morphia as a sedative is far inferior to chloral. smaller, stimulant doses (five minims of the liquor three times a day, as vocated by Radcliffe) it seems to be useful in slight cases, but it should t be given if there is considerable mental excitement. I have known ɔh excitement to be distinctly increased by morphia. Henbane, hyoscyıin, conium, Indian hemp, Calabar bean, cimicifuga, and other sedatives ve been employed, but are of doubtful value.

Among the so-called nervine tonics, arsenic has long enjoyed a high repuion, which it certainly to some extent deserves. It is usually given by the mouth, and the dose should be gradually increased to ♏ x or even ♏ xv of the liquor three times a day. It may be combined with morphia (Steiner). The comparative intolerance of the stomach for arsenic has led to its hypodermic injection (Eulenburg, Hammond). By this method much larger doses can be given without unpleasant symptoms, but the emotional disturbance occasioned to children by the operation is a drawback to the method in a disease in which it is so important to maintain emotional tranquillity. In older patients this drawback is of less moment. Hammond recommends, as an initial dose, from five to thirty minims (!) of Fowler's solution, diluted with an equal quantity of glycerine, and, as the best place for the injection, the loose skin on the front of the forearm. Arsenic may be given by the mouth until intolerance is reached, and then continued by hypodermic injection.

Zinc is another drug that has long been praised in the treatment of chorea; both the oxide and sulphate have been given, the dose being slowly increased up to a scruple of either. So far as I have seen, it scarcely deserves the encomiums which have been bestowed upon it, and the apparent influence of the increasing doses has probably been sometimes due to the time necessarily occupied in their administration.

Strychnine is of little value in the early stages, but it is often distinctly useful when the disease lingers on in slight degree. I have frequently seen slight cases which had been stationary for a considerable time rapidly recover under its influence. Calabar bean has been recommended by Harley, and its alkaloid, eserine, by Bouchut; the latter in doses of $\frac{1}{25}$ grain two or three times a day. The published facts are not, however, very convincing as to its utility. Curara has been employed in obstinate and chronic cases with alleged advantage (Diamond, Wright), but it is doubtful whether the employment of so dangerous an agent is ever justifiable. The use of salicylate of soda, recommended by Weir Mitchell, would seem to be justified by the relation of the disease to rheumatism, but the cases are few in which it has a distinct influence on the disease.

Electricity has been employed in various ways, especially the voltaic current to the spine, but it is very doubtful whether the agent has any real influence. The spasms are usually lessened by the passage of the current through the limbs, but the effect quickly passes away, and I have never observed evidence of permanent benefit. A few instances of rapid cessation of chorea on the application of magnets have been reported by Benzol and Hammond, but the latter has since stated that in other cases the method has invariably failed. Massage has been employed, and has been thought to do good;* in various forms of spasm it has a marked sedative effect. Another therapeutic measure which has been recommended, but is of very doubtful value, is the freezing of the skin over the spine by ether spray.†

Rhythmical movements and mild gymnastics are of service in the later ges of the disease, when the normal functions of the centres are being stored, but they are not advisable in the earlier stages except in cases of ry slight degree.

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* Goodhart and Phillips, *Lancet*, August 5th, 1882.

† Lubelsky, 1870, and others since.

The treatment that has been most frequently beneficial in the chorea of e second half of life is similar to that of the juvenile form.

"ELECTRICAL CHOREA."

The term "electrical chorea" has long been applied to a peculiar malady at is met with chiefly in Lombardy and some adjacent parts of Italy. he malady resembles chorea in being manifested by spasmodic movements, it differs in the character of the movements and in the course of the sease, and in the addition of progressive palsy and muscular wasting. he muscular contractions are sudden and shock-like, and thus resemble ose that are caused by momentary electrical stimulation. The course of e disease is progressive, and a large proportion of the cases terminate tally. It is doubtful whether the disease has any resemblance to chorea in ture, and hence it has been called (by Grocco) "Dubini's disease," from e physician who first described it. It affects both sexes and occurs at all ges. Its causes are unknown, but it has been ascribed to some obscure alarial influence, on account of its occurrence chiefly in certain districts. ven in these, however, it appears to be rare. Cases have been described which the symptoms commenced after a fright, but it is doubtful whether ese are of the same nature. The sudden clonic character of the contracons is not, as we have seen, confined to this disease; it is met with in me cases of ordinary chorea, in the hysterical variety, and in some oreoid attacks in adults and the old.* It is also a characteristic of the ses described as "paramyoclonus multiplex." But all these forms differ om the Italian malady in their benign character, which at present seems e most important diagnostic indication.

The shock-like muscular contractions that constitute the first and most ominent symptom of the disease usually commence gradually in one arm -often in the upper part of the arm—and spread thence, usually to the g on the same side before they invade the opposite limbs. After a few onths or less, the limbs first affected gradually become feeble, the muses waste, with loss of faradaic irritability, and the palsy spreads until it comes general. In many cases there are epileptiform convulsions, which ay be unilateral. In the more acute cases there may be considerable eletion of temperature throughout the course of the disease.

In fatal cases no constant morbid changes are found in the central nervus system, and not only the nature of the disease, but also the precise part the nervous system primarily disturbed, is a matter of conjecture. The most plausible theory assumes that the cerebral cortex is the part primarily diseased (and thus the unilateral commencement and convulsions are accounted for), but that the spinal cord suffers secondarily and causes the changes in muscular nutrition and irritability. It is doubtful whether any treatment influences the disease, but from the rarity of the malady therapeutical experience can accumulate but slowly.

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* The term "electrical chorea" has been applied by some writers to the cases that do not erwise differ from ordinary chorea. But since it has long been used as a designation for Italian malady, this use of the word seems undesirable.

"MYOCLONUS MULTIPLEX."

Clonic spasm is a symptom that occurs in various forms and distribution, and constitutes part of the manifestation of many morbid states of the nervous system. The conditions of which such spasm is a symptom are thus numerous, and, moreover, they are linked together by intermediate forms which render very difficult the separation into definite types of the cases that are described from time to time. A few years ago Friedreich* described, under the name "paramyoclonus multiplex," † a case of clonic spasm affecting the limbs of an adult, and a few cases of more or less similar character have been since described by others.‡ There has been much discussion as to what should or should not be included under the name proposed by Friedreich, and what is the precise relation of the disease to other allied and better-known maladies. Friedreich's patient was a man of fifty, suffering from phthisis, in whom the symptoms commenced a fortnight after a fright, which caused a sensation of stiffness over the whole body. They consisted in clonic contractions in the upper arm muscles and supinator longus, and in the muscles of the thigh, especially the quadriceps, the limbs of both sides being similarly affected. The frequency of the contractions varied from ten to fifty a minute; they were lessened by voluntary movement, ceased sometimes for a quarter of an hour or so, and were always absent during sleep. The only other symptom was an increase of reflex action, and especially of the myotatic irritability. Some years later the patient came under the care of Schultze, who quickly removed the spasms by a few applications of voltaic electricity, but they afterward returned and continued until the man's death from disease of the lungs and kidneys. No marked change could be discovered in the muscles or spinal cord.

The cases that have since been described as examples of this affection have differed considerably in their features. Most of the patients were males, and they varied in age from ten to fifty-two. In most of them the muscles of the back were involved as well as the limbs, in some the muscles of the neck, and in some the face, the abdomen, or diaphragm.* In no case, however, have the extremities of the limbs been affected. In some instances the clonic contractions have been occasionally varied by tonic spasm. Voluntary movement lessened the spasm in some cases (as in Friedreich's), but made it worse in others. In one case the movements continued during sleep. In some of them the clonic spasm, instead of being constant, occurred only in paroxysms, sometimes of extreme violence. Thus, in a case recorded by Allen Starr, the movements of the body occasioned by the violence of the spasmodic contractions, alternately in the muscles of the front and back of the trunk, were so violent that "the head was thrown about and the body was tossed about in the chair," and if the attack came on when the patient was walking he was sometimes thrown down. A similar case of paroxysmal clonic spasm was recorded by Pritchard in 1822, as "Convulsive Tremor," and several others, differing, however, considerably among themselves, have been described by Hammond under the same generic designation. On the other hand, in some recorded cases the spasm has been trifling in degree, even insufficient to cause any movement of the parts, and only to be seen when the skin was bared. It is thus clear that the cases present very wide differences, and the only common characters are the sudden shock-like character of the muscular contractions, their bilateral symmetry, and the freedom of the extremities. In all cases the symptoms have run a benign course; in most of the cases they have passed away, and even those of most prolonged duration have been remarkably amenable to electrical treatment.

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* Friedreich, *Virchow's Archiv*, Bd. lxxxvi, p. 421 (see also Schultze, *Neur. Centralbl.*, *1886*).

† The prefix "para" has been since omitted by Reinecke, and seems to be needless.

‡ Seeligmuller, *Deut. med. Wochenschr.*, *1886;* Remak, *Arch. f. Psych.*, xv; Marie, *Prog. Méd. 1886;* Homen, *Arch. de Neurologie*, *1887;* Allen Starr, *Am. Journal of Nervous and Mental Diseases*, July, 1887.

The condition is regarded by several writers (Schultze, Marie, etc.) as closely allied to facial spasm; by others as related to the form of chorea in which the spasms are clonic. The malady is perhaps intermediate in character between chorea and facial spasm or torticollis, and more nearly allied to senile chorea than to any other malady. We have seen that senile chorea sometimes runs a favorable course, and that, even when severe, it seldom causes death. It is doubtful whether the bilateral symmetry of the affection affords any real ground for placing it far from other diseases that it resembles. One curious case has come under my notice in which paroxysms of clonic spasm, somewhat resembling those of myoclonus multiplex, had an irregular distribution.* The most suitable treatment of the condition appears to be by nervine tonics, with bromide as a sedative, and the use of voltaic electricity, which has in some way produced a striking result in many cases. A current as strong as the patient can conveniently bear should be passed from the spine to the affected muscles daily, for a quarter of an hour. If this fails and the spasm is severe, the hypodermic injection of morphia may be tried.

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* The patient was a healthy-looking man, aged thirty-nine, who presented the following condition. His head was in constant movement, jerked to the right, and constantly rotated, as in torticollis, while coincident spasm involved the right shoulder muscles. Slight tonic contraction of the muscles of the face caused the eyes to be partially closed, the eyebrows raised, and the angles of the mouth to be drawn down, and gave to the face an expression of suffering. The least excitement increased the spasm. If he was spoken to, for instance, the clonic spasm extended to the muscles of the forearm and to those of mastication and of the face, changing the expression of pain to one of anguish. When he himself attempted to speak, the resulting disturbance almost baffles description. The jerkings of the arms became most violent, and the head was rotated from side to side with extreme rapidity for a few seconds. A similar paroxysm occurred on rising from the recumbent posture in the presence of

SALTATORIC SPASM.

The term "saltatoric spasm" is applied to a rare form of clonic spasm in the legs, which comes on when the patient attempts to stand, and causes springing or jumping movements, whence the name.* It was first described by Bamberger, in 1859, and only a few cases have been recorded.† The affection occurs in both sexes, and seems to be rather more frequent in males than in females. The ages of the sufferers have varied between ten and seventy years. In some there has been a history of previous functional nerve disturbance, epilepsy, hysteria, etc., and in most the onset has been preceded by influences, physical or other, depressing the nervous system. Immediate exciting causes have not, as a rule, been traceable. In a few there have been premonitory symptoms, slight tremor, stiffness, or a feeling of stiffness, in the legs. In one instance the spasm followed a convulsive seizure. The actual onset has generally been sudden. The symptoms consist of alternating contractions in the flexors and extensors of the legs, following each other with great rapidity, and causing jumping movements of such violence that the feet may leave the floor at each spasm, and the patient is thrown to the ground unless supported. In some cases the spasm has been slighter, and only the heels have been raised from the floor. The spring is the result of spasm, not only in the calf muscles, but also in those of the hip and knee joints, while in many instances the muscles of the back have also contracted and have caused a backward inclination of the trunk. This peculiar spasm occurs only on an attempt to stand; as the patient sits or lies down the mus-

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a stranger; he would stoop for a moment, bend the head as if in expectancy, and then the violent movement occurred. Over the slighter movements he had some control, so that by a great effort he could open the eyes; the eyeballs were usually directed to the right, but he could slowly turn them in any direction. So violent were the paroxysms on an attempt to speak, that for weeks he never uttered a word. He could, however, write fairly well, with occasional interruptions from the spasm. The man's history showed no cause, immediate or remote, for the affection, which had commenced gradually six months before. Under treatment, rest and hypodermic injections of morphia, he gradually improved, and became able to speak to the other patients in the ward, although it was much longer before he became able to speak to a stranger. Ultimately the spasm became trifling, but it had not quite disappeared when he passed from observation.

* It has been also called "static reflex spasm" by Erlenmeyer.

† Bamberger, *Wien. med. Wochenschr., 1859;* Guttmann, *Berlin. med. Wochenschr., 1867;* and *Arch. für Psych.*, Bd. v, 1876; Frey, *ib.*, Bd. vi, 1875; Gowers, *Lancet*, ii, 1877; Kollmann, *Deut. med. Wochenschr., 1883*, No. 40, and 1884, No. 4; Erlenmeyer, *Cent. f. Nervenkrank*, *1887*.

cular contractions cease, often entirely, although sometimes a little clonic spasm or rigidity has persisted for a short time. In some cases, pressure on the feet, as the patient lay, caused slight spasm, similar to that produced by an attempt to stand. In no case did the spasm, excited by standing, spread to the arms, but in some instances other voluntary movements, even in the recumbent posture, caused a little clonic spasm, which involved also the arms. Motor power has been normal in most cases, but in some there was trifling weakness after the spasm had existed for some time. Sensation was unaffected in all, but dull pain in the legs, or tenderness of the spine, were present in some of the patients. In one of Bamberger's cases there were other peculiar symptoms—palpitation, dyspnœa, inequality of pupil, with spasm on one side of the face. In Kollmann's patient other severe disturbances of the nervous system led to death.

The affection has generally continued for some months; in only two was its duration less than one month, while in one case it continued till the death of the patient, six years after the onset of the symptoms. The cessation of the spasm has, in most cases, been gradual.

The spasm is evidently reflex in character, and has been regarded by most writers on the subject as proceeding from the spinal cord, and the result of a peculiar exalted irritability of the spinal reflex centres, especially of those that subserve the coördinated movement of jumping, etc. Freusberg* has shown that alternating contractions in the flexors and extensors of the legs may be coördinated in the lumbar enlargement of animals, and I have found evidence of a similar fact in man: in a case of paraplegia, with absolute motor palsy, and evidence of a transverse lesion in the dorsal region, pressure on the soles set up alternate flexion and extension of the hip-, knee-, and ankle joints, by which the feet were successively drawn up and pushed down.† Apparently the area of increased irritability varies in different cases, since in a few, apart from standing, a peripheral impression has caused movements that spread widely.

The disease in most cases has not appeared to be rapidly influenced by treatment. The spasm has continued, in spite of the administration of sedatives; the most important and effective measure appears to be the improvement of the general health, and of the strength of the nervous system, by tonics. The case of shortest duration is one recorded by myself, in which slight but characteristic symptoms (another case having previously been under my observation) were at once cut short, on the second day of their existence, by a copious diaphoresis in a hot-air bath.

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* *Pflüger's Archiv*, Bd. ix.

† It is to be noted, however, that Woroschiloff (Ludwig's *Arbeiten*, *1874*, p. 110) obtained evidence of a centre in the upper part of the spinal cord of the rabbit, by which springing movements of the legs were apparently coördinated. A discussion of some other experimental facts bearing on the subject will be found in my paper on the disease (*Lancet*, *1877*).

HABIT SPASM.

Children often, and adults sometimes, present spasmodic movements, such as winking, twitching the mouth, jerking the head, movements that have a half-voluntary aspect, but which the individuals are unable to control. The patient is said to have "got a trick" of moving the part. This condition has been termed "habit chorea" by Weir Mitchell, but the term "chorea" it not strictly applicable to it, and "habit spasm" is, I think, a better name.

The condition is met with chiefly in childhood, especially in the second half, but it sometimes commences in youth, and even in adult life. In young women, it is often associated with symptoms of hysteria, and there may be a difficulty in deciding whether certain spasmodic movements are to be regarded as examples of this affection or of hysterical spasm. When it commences in childhood, the affection commonly ceases after a few months or years, but it occasionally goes on to middle life or even longer. Rarely it begins late in life, and is then generally permanent. In early life, it occurs especially in nervous and excitable children. The affection is said to be more common in females than in males, but it is very often seen in boys. It usually begins between the ages of six and fourteen; I have, however, known it to commence as early as four. Some impairment of general health often precedes the development of the movements; occasionally they appear to be due to some special influence depressing the nervous system, overwork at school, a fright, or some injury. In one case, for instance, the onset followed a fall into the water. They may succeed true chorea, or some local irritation in the part that is the seat of the movements; conjunctivitis, causing blepharospasm, may set up winking movements. In boys the affection is often due to masturbation, and from this cause very severe cases are sometimes met with.

Frequently there is a history of other neuroses in parents or other relatives. More than one child in the same family may suffer, but they seldom present quite the same movements. I have seen, for instance, a different habit spasm in twin sisters. It is probable that the affection often arises by imitation. There is not often, perhaps, direct imitation, but the witnessing such movements is apt to produce a peculiar excitability, which finds expression and relief in movements of a similar nature. In cases in which something like direct inheritance can be traced, it is probable that this influence has been at work. A father, for instance, had such movements in the face all through his life, and two of his children likewise presented them. In many cases, however, no causes can be traced, and the affection seems to be the result of the restlessness of childhood, specialized, as it were, in a particular direction.

The movements usually occur at intervals of a few minutes, but sometimes they are almost continuous. Their character varies much in different cases, and even in the same individual. One kind of action, after lasting for a time, may give place to another, or two or three kinds may alternate at the same period. The most common is blinking of the eyelids, by a sudden contraction of the orbicularis palpebrarum, sometimes accompanied with depression of the eyebrows. Another that is very common is a contraction of the zygomatic muscles, moving the angles of the mouth, first to one side and then to the other. A movement of the head is also common, a lateral deviation, a rotation, or a nod. In one case there was a backward movement of the head with simultaneous elevation of the eyebrows, as in looking up. In another case spasm at the back of the neck was succeeded by a contraction in the sterno-mastoid. Movement of the arms is also common, shrugging the shoulders, or a peculiar fidgety action of the hand, or some other movement of the arm. One boy would put both his arms forward and then stoop, and he did this under all circumstances, even in church. A movement of the leg is less common; the patient last mentioned afterward got into the way of kicking his leg forward in walking, doing so about every dozen steps. Of the twins mentioned above, in one, in whom the spasms began after a fall at three, there was a slight movement of the leg and a twist of the arm, while in the other a slight movement of the head was accompanied by a peculiar action in walking, a half turn, as if she were looking for something that she had dropped.

Respiratory spasm is also very common; there may be a sudden inspiration, sometimes accompanied by a laryngeal sound, or there may be a sudden audible sniff, or a kind of sob. These may be combined with some other action. Thus in a boy of eleven, a quick movement of the mouth alternated with closure of the eyes, or with a sudden inspiration that was attended by a slight nasal snore. A peculiar cough is also common in these cases; often it has a laryngeal character, and it may be extremely troublesome. It is occasionally first excited by some laryngeal catarrh and actual cough. One boy began by making a noise in his throat every few minutes, half cry, half cough, so loud that it could be heard outside the house; he said that it was occasioned by a sense of difficulty in breathing. It continued for two years, and then ceased, but soon afterward twitching commenced in the left side of the mouth, and gradually extended to the whole of the left half of the body, and this continued, varying in degree, for a year. Occasionally these laryngeal sounds are repeated several times in succession. In a case recorded by Blachez the noise made was a piercing cry, compared to the squeak of the child's toy, but much louder.

These habit movements, as we have already seen, usually cease after a time, but they sometimes persist, and become a source of great annoyance to a patient who has reached adult life. The inability to control them by an effort of the will becomes very marked in such cases. In one instance a girl, who began to blink the eyes in early childhood, still did so at nineteen. In another case, a clergyman of thirty-seven was greatly annoyed by an involuntary smile, of somewhat meaningless aspect, which would cross his face from time to time, without the slightest corresponding emotion, and even when he was engaged in reading in public the most solemn parts of the Church service. It never troubled him when he was preaching or in conversation, but it often occurred when he was looking at another person, and sometimes gave rise to misconception. It commenced at the age of sixteen, and was at first more than a smile, being actual laughter, but it gradually subsided into its permanent form. In another case a lad of eight began to suffer from twitchings, now in one arm, now in the other, two or three quick movements at a time. Such movements persisted, changing their form, until he came under observation at twenty-four, when he presented frequent movements of the face, elevation of the eyebrows, twitching of eyes, etc., and occasionally a sudden up and down movement of the jaw. He could prevent them "when he thought of it," but when his mind was otherwise occupied the spasm increased. Again, a young man, aged twenty-two, presented frequent winking of the eyes and movement of the mouth, which had existed for ten years, and had succeeded a peculiar condition that began at ten years old, in which, besides twitching movements of the hands, he had an irresistible impulse to repeat actions; if he had touched an object he felt obliged to touch it again.

These habit spasms are generally increased by observation, and it is very important that little notice should be taken of them by the friends of the patient. Sometimes the movements will then cease without further treatment. They are seldom under direct voluntary control, and the endeavor to prevent their occurrence may be futile, especially if the attempt is made under the influence of fear of threatened punishment. But the promise of a reward at the close of each day on which the spasm has not occurred will sometimes gradually cause their disappearance; a strong desire, free from any depressing emotion, effects that which the will cannot directly achieve. Any obvious defect in the general health must be made good, and change of air is often very beneficial, especially when a change in companionship can be secured at the same time. The deterring influence of strangers is often very marked. Of drugs, arsenic has certainly most influence. Weir Mitchell has known the hypodermic injection of arsenic to succeed, when other things failed. Nerve tonics, such as quinine and strychnia, may with advantage may be added. If there is much excitability of brain, or if the spasmodic movements are severe, bromide of potassium may be needed, and it is especially useful for the spasmodic cough. Occasionally a local blister is of service, and gymnastics may be employed when the spasm is in the limbs. The smiling clergyman mentioned above ceased to be troubled after he had, for a few weeks, taken some arsenic and iodide of iron, and a dose of bromide each time he had to conduct the service in church.

PARALYSIS AGITANS.

Paralysis Agitans, or Shaking Palsy, is a disease of the second half of life, characterized by the symptoms indicated by its name, muscular weakness and tremor, and also by muscular rigidity. The symptoms usually commence locally and gradually, but tend to spread and to become general. From the fact that it was first fully described by Parkinson in 1817, it has been called "Parkinson's disease," but the name which he gave to it, of "shaking palsy," is both apt and adequate.*

Causes.—The disease is more frequent in men than in women, the proportion being as five to three. Of eighty cases of which I have notes, fifty were men and thirty women. It usually commences after forty years of age. Nearly half the cases begin between fifty and sixty, and about one-fifth in each of the two decades, forty to fifty and sixty to seventy, but, on account of the lessened number of persons living, it is probably twice as frequent in the latter as in the former decade. It occasionally begins between thirty and forty, very rarely under thirty.† Cases have been recorded in which the disease began still earlier, as at twenty-one (Buzzard), nineteen (Duchenne), and seventeen (Berger). Over sixty-five it is rare; my series includes one case beginning at sixty-seven, and two at sixty-nine, but no later one. It is thus essentially a disease of the degenerative period of life, but not of extreme senility. Little variation exists between the sexes in their relative liability at different ages. I have found the average age at commencement to be the same in each sex, fifty-two years.

Heredity can seldom be traced, perhaps in not more than fifteen per cent. This is the proportion in my cases and also in a series of cases observed by Berger.‡ Although the proportion seems small,§ yet in some instances a strong history of allied affections shows that hereditary influence may be powerful. The sister of one patient, and the mother of another, suffered from paralysis agitans, and the brother of a third was said to have suffered all his life from a peculiar non-progressive form of tremor. In other cases there has been a history of insanity or epilepsy in near relatives. The disease has been thought to be more frequent in the "laboring" classes, but the influence of station in life, and also of occupation, has been certainly exaggerated by some writers.

Exciting causes cannot be traced in more than one-third of the cases, and vary much in character. The most frequent are emotion, physical injury, and acute disease. Prolonged anxiety and severe emotional shock often precede the onset. Sudden alarm may cause general tremor, so that the verb "to tremble" is in process of conversion into a synonym with "to fear." Usually the tremor subsides when the alarm is over, but it has been known to persist and develop into this disease. It is noteworthy, moreover, that the direction of alarm may localize the commencement of the affection. A remarkable example of this was presented by a woman, who, at thirty-seven years of age, was sitting quietly at work, when a stream of water suddenly flowed from a tap on to her left wrist. She was much startled; the left arm immediately began to shake, and the tremor persisted, passing to the leg and afterward to the limbs on the opposite side. When I saw her, a year later, she presented all the characters of the disease in its typical form. Physical injury is occasionally a distinct excitant, but it must be remembered that this usually involves also emotional shock. That the physical injury is not merely coincident is shown by the fact that the tremor usually commences in the part injured. Thus, in two of my cases the exciting cause was a fall on the shoulder, and in each the tremor commenced in the arm injured.* A contusion of the thigh has been followed by tremor in the limb, ultimately becoming general (Charcot). The disease has also followed an injury to the radial nerve (San Martin). Injury may determine the spread of tremor which has already commenced. Thus in one case, recorded by Charcot, dislocation of the jaw, immediately reduced, was followed by tremor in it, which persisted. Traumatic influences sometimes seem to be effective by the concussion of the nerve centres. In one of the most severe cases I have seen, which commenced at forty, the only traceable cause was a fall from a horse four months before the onset; there was no injury to limb. I have seen one other similar case. Muscular exertion seems to have far less influence than might be anticipated from the effect of prolonged and unaccustomed effort in causing tremor. It seemed to determine the onset in one case in which emotion probably coöperated; a woman was much shocked at a neighbor being killed in a railway accident; she went to the funeral, carrying a heavy child on the left arm; the arm felt very tired afterward, and the feeling of fatigue persisted and gradually changed to one of stiffness, which proved to be the local commencement of paralysis agitans. Exposure to cold was the apparent cause in one or two recorded instances. Of acute diseases, I have known dysentery and typhoid fever to precede the onset. Malaria is commonly regarded as one of the causes of the disease, but on evidence which is not conclusive. Paralysis agitans is more frequent in England, where ague is almost unknown, than in most malarious countries. I have seen one case in which the disease followed repeated attacks of remittent fever, but the patient was at the same time (during the American war) exposed to great privation and fatigue. Sexual excess is perhaps a rare cause in comparatively young persons (Buzzard). Toxic influences which cause general tremor (alcohol, lead, brass-working, etc.) seem to have little if any influence in producing true paralysis agitans.

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* To the name "paralysis agitans," it has been objected by Charcot that either the weakness or tremor is sometimes slight and occurs late; but this fact does not lessen the general applicability of the name, since in the majority of the cases both symptoms are conspicuous.

† Of my cases, one-ninth began between 30 and 40, and only one between 20 and 30, and that was at 29.

‡ Eulenburg's "Real Encyclopædia."

§ In senile maladies the facts that can be ascertained regarding heredity probably fall short of the truth to a greater extent than in the maladies of earlier life, because, as life goes on, the death of older relations lessens the opportunities of ascertaining the facts. It is often astonishing how much disease inquiry sometimes reveals in the families of those who imagine, before the inquiry is made, that they are absolutely free from all morbid heredity, so carefully have unpleasant facts been concealed by those to whom they are known.

SYMPTOMS.—In a well-marked case of this disease, such as is shown in Fig. 316, the aspect of the patient is very characteristic. The head is bent forward, and the expression of the face is anxious and fixed, unchanged by any play of emotion. The arms are slightly flexed at all joints, from muscular rigidity, and (the hands especially) are in constant rhythmical movement, which continues when the limbs are at rest, so far as the will is concerned. The tremor is usually more marked on one side than on the other. Voluntary movements are performed slowly and with little power. The patient often walks with short, quick steps, leaning forward as if about to run.

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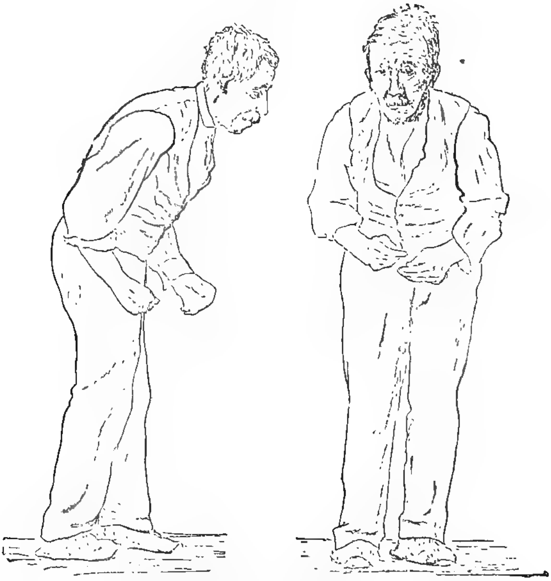
* In one curious case the arm in which the symptoms commenced had been the seat of slight tremor since a burn of the palm in youth.

A prodromal stage characterized by rheumatoid and neuralgic pains is met with or precedes the onset in rare cases. Usually the initial symptoms come on very gradually, as tremor, stiffness or weakness, in one hand. The tremor may at first only occur on emotion or fatigue, but is afterward constant, and slowly spreads from the part in which it was first noticed.

Fig. 316.



Paralysis Agitans. (*After St. Leger.*)

In the majority of cases (two-thirds) the tremor precedes the weakness; occasionally this relation is reversed. Of sixty-three cases in which the mode of onset was noted, it was by tremor alone in forty-six, and by weakness, with or without a sense of "stiffness," in eleven, while both seemed to come on together in six. The commencement is four times as frequently in the arm as in the legs, very seldom in both (arm sixty-one, leg nine, both five cases), and more frequently in the left arm than in the right (left thirty-five, right twenty-six).*

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* In my cases the affection began in the left leg more frequently than in the right, but the numbers are too small to justify the inference that this is the rule.

In the arm the tremor usually commences in the hand, sometimes in the forefinger and thumb, but I have met with four otherwise typical cases in which it began in the shoulder. In two of these the cause was a fall on the shoulder. From the part first affected the tremor slowly spreads, and the usual mode of extension is from the arm to the leg on the same side, next to the opposite arm, and lastly, to the opposite leg. The disease is thus hemiplegic in its progress. Much less commonly the opposite arm is affected before the leg on the same side.* I have known the symptoms to extend to the leg on the same side and thence to the opposite leg, and it has been known to pass from the arm on one side to the leg on the other. The extension from arm to leg on each side is by far the most common course.

When the disease begins in the leg, the march of the tremor is less uniform. In two the extension was from the leg to the arm on the same side and then to the opposite arm. In two cases it was to the other leg, then to the arm on the side first affected, and, lastly, to the other arm. In one case, in which the tremor passed from the leg to the arm on the same side, it commenced at the shoulder and continued greatest in the upper part of the arm, a point of interest in connection with the fact that not uncommonly in hemiplegia, when the leg suffers more than the arm, the upper part of the arm is paralyzed in greater degree than the hand. Often, when the affection begins in the leg and passes to the arm, it speedily attains a much greater degree in the latter than in the former.

The weakness and fixation of limb usually succeeds the tremor, and is greatest where the tremor has existed longest and is most marked. In exceptional cases, however, the loss of power and rigidity precede the tremor at the onset, and still more frequently are marked in parts to which the tremor has not yet extended. Thus, in one case in which the tremor, beginning in the left arm, passed to the left leg and then to the right leg and was absent in the right arm, the latter was much weaker than the leg. On the other hand, there is sometimes little weakness although there is much tremor. The several symptoms may now be considered in greater detail.

The *tremor* is an alternating contraction in opposing muscles, causing a rhythmical movement of the parts to which they are attached. It is usually greatest in the hands and fingers, partly from the contraction of the forearm muscles, partly from that in the interossei; the latter causes a movement of the fingers at the metacarpo-phalangeal joints similar to that by which Orientals beat their small drums. This movement may be chiefly in the thumb and forefinger, which may move as in the act of rolling a small object between their tips. Not very rarely the movement is chiefly at the wrist, lateral or antero-posterior, or there is distinct pronation and supination. The muscles of the upper arm are usually less affected and those of the shoulder still less. In exceptional cases, as already mentioned, the upper part of the arm is most affected and the tremor is less in the forearm and still less in the hand. In one such case the chief contractions were in the pectoralis, deltoid, and teres major, slighter in the triceps and biceps, and very slight in the forearm muscles.* In the legs the tremor is usually greatest in the muscles moving the ankle joint, and the heel may beat the floor as the patient is sitting; it is slight in the toes, but may be distinct in the thigh, sometimes in the adductors, sometimes in the flexors of the knee. The trunk muscles, especially those of the back, are occasionally involved. I have never met with tremor in the muscles of the abdomen, even when the lumbar muscles were distinctly affected. Usually the head is free from tremor except such as may be communicated to it from the distant oscillation. It does not, however, always escape, as some have asserted. In perfectly characteristic cases of paralysis agitans the head may present distinct tremor in consequence of contractions in the trapezius, splenius, and even in the sterno-mastoids—contractions which may be distinctly felt. I have once met with a slight rotatory tremor of the head due to the deeper muscles.† The masseters are occasionally affected, causing a movement of the jaw, which may amount to one-sixth of an inch; the contractions may be greater on the side on which the limbs are most involved. The jaw is usually affected late—after the limbs, but in one case the affection of the jaw came on after the limbs on one side were involved and before the affection spread to those on the other side. Occasionally the tongue is affected; very rarely the muscles of the face. I have once seen distinct persistent tremor in the orbicularis palpebrarum, and tremor in the lower facial muscles has been noted by Westphal. When there has been tremor of the tongue I have never detected any movement in the soft palate.

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* One such case deviated from the ordinary type, in that the upper arm muscles were more affected than those of the forearm, and the intrinsic muscles of the hands were free from spasm.

The movement varies much in its range. It may be so slight as to need close observation to detect it, or may amount to two inches at the extremity of the fingers. It is always slight at the commencement and increases with the progress of the disease.

The time of the movement (according to many tracings that I have taken) varies from about 4.8 to 7 complete oscillations per second (see Fig. 317). It lessens in frequency as it increases in range; the fine tremor of the early stage is often distinctly quicker than the coarser tremor of the later period. Thus in an early case, the very fine tremor varied from 6.4 to 7 oscillations per second, whereas in a later case, with a range of movement of the hand from one to two inches, the frequency was from 5 to 5.4 per second. The tremor in the leg has nearly the same rate as that in the arm in the same case. In one patient the frequency in the arm was 6 per second and that in the leg was 6.8. The degree of movement is sometimes very uniform, in others it is somewhat irregular, but it never approaches the irregularity of some other forms of tremor. I have only once observed a tendency to a rhythmical variation.

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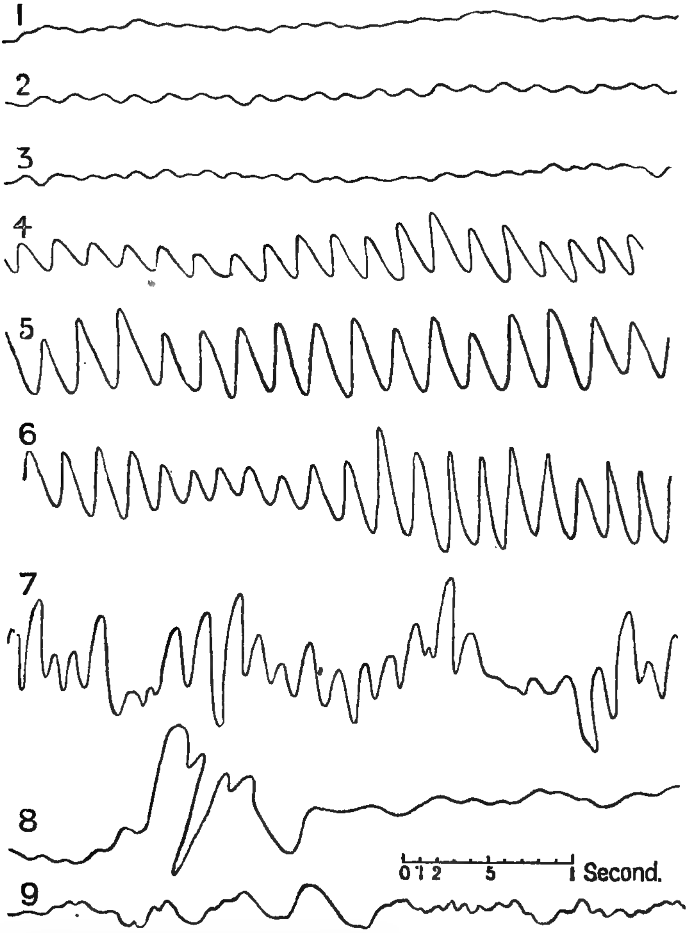
* I may remark that in all the exceptional cases mentioned in the text, the diagnosis of the disease was certain; all doubtful cases have been excluded.

† Slight but distinct tremor can indeed be felt on the neck muscles not at all rarely. Since I have carefully searched for it, I have found it in no less than eight out of thirty-seven cases.

The great characteristic of the tremor of paralysis agitans is, as Parkinson pointed out, that it continues during rest. The hands go on moving when they are resting on the patient's knee, and the legs when he is sitting. A voluntary movement may stop the tremor for a few seconds, sometimes for

FIG. 317.



Myographic tracings of various forms of tremor, taken directly from the moving parts. Reduced to one half.*
1. Tremor after hemiplegia; very fine, rather irregular.
2–6. Paralysis agitans; 2 and 3, very fine tremor; 3, 4, 5, coarse tremor.
7. Insular sclerosis; very irregular in range, although regular in time.
8. General paralysis of the insane.
9. Hysterical tremor.

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[*] These tracings are fairly typical of a large number I have taken of various forms of tremor.

many, but it recommences and accompanies the movement. Hence, the patient's handwriting reveals his disease; the letters may be fairly formed, but every line is a zigzag. In slight cases the irregularity may be so fine as to need a magnifying glass to recognize it. By an effort the patient can often stop the shaking for a moment, but it then recurs with augmented violence. Although it is the rule for the tremor to continue during rest, the rule is not invariable; exceptions are occasionally met with. In the early stage of the disease, prolonged rest frequently lessens the tremor considerably, and this in cases in which, at a later stage, such rest is without influence. Moreover, in a very early stage of the disease, the tremor may be distinct and even considerable on voluntary movement, and may almost or quite cease as soon as the limbs are at rest.

Lastly, rare cases are met with in which the disease has evidently existed for some time, and yet tremor occurs on movement only. This is especially the case when the fixation of limb preponderates over the tremor. For instance, a woman, aged sixty, had gradually, during four years, passed into a condition in which the features had become expressionless; there were the characteristic posture and slowness of movement to be presently described, considerable weakness of limb, and great sense of heat. During rest, there was no conspicuous shaking, and only the finest possible tremor could be discerned in the right, the weaker, hand. But every voluntary movement was attended by regular, coarse, typical tremor, and this not only in the limbs, but in the masseters, face and tongue. In another case, with constant tremor in the fingers, tremor at the elbow joint occurred only on movement. I have known the tremor in one hand to be constant, and in the other, last and least affected, to occur only on movement. Rarely fine tremor during rest becomes coarser during movement. It must, therefore, be remembered that the persistence of the tremor during rest, although usual, is not universal, and its limitation to or increase by voluntary movement does not disprove the indication of other symptoms that the case is one of paralysis agitans.

If the tremor is unilateral, and the shaking of the hand is prevented by force, the other hand, previously free, usually begins to shake. The same result may follow the arrest of the movement by an effort of the will, if this can be effected. Sleep usually brings stillness to the shaking limbs, and this although the tremor is considerable. Now and then, however, some tremor persists during sleep. I have known the tremor to persist, during sleep, in the arm, and to cease in the leg. In rare cases there are paroxysmal exacerbations of the tremor, excited especially by emotion.

Muscular weakness and rigidity usually come on together, and are as characteristic of the disease as is the tremor. The loss of power varies much in degree. At first slight, it gradually increases, and is usually greatest in the part in which the tremor developed first and most. The patient may ultimately be unable even to move the index of the dynamometer, or to rise from his seat. But the paralysis is never absolute—some power always persists. Voluntary movement is not only feeble; it is also slow. It may be slow in execution, or there may be a delay in the commencement of movements that are performed with fair rapidity. This seems to be, in part at least, the result of muscular rigidity, which causes a resistance to passive movement. Another effect of the rigidity is to impress certain characteristic postures on the limbs. These are determined by the fact that the rigidity preponderates in certain muscles, chiefly in the flexors. The arms are flexed at the elbow joints, sometimes slightly, sometimes almost at a right angle. The wrists are usually slightly extended. The position of the fingers varies; in some cases they are slightly flexed at all joints, in the position that they naturally assume during rest; more often they are flexed at the metacarpo-phalangeal joints and extended at the others, from preponderant contraction in the interossei.* There may even be over-extension of the last phalanx, most marked in the thumb, perhaps because of the pressure of the tip of the thumb against the first finger. Usually the rigidity can be readily overcome, but in extreme cases (as shown in Figs. 318 and 319) the contracture of the interossei may go on to the unusual degree of permanent shortening, so that the metacarpo-phalangeal joints cannot be passively extended beyond a right angle, just as in contraction of the palmar fascia.† Occasionally only one finger (as the index) is thus affected. In the legs the rigidity involves chiefly the hip- and knee-joints, causing slight flexion of each, and adduction of the

FIG. 318.

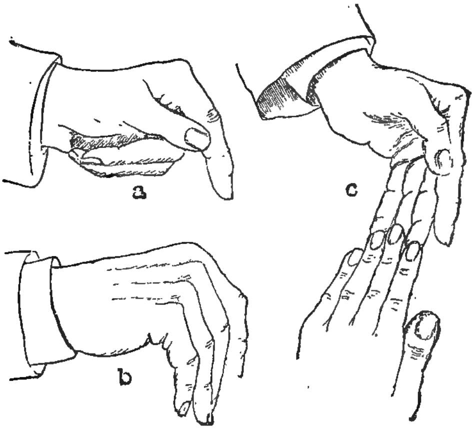

FIG. 319.

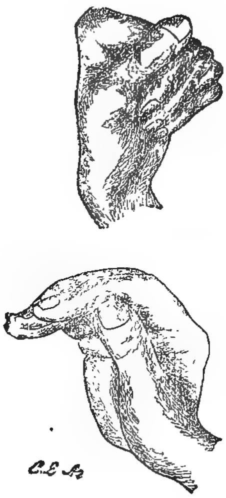

Fig. 318.—Paralysis agitans. Posture of hands from contracture of the interossei: *a*, left hand; *b*, right hand. In the left hand the contracture is greater than in the other, and has led to some permanent shortening of the interossei, so that the fingers cannot be extended even passively. The maximum passive extension is shown at *c*.

Fig. 319.—Contraction of fingers in paralysis agitans. Maximum voluntary extension.

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* In rare cases the digits deviate toward the ulnar side, as in chronic rheumatoid arthritis.

† The contraction in these cases is not of the palmar fascia. Mr. W. Adams was good enough carefully to examine one case for me, and fully confirmed this statement.

thighs. It may extend to the feet, and even cause talipes equino-varus, and distortion of the toes—extension of the first, and flexion of the other phalanges so as to cause a claw-like deformity. Permanent contraction of these muscles is very rare, but I have known each foot to be fixed in inversion.

The head is usually carried forward, and the upper part of the spine is bent in the same direction (Fig. 316). The facial muscles seem to be unable to respond to the varying changes of emotion; the expression of face is a fixed, anxious look, unvaried by smile or frown. In very rare cases, which must be regarded as paralysis agitans, the head is bent strongly backward. In one such case the affection commenced in the head, but I have seen a similar case in which the arms first suffered. There is sometimes a difficulty in protruding the tongue.

In consequence of this muscular condition, certain complex movements are performed in a peculiar and characteristic manner. One of these is walking. The patient rises up slowly from the chair with head and shoulders bent forward. His steps are short, and the first may be taken slowly and with difficulty, but they become quicker and quicker, until the patient seems to be about to run ("festination"), and often, from the inclination of the body, to be on the point of falling forward ("propulsion"). It has been thought that this tendency to run is due simply to the forward inclination of the body; the patient, as Trousseau expressed it, has to run after his centre of gravity, but that this is not the whole explanation is shown by the fact that some patients, suddenly jerked backward, may exhibit a tendency to walk backward, which they are unable to avoid ("retropulsion"), and this although they stoop forward. It is indeed not uncommon for patients to be able to walk backward better than forward. In one case an irresistible tendency to run backward existed only on first rising in the morning. Usually the patients' equilibrium is easily disturbed, and even when they walk fairly well, they have considerable difficulty in turning suddenly. One patient, in turning, often took a few unintended steps to one side.

The slowness of muscular action is usually conspicuous in all movements, but it rarely affects the muscles of the eyes. If the patient is to look in a given direction, the eyes are instantly turned, while the head slowly follows them.* Speech has usually a well-marked characteristic: the voice is monotonous, being no more varied by emotional expression than is the face. There is a delay in commencing a sentence, but, once commenced, the words are uttered rapidly, often with some confluence of syllables; there is "festination" in speech analogous to that in walking; a condition the opposite of the separation of syllables met with in disseminated sclerosis. It is as if the patient tried to speak with a minimum of exertion, and to get his utterance over as soon as possible.

As already stated, although the weakness and rigidity are usually secondary to the tremor, they may come first. We have seen that they precede marked tremor in one-fourth of the cases. It is not at all rare for them to precede the tremor in parts secondarily affected. Thus a patient may present the ordinary tremor and weakness in one hand, and there may be no tremor in the other, which may yet be weak and present the characteristic interosseal position and slowness of movement. When the rigidity and weakness constitute the first symptom of the disease, they sometimes become general before the tremor is conspicuous. Often, however, in such cases slight shaking may be perceived if it is carefully looked for. On the other hand, there is sometimes very little loss of power, although the tremor is great. In one man, for instance, with severe shaking in the left arm, slight in the left leg, and scarcely any in the right arm, the grasp of the right hand was sixty-two kilogrammes, and that of the left was fifty six.

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* Debove, however, has noted a retardation in the movement of the eyes which, if the patient was reading, occasioned a characteristic pause at the end of each line, but this is extremely rare.

The myotatic irritability in the limbs is usually normal, the knee jerk is not increased, and although the foot, when resting on the ground, may be jerked by clonic spasm in the gastrocnemius, just as it is in the foot clonus, no clonus can be obtained by passive flexion of the ankle. In exceptional cases, however, there is distinct excess in these contractions, generally limited to an increase of the knee jerk; in very rare instances a typical foot clonus can be obtained. The superficial reflex action is also, as a rule, normal. The sphincters are rarely affected; occasionally, in the advanced stage, the power of retaining the urine is weakened, but I have never met with actual incontinence.

The nutrition of the muscles does not suffer until late in the disease. The continuous activity might be expected to lead to hypertrophy, and it has been said to occur, but it is doubtful whether there is ever a real increase in bulk. In most cases, as the disease goes on and rigidity sets in, the muscles lessen in size, and there may even be conspicuous wasting. The electric irritability of muscles and nerves may be unchanged, but a slight increase may often be detected when the unilateral character of the disease permits comparison with the other side, and, at a late period, there may be a slight diminution in irritability. I have seen instances of both these changes. The alteration is never considerable, and is always the same to each form of electricity.

Sensory and Vasomotor Symptoms.—Cutaneous sensibility is never affected in paralysis agitans, but subjective sensations are frequent. Aching pains in the limbs, more or less "rheumatic" in character, are occasionally complained of in the early stage, and they may correspond in seat with the commencing tremor. When the movement is considerable, it occasions a great sense of fatigue, which is accompanied, after a time, by extreme restlessness, and every few minutes some slight change of posture is desired. Frequently also (in three-quarters of the cases) there is some abnormal sensation of temperature. The most common is a sense of heat, to which Charcot first directed attention. I have found this to be present in half the cases in which the point was investigated (twenty-two out of forty-seven). When slight, it may only cause the patient to dislike hot rooms or many bedclothes; but when more considerable it is a source of considerable discomfort, and only the thinnest covering can be endured at night, even in the depth of winter. Such sufferers are, as a rule, more comfortable in winter than in summer. The sensation may be referred to the interior of the body, or to the limbs, and sometimes it is localized in the most affected limb, especially in the extremity. One patient, whose right hand alone shook, complained of burning heat in the palm, passing up the radial side of the forearm to the bend of the elbow. Another, with tremor in the left arm, never would allow this arm to be under the bedclothes. Grasset asserts that this local sense of heat is always the accompaniment of an actual elevation of the peripheral temperature, which is higher than in another individual in the same part; but this needs confirmation; the general temperature of the body, as a rule, is normal.*

The sensation of heat may be absent, when the tremor is violent, and it may precede the movement. Thus in one of my patients the sensation preceded any tremor for four years. In other cases, instead of a feeling of heat, there is an abnormal sensation of cold; the patient always feels chilly. This is only half as common as a sensation of heat (eleven out of forty-seven cases). I have even known sensations of cold and heat to alternate; in one case, as the disease progressed, the occasional sensations of cold gradually yielded to an enduring sense of warmth, but the alternation may persist even until the late stages; at one time the patient feels cold, and at another bursts into perspiration. It is probable that these sensations, and the local elevation of temperature, are due to vasomotor conditions.† The sense of heat is very frequently accompanied by increased perspiration, which may be general and profuse on the least exertion of mind or body. One patient could not dictate the simplest letter, even in winter, without first having his coat taken off, so intense was the perspiration caused. Sometimes sweating is local, corresponding to the sense of heat. In one patient, with left-sided agitation, the left axilla was always wet with perspiration, while the right was always dry. This phenomenon sometimes corresponds with another indication of disturbed function of the sympathetic, a small pupil. In unilateral paralysis agitans I have five times seen the pupil on the affected side smaller than the other, and in one of these cases the patient perspired much on the corresponding side of the head. Frequently both pupils are small, but this may be merely the common senile myosis. They always act to light. Œdema of the legs may be present in the later stages of the disease. The urine is sometimes increased in quantity. According to Chéron it usually contains an excess of phosphates.

The intellect may be unaffected throughout, except by the irritability which usually accompanies the physical restlessness. Often there is mental depression; it may be difficult to say whether this is more than is the natural result of the physical ailment. Pronounced mental symptoms are occasionally present, however, in the later stages of the disease, commonly limited to mental weakness and loss of memory, but sometimes accompanied by a tendency to delusions.

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* I have once found the affected side (in which there was an intense sense of heat) .6° F. warmer than the other.

† It is also possible that the sensation may be due, in part at least, to changes in the sensory centres of the brain, and that the vasomotor disturbance may be an associated effect of these or other central changes.

Varieties.—The aspect of a case of paralysis agitans varies considerably, according to the extent and distribution of the symptoms, which led Marshall Hall to distinguish "hemiplegic" and "paraplegic" forms, and to these a "monoplegic" form, in which only one limb is affected, has been added by Berger. These are not, however, varieties, and should not be described as such; they are merely stages of a disease which tends to become universal. The aspect of the case is also influenced by the kind of tremor, whether fine or coarse. It is influenced, also, by the amount of weakness and fixation of limb, and the relation, in point of time, between this and the tremor, since, as already mentioned, the general aspect, rigidity, position of limb, and muscular weakness, may be conspicuous before the tremor is perceptible. It is this feature that gives rise to the best-marked variety of the disease.

Complications.—An attack of ordinary hemiplegia may occur during the course of paralysis agitans without evidence of any closer connection between the two diseases than is involved in the patient's age. The tremor lessens or disappears during the acute stage of the paralysis, but returns with power of movement, and often becomes much greater than before the palsy, as Parkinson long ago pointed out. It is said that sudden transient hemiplegic weakness may occur from time to time without any lesion being found in the brain to explain it (Berger); but it is doubtful whether such attacks have had any real connection with the disease they accompanied. The mental failure, common in slight degree in the later stages of the disease, sometimes occurs early and impresses a special character on the aspect of the case. I have once met with convulsive attacks resembling ordinary epilepsy, as a complication of paralysis agitans. The patient was a woman, and both symptoms commenced at the same time, at fifty-nine years of age. Buzzard has recorded a case in which there was a semi-cataleptoid condition of the limbs; when raised they remained so for several minutes and then slowly fell. Cramp in the legs and soles of the feet is very troublesome in some cases.

Course.—The disease is always chronic, and usually progressive, in its course. The varieties which it presents in its mode of extension have been already described. The rate of progress is sometimes extremely slow. It may remain for two or three years limited to the limb first attacked. Usually, if commencing in the arm, the leg on the same side is involved within two years. Sometimes the extension to the leg takes place in two or three months, and occasionally the arm and leg are affected at the same time. The date of extension to the opposite side varies from six months to three or four years after the onset. The shortest time in which I have known all four limbs to be affected, in a case beginning locally, was nine months. Not unfrequently the tremor is limited to one arm for a considerable time and then rapidly spreads. For instance, in one case the left arm was alone affected for two years, then extension to the leg occurred, and, three months later, to the right arm and leg. In other cases, again, the affection may spread, in the course of a few months, from the arm to the leg on the same side, and two or three years may elapse before the other side suffers. Usually the tremor spreads when it is still moderate in the part first attacked, and there is a gradation of severity in the different limbs, roughly proportioned to the duration of the tremor. Sometimes, however, it becomes severe in the part first attacked before it spreads, or it may be slight in other parts even when general, and intense in the limb in which it began. Very rarely the tremor lessens as the disease advances, and rigidity fixes the limbs. Thus, in one patient, as the second hand became affected, the first became stiff and rigid, and the tremor lessened until it was much less than in the second arm. The variations in extension are thus so great as to make it difficult to foretell the course of a commencing case.

Duration and Cause of Death.—Paralysis agitans does not directly cause death, and the advanced age of most of its subjects renders its duration and its influence in shortening life difficult to determine. It usually lasts several years. The longest case which has come under my own observation had existed for ten years, but the disease has been known to last for thirty years. Death sometimes occurs from exhaustion, bedsores, etc., in the later stage; more frequently from intercurrent affections, especially of the respiratory organs, facilitated by the progressive muscular weakness, which involves the thoracic muscles as well as others. The tremor has been observed to cease before death.

PATHOLOGICAL ANATOMY.—In most of the cases of paralysis agitans that have been examined by modern methods and by competent observers (Charcot, Westphal, Berger, etc.), no changes have been discovered in the central nervous system or in the sympathetic ganglia; and this, although some of the cases were well marked and unilateral, so that the two halves of the nerve centres could be compared. This fact renders it doubtful whether the changes described by some—for the most part older—observers, had any relation to the disease. An hypertrophy of the nerve cells of the pons Varolii, which was described by Luys, has not been found by others. An induration of the pons, medulla and cord was noted by Parkinson, and has since been reported by several observers. The fact, however, that until recently insular sclerosis was confounded with paralysis agitans, throws doubts on the significance of these observations.

PATHOLOGY.—In the absence of any anatomical evidence of the seat and nature of the disease, the pathology of paralysis agitans has been the subject of copious speculation, into much of which it is not profitable to enter at length. The facts of the disease suggest, however, certain conclusions which may be briefly pointed out. We are justified in regarding the affection as one of the central nervous system, since we know nothing of clonic spasm in whole muscles, in consequence of a primary disease of the muscles themselves, or in consequence of idiopathic disease of the nerves. Such clonic contractions must be ascribed to the abnormal action of nerve cells. Tonic contractures may be the result of primary muscular changes, but they also are more commonly the result of a morbid state of the nerve centres, and the correspondence in seat of the clonic spasm and contracture of paralysis agitans makes it almost certain that both are due to the same cause; the fact that tonic contracture may precede clonic spasm prevents us from regarding the former as the result of muscular changes induced by the latter. That the morbid action is not primarily in the spinal cord is probable from the facts that, as a rule, even when severe, the tremor ceases during sleep, and that the unilateral commencement and hemiplegic extension are unlike those of spinal cord disease. The regions which have been thought to be the seat of the morbid process, by different authorities, are the pons, the corpora quadrigemina and the cerebellum. Regarding the two former the theories are purely speculative. We know at present little of the function of the large amount of gray matter which is interspersed among the fibrous tracts of the pons, or the symptoms of its disease, but the effects of lesions, degenerative or acute, of other parts of the pons lend no support to the hypothesis. Nor have symptoms in any way resembling paralysis agitans been observed in disease of the corpora quadrigemina. The theory that regards the cerebellum as the part affected is due to Dr. Hughlings Jackson, but the only actual fact on which it is based is that in some rare cases of cerebellar disease the interosseal position of the hands has been conspicuous. This has led him to suppose that there is a normal antagonism between the cerebral and cerebellar influence, the former tending to cause flexion of all the phalangeal joints, the latter flexion of the metacarpo-phalangeal and extension of the others by the interossei. But the symptom is extremely rare in cerebellar disease; as a rule, it is absent, and, by the converse process of reasoning, it might with equal cogency be ascribed to unantagonized cerebral influence. The same posture of hand is seen in tetany, in many cases of post-hemiplegic disorder of movement, and, what is less equivocal, it is a common posture of the hand during epileptic convulsions, in which the excess of nerve force is certainly not evolved from the cerebellum. If we turn to another class of facts, the cases in which tremor results from organic disease of the nervous system, we find the disease situated, as a rule, within the cerebral hemispheres, in the optic thalamus, posterior part of the internal capsule, foot of the corona radiata (Nothnagel), parietal lobe of the cortex (Chvostek), and island of Reil (Leyden).

In considering further the indications afforded by the symptoms as to the part of the brain that is the seat of the primary disease, the first obvious considerations are those that are mentioned in the discussion of the pathology of chorea, and are equally applicable to paralysis agitans. If we ascend the motor path from the spinal cord, the first nerve cells we meet are those of the motor cortex, and if the stimulation causing the tremor comes from the brain, it must proceed immediately from these cortical nerve cells. The question then presents itself—Are not these cells the primary seat of the disease? The early symptoms of paralysis agitans are almost exclusively motor, and there is nothing in them that is inconsistent with their origin in the cortex. Their peculiarities must, on any hypothesis, be due less to the seat than to the nature of the morbid process. The only sensory symptoms that are present are secondary in time and inconstant in occurrence; they are therefore probably secondary in their origin and distinct in their causation. We do not at present know on what they depend, or whether the abnormal sense of temperature is due to derangement of the sympathetic, or is the result of a change in the sensory centres in the brain. In the occasional inequality of the pupil, which is smaller on the most affected side, we have evidence of diminished action of the sympathetic, and the local increase in temperature occasionally observed may have a similar significance. But we know of no mechanism by which the motor symptoms can arise from a primary derangement of the sympathetic, and it is probable that this is simply secondary. It is certain that the functions of the sympathetic are represented in the cortex, and may be deranged by cortical disease. Lastly, it may be remarked, but without laying too much stress on the point, that the tremor of fear, which bears a close resemblance to that of paralysis agitans,* is most readily conceived as of cortical origin. At the same time it must be remembered that all disturbed function of high motor centres is expressed through lower centres (*e. g.* through the motor nerve cells of the spinal cord) the functions of which are thus disturbed in a similar though secondary manner, and such secondary disturbance, when long continued, may tend to acquire relative independence.

The slight change in the electric irritability of the peripheral nerves may have the same significance as in chorea, and the remarks on p. 978 are equally applicable to paralysis agitans. The structural changes in the muscles present in rare cases are no doubt merely the result of their long-continued active contraction

The precise character of the motor disturbance in shaking palsy must depend on the nature of the morbid process. This is apparently one that leads to a treble change. There is the intermitting release of nerve force, causing the tremor; there is a more continuous and slighter activity of the cells, producing the rigidity; and there is lessened capacity for activity, causing the weakness. It is possible that these are the result of slight differences in the precise character of the morbid change in the same cells. What that change is we have as yet no indication. The persistence of the disturbance of function shows that it must depend on changes in nutrition, but these are apparently far too fine to be revealed by alterations of structure recognizable by the microscope. Their relation to age as well as their character justifies us in regarding them as "degenerative," while their character on the one hand, and the negative results of microscopical research on the other, alike show that the alterations in nutrition must begin in the nerve elements themselves. Tremor, no doubt, depends immediately on physiological arrangements in the nervous system, which determine the relative action of antagonistic muscles. We have already considered this question in connection with the pathology of nystagmus (p. 628). Every contraction of one set of muscles involves a related contraction of their opponents; this relation doubtless facilitates the production of alternating movements, and it is easy to conceive that tremor may result from its derangement.

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* The resemblance must have struck any medical observer of the state of a person, especially a woman, who has just been through a sudden and conscious danger.

DIAGNOSIS.—When its symptoms are well marked, no disease is more easily recognized than paralysis agitans. The patient's aspect, carriage of body and limb, and continuous movement, make up an unmistakable picture. A difficulty sometimes arises in cases in which at first the tremor is absent, and the disease is indicated only by the loss of power and the fixity of feature and of limb, the slowness of movement, and the forward stoop. A knowledge of the significance of these symptoms, which are as characteristic as is the tremor, will prevent error. The greatest difficulty is when weakness and stiffness of the limbs come on without the usual attitude and facial expression, and especially if there is also chronic mental failure. The absence of increase in the myotatic irritability constitutes a difference from most other diseases causing similar weakness, but in some cases it is necessary to wait for a time before a certain diagnosis can be made.

In some cases the weakness of the legs is the chief source of inconvenience to the patient, and the case may be thought at first to be one of disease of the spinal cord, but attention to the state of the arms and face will generally show the nature of the case, and slight tremor can often be detected on close examination.

There are certain other diseases, however, from which there may be a difficulty in distinguishing cases that are not very well marked. Old persons sometimes present fine tremor of limb which bears considerable resemblance to that of commencing paralysis agitans, but does not present the same tendency to increase, or the associated rigidity. This "senile tremor" occurs chiefly in extreme old age, while paralysis agitans usually commences somewhat earlier. In senile tremor the head is especially affected, and often is the part in which the shaking commences, while in paralysis agitans it usually escapes, and its affection is always slight. Senile tremor often commences on both sides, paralysis agitans almost always on one. There is not, however, any sharp line of demarcation between the two affections, and it is difficult to say in which category some cases should be placed.

In "disseminated" or "insular" sclerosis, tremor occurs on movement only, and is usually more jerky and irregular in character than in paralysis agitans. In the cases of shaking palsy in which the tremor ceases during rest, there are the general aspect, carriage, and rigidity, which are absent in disseminated sclerosis. Moreover, insular sclerosis usually occurs before, paralysis agitans after thirty-five. In the former the head is usually conspicuously affected, and there is nystagmus, a symptom unknown in the latter. In sclerosis, articulation is syllabic; in paralysis agitans the words are uttered quickly and there is a tendency to run them together, rather than to separate the syllables. In the majority of cases these indications will enable the diagnosis to be made without difficulty. In very rare cases (Herterich, Schultze) insular sclerosis has been found, although the symptoms of paralysis agitans were present during life, but it is highly probable, in one case at least, that the coincidence was accidental.

The tremor that succeeds hemiplegia is fine, and is limited to the seat of paralysis, of which there is the history to guide the diagnosis. The differences between clonic retrocollic spasm* and paralysis agitans might seem to preclude the possibility of error. But the rare cases of paralysis agitans in which the head is carried backward may be mistaken for retrocollic spasm, and the finer clonic form of retrocollic spasm may also (as I have seen) be mistaken for paralysis agitans. The diagnosis rests on the characteristic affection of the hands in paralysis agitans, which commonly precedes the affection of the head, on the limitation of the spasm to the muscles moving the head in retrocollis, and on the fact that in this disease there are contractions in the frontal muscles, synchronous with those at the back of the neck.

The PROGNOSIS of a typical case of paralysis agitans is very unfavorable, so far as recovery is concerned. The disease seems to consist in degenerative changes, which are often as incapable of arrest as are the advancing years. A trifling amelioration or retardation of progress is all that can be hoped for. When the symptoms depart from the ordinary type, the prognosis is so far better in that there is a little more chance of doing good, since in a few of these cases remarkable improvement has been obtained by treatment. The danger to life, however, is almost as small as is the chance of recovery. The disease always lasts several years, and its duration is likely to be the longer the slower its advance. The more the case approaches to senile tremor, that is, the finer the movement and the slighter the tendency to rigidity, the less is the disease likely to interfere with the patient's comfort.

TREATMENT.—All causes of mental strain and of physical exhaustion should, as far as possible, be prevented. Life should be quiet and regular, freed, as far as may be, from care and work. All treatment that may fatigue or exhaust the patient's strength should be avoided. Among internal remedies, all varieties of nerve sedatives have been employed, and each has been praised by some and found to fail by others. Many of them, especially morphia, conium (Berger), hyoscyamin (Charcot and Oulmont), and Indian hemp, quiet the tremor for a time. Of tonics that have been recommended, carbonate of iron (Parkinson), chloride of barium (Brown-Séquard), and strychnine have been found, on extended trial, to deserve no special confidence. Arsenic, by the mouth, occasionally seems to do some real good; its subcutaneous use has been recommended by Eulenburg. Curara and bromides are valueless. My own experience is to the effect that arsenic and Indian hemp, the latter sometimes combined with opium, are of most use. I have several times seen a very distinct improvement for a considerable time under their use. In one case tremor had commenced in the right arm and leg an hour after a railway accident, and extended, three months later, to the left arm. Two years subsequently there was constant lateral movement at the wrist joints, but no tremor of the fingers. A great improvement occurred on Indian hemp, and a year later the tremor had almost ceased, being occasional only. It is in cases which, like this one, present some deviation from the common type, that benefit is most frequently experienced. Electricity in all forms is useless. Static electricity has been recommended by Reynolds, and later by Charcot. Some years ago I tried it in several cases very thoroughly, but could not discover the slightest improvement from its use. So, too, with voltaic electricity. I can entirely confirm the conclusion of Berger, who treated twenty cases sedulously in this manner without any of them being the least improved by it. Nerve stretching has been employed, but without any enduring result (Westphal). In one case stretching the brachial plexus is said to have lessened the tremor in the leg and removed it in the arm, but it caused paralysis of the limb with extensive wasting of the muscles.

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* The form of wry-neck in which the muscles at the back of the neck are involved.

OTHER FORMS OF TREMOR.

Senile Tremor.—In extreme old age slight tremor is often observed without the muscular weakness and rigidity that occur in paralysis agitans. At first it is noticed only on voluntary movement, and is generally influenced to a greater extent by movement than is paralysis agitans, ceasing or almost ceasing during rest, and always passing away during sleep. It usually commences in the arm, and often in both arms at the same time, but the head is affected much more frequently than in shaking palsy, and occasionally the tremor begins in the muscles of the neck. The tremor is always fine, the range of movement being very small. After a time it occurs during rest as well as on movement. It is little influenced by treatment. As already stated, it is doubtful whether this senile tremor is essentially different from paralysis agitans. Some cases are met with of a character intermediate between the two affections.

Simple Tremor.—Young or middle-aged persons, of both sexes, sometimes become affected with tremor, usually fine but sometimes irregular and unequal in the degree of movement, unaccompanied by weakness or rigidity. It is usually excited by movement and emotion, and commonly ceases during rest. The will can often control it for a time, and it interferes much less with complex actions than does the tremor of paralysis agitans. The handwriting, for instance, rarely exhibits irregularity. The parts affected are chiefly the hands and head, but the muscles of the face and tongue often present irregular tremor on movement, and the patient's aspect may closely resemble that of one affected with delirium tremens or general paralysis of the insane. It frequently gives rise to grave misconception regarding the habits of the sufferer. The shaking may disappear for a time, spontaneously or as the result of treatment, but it may also persist, getting neither better nor worse, for the whole of life.

The causes of this simple tremor are often obscure. It is sometimes apparently due to emotion, more frequently to prolonged anxiety, but still more frequently no exciting cause can be traced. Its occurrence is certainly influenced by an inherited neuropathic tendency. Occasionally the inheritance is more direct. A case has been mentioned in the account of paralysis agitans in which one member of a family suffered from the latter disease, and another from simple tremor. Two remarkable instances of this inheritance have been related by Liegey.* A gentleman suffered throughout his life from tremor, and died at eighty-two; his sister also trembled, and his mother, after a mental shock, presented tremor during the latter part of her life. In another case four members of the same family suffered from tremor of hands and lips, interfering with speech. In three it commenced in early adult life, but in one, a female, not until the cessation of the menses. A daughter of the latter suffered from symptoms resembling those of disseminated sclerosis.

This simple tremor has no tendency to shorten life. In spite of the occasional collateral relation to paralysis agitans, it seems to have little tendency to develop into the latter disease, and certainly is less closely connected with it than is senile tremor. When it has lasted for several years it is seldom influenced by treatment. In early cases some good may be done by nervine tonics and sedatives.

Hysterical tremor is very common. It may occur in two forms, one fine, the other coarse. The latter often consists in a rhythmical movement of the hands or head, continuing apart from any voluntary effort. The fine tremor occurs especially on movement, and its characteristic is irregularity (see Fig. 317, tracing 9). It varies in degree from time to time, presents a transient, often jerky, increase in range or acceleration in time, which are almost pathognomonic. Its characters and treatment are described more fully in the chapter on hysteria.

Asthenic Tremor.—In simple weakness, such as follows an acute disease, muscular tremor is common. It is fine in range, occurs only on movement, and is usually most marked after some slight exertion, as an effect of fatigue. The muscular weakness, the conspicuous general debility, and the distinct cause of the asthenia, render the nature of the case clear.

Toxic Tremor.—In mercurial poisoning, tremor is the conspicuous symptom, and also in some cases of lead poisoning and in chronic poisoning by alcohol, opium, and chloral. These cases are described at another page.

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* *Journal de Méd. de Bruxelles, 1882.*

WRY-NECK: TORTICOLLIS.

The term "torticollis" or "wry-neck" is applied to those conditions in which contraction of the muscles of the neck causes an unnatural position of the head. The contraction may be a persistent shortening of a muscle, or it may be an active spasm in one or several muscles. Besides these two chief varieties, a similar effect may be due to two other causes, which are chiefly important in respect to diagnosis, and may therefore be briefly mentioned. One is simple rheumatism of the muscles of the neck, impeding movement. The "stiff neck" of adults scarcely merits the distinctive name of wry-neck, but in children more pronounced deviation to one side or backward is occasionally met with. The affection follows exposure to cold, and is marked by considerable tenderness of the cervical muscles, which is clearly the chief cause of the unnatural position. These cases are really rheumatic, and not nervous in nature, and have been already alluded to in the account of the diagnosis of meningitis. The second condition is one that may be termed "false torticollis," because the deviation of the head depends, not on the state of the muscles, but on some other cause. The deviation, however, usually puts the sterno-mastoid on the stretch, and may thus give rise to an erroneous impression that the muscle is contracted, and is the cause of the deviation. The most common cause of this form is disease of the cervical vertebræ. The distinction from the true form will be considered in the section on diagnosis.

FIXED WRY-NECK: CONGENITAL TORTICOLLIS.

Fixed wry-neck depends upon shortening of some muscle, almost always the sterno-mastoid, which is often also atrophied, and sometimes firmer than normal. The condition is met with chiefly in children, and is thought to depend, in some cases, upon injury to the muscle from the traction on the neck that occurs during birth (Stromeyer). In other cases it is supposed to be due to developmental shortening of the muscle, the result of the inclined position of the head of the child in the pelvis.* It may not be noticed for some time after birth, in consequence of the shortness of the neck of young children. A similar condition may arise in adult life from an injury to the muscle, leading to secondary inflammation, and cicatricial contraction.

In fixed wry-neck the head deviates a little to one side, in the manner to be presently described as characteristic of active shortening of the sterno-mastoid, and it cannot be turned toward the side on which the muscle is contracted. The cause of the deviation is at once seen when the neck is examined; the sterno-mastoid on the side toward which the head cannot be turned is rigid, and stands out conspicuously; there is no active spasm. The affection is one of small importance. It can be readily and permanently cured by division of the tendon of the contracted muscle. When secondary changes have taken place in the articulations, it may be necessary to keep the head in position, for a time, by some support.

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* It is much more frequent on the right side, which gives support to the theory, as Busch points out, since in 70 cases out of 100 the head is in the "first position," which entails an inclination such as would permit the right sterno-mastoid to shorten.

SPASMODIC WRY-NECK.

Spasmodic wry-neck is a very different affection from that just described, although the two have been sometimes confused in descriptions of the disorder. The spasmodic form is a malady analogous to the facial spasm that has been described in the section on diseases of the facial nerve.

It is necessary to consider together all forms of active spasm in the muscles of the neck. Neither the character of the spasm, nor its distribution, afford ground for the separation of the varieties that it presents. The spasm may be either tonic or clonic, and both kinds of spasm may be met with in the same case, and even at the same time. The most important difference in the distribution of the spasm is, that while in most cases the spasm is either confined to one side, or is so much greater on one side as to cause a lateral deviation of the head, in a few cases the spasm is equal on the two sides and is either confined to or greater in the muscles at the back of the neck, so as to produce a backward movement of the head. The second variety may conveniently be distinguished as "retrocollic spasm."

ETIOLOGY.—Spasmodic torticollis is more common in women than in men. Of 24 cases, of which I have notes (all those of hysterical nature being excluded), 7 were in males and 17 in females. It commonly begins in the middle period of adult life, between thirty and fifty years of age. Two-thirds of the cases commence during this period. It is rare under thirty,* but I have met with cases that began at twenty-eight and twenty-five. It occasionally begins after fifty; the latest case that has come under my notice began at sixty-six. The two early cases were in males; otherwise, both sexes seem to suffer in about the same proportion at the different ages. Most cases in females under thirty are of hysterical nature, and this is also probably true of the rare cases in which similar spasm is met with in boys.†

Neurotic heredity occasionally predisposes to this as to other maladies of the same class. In some cases there is a history of epilepsy or of insanity, and occasionally of diseases more closely allied to torticollis. The brother of one patient had suffered from facial spasm, and the sister of another (who was suffering from the true, not the hysterical form) had suffered from torticollis itself, and had recovered. In a few instances, the patient has himself suffered, at a previous time, from some other functional disorder of the nervous system. Thus, in one case of persistent torticollis, the patient ten years previously had been affected with general tremor, which lasted for three years and then passed away. The affection has followed an attack of melancholia.* It is possible that true torticollis may grow out of a habit spasm; thus, one patient had been liable, for twenty years, to nodding movements of the head, when he was engaged in earnest conversation.† Depressing emotion often precedes the onset, but less frequently than in the case of facial spasm. In other instances, there is a history of some feebleness of general health. I have more than once known the affection to follow a fall at an interval of a month or two, and a similar sequence has been recorded by others; in one recorded case, spasm on the right side of the neck followed a fall on the left temple.‡ In some cases the disease seems to be distinctly excited by exposure to cold. Thus, one patient had a severe cold and a rheumatic stiff neck; as the stiffness passed off, clonic spasm came on. But an initial feeling of stiffness is sometimes taken as evidence of exposure to cold, without any real grounds for the assumption. Excessive use of the muscles is probably an occasional cause.§ It is possible that, in rare cases, spasm in the neck is caused by malarial poison.|| In not a few cases, no immediate cause can be traced; the affection comes on in robust individuals, who are apparently in perfect health at the time of the onset.

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* In the only earlier case that I have seen, in which the spasm commenced at thirteen, and still existed at twenty-nine, the movement was somewhat unusual in its character, and was probably a persistent habit spasm, from another form of which the patient had also suffered.

† See note on p. 1017. It is important to remember that most varied and obstinate forms of clonic spasm occur in the subjects of hysteria, and are, apparently, in some way, due to this disease. They are described in the chapter on hysteria.

Among the causes of torticollis commonly described, is paralysis of the muscles of one side, permitting their opponents to draw the head to the other side. This is a theoretical cause, which has little counterpart in fact. It is doubtful whether a true paralytic torticollis is ever seen. The amount of muscular action that is needed to keep the head at rest in the mid-position is extremely small; the rotators are numerous, and all are never paralyzed.

SYMPTOMS.—The spasm itself may be the first symptom, but in many cases this is preceded by pain, or by some unpleasant sensation. The pain may be of an acute neuralgic character, or it may be a dull rheumatic pain. It may be felt in the neck, or in the occipital region on one side, or behind the ear, or in the arm. It often continues for a time, after the spasm has

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* Allen Sturge, *Lancet*, *1879*, i, 160.

† Reynolds states that he has known the disease to follow "writers' cramp" (*System of Med.*, vol. ii, p. 787); but it is probable that it was merely the commencing spasm (beginning, as it sometimes does, in the hand) that interfered with writing. This was so in one case under my own observation, that shown in Fig. 322.

‡ T. Fournier, "Le Tic Rotatoire," *Thèse de Strasbourg*, *1870*.

§ Annandale has recorded a case of spasm in the neck apparently due to this cause. The subject was a girl aged twenty-four, who, in the course of her work as a weaver, had occasion to move the head rapidly first to one side and then to the other, but especially to the left, and in the spasm the head was inclined and rotated to the left side. But the case was very unusual in that there was no spasm when the head was turned to the left, although clonic spasm came on at once when it was moved out of that posture (*Lancet*, *1879*, i, p. 555).

|| As in a case of intermittent contractions of one sterno-mastoid (coming on each day at the same time, and lasting four or five hours) in a child of four, recorded by Simon. The child had had intermittent fever, and the attacks were quickly cured by quinine (Ref. in *Lancet*, *1879*, i, 26).

developed. In other cases, again, a sense of stiffness in the neck attends the commencing spasm. Less commonly the onset is preceded by sensations of a more or less vertiginous character, which may, perhaps, indicate some morbid action in the nerve centres, of which the spasm is the result. Thus, in one case the spasm commenced a few weeks after a sudden, brief, "whirling" sensation in the head. Another patient at first, when walking, experienced a strong tendency to deviate to one side, toward which there was a slight rotation of the head, but as the rotation increased to distinct spasm, the tendency to deviate ceased.

The actual onset of the spasm is usually gradual, and some months pass before a considerable degree is attained. Very rarely it comes on acutely; in one case it became severe in the course of a single week, and in another at the end of a single day. Once developed, its increase is usually steady, but now and then it ceases after a few months, to recommence before many more months have passed. The tendency of the head to go to one side is often first felt when the patient is walking or excited, and ceases during rest or mental tranquillity. The seat of the first spasm is generally that in which the contractions are ultimately most severe; in exceptional cases it commences in some other part. In two cases it began in the hand, and in one in the face, although in each the neck was ultimately most affected.

The symptoms vary according to the muscles that are most affected. These are generally the large muscles of the neck, and in the most common form, torticollis proper, the spasm is either confined to one side, or is greater on one side than on the other, and hence its effect is to cause a deviation of the head to one side, either a rotation or a lateral inclination. Less commonly, as already mentioned, the muscles of both sides are equally involved, and the head is then inclined backward. Usually many muscles are affected; in rare cases one only. The sterno-mastoid is the muscle most frequently involved, and in the cases of solitary spasm it is almost always this muscle that contracts. Of twenty-four cases, there was some spasm in the sterno-mastoid, on one side or on both, in no less than twenty-two. In two-thirds of the cases (fourteen), the muscle was affected on one side only, and in five no other was involved. The muscle most frequently associated with the sterno-mastoid is the upper part of the trapezius, and it is generally the trapezius on the same side. Of seven examples of this combination, the associated trapezius was on the same side in six on the other side in one. The splenius is associated with the sterno-mastoid about half as frequently as the trapezius, and it is usually the muscle of the opposite side. The scaleni occasionally contract; I have once known both scaleni to be involved with one sterno-mastoid. Often there is also spasm in the platysma myoides, which draws up the skin into characteristic folds, but does not influence the position of the head. In rare cases, of which I have only seen one, rotation of the head is produced by the deeper muscles alone, probably the rectus and obliquus, and no accessible muscle can be felt to contract.

The side on which the muscles contract most strongly is more frequently that of which the muscles turn the head to the left, and hence this is the common direction in which the head deviates. Thus the right sterno-mastoid was involved most or only in thirteen cases; the left sterno-mastoid only in seven. The right trapezius, which also turns the head to the left, was likewise affected more frequently than the left, and the left splenius, which turns the head to its own side, was involved far more frequently than the right. Although the cases are not numerous, the uniformity with which this rule obtained can scarcely be a matter of accident. It might be anticipated, *a priori*, that the muscles which move the head to the right would suffer most, because they are associated in action with the right arm, but it must be remembered that many energetic movements of the right arm are effected toward the left.

In the cases in which the muscles of the two sides are equally involved the sterno-mastoids are also most frequently affected, but usually in slight degree. The trapezii usually contract strongly. Rarely all the muscles of the neck seem to be in action.

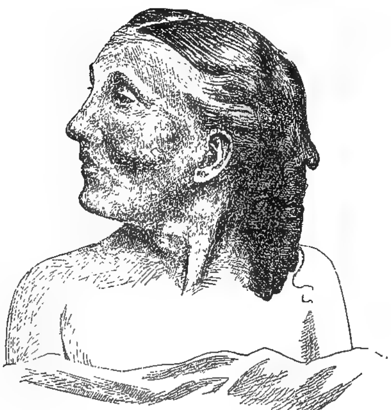
The movement of the head caused by the spasm necessarily differs according to the muscles that are chiefly involved. We have seen that the spasm is seldom limited to a single muscle; more often several muscles contract together, and the movement of the head is that due to their combined action. As the combinations are numerous, and the muscles contract in various degrees, the resulting movement of the head varies considerably in different cases, and sometimes in the same case at different stages of the disease.

The *sterno-mastoid*, acting alone, causes rotation of the head so as to bring the mastoid process nearer to the inner end of the clavicle. In this movement the face is turned toward the opposite side, the chin is put forward, and the head is slightly inclined toward the side of the acting muscle.

The highest part of the *trapezius*, acting alone, causes only a slight rotation of the head toward the other side, but it inclines the head strongly toward its own side, and at the same time draws the head backward. It also raises the shoulder, rotating the scapula. The middle and lower parts of the trapezius are seldom involved.

The *splenius* of one side inclines the head, and very slightly rotates it, toward the same side.

FIG. 320.

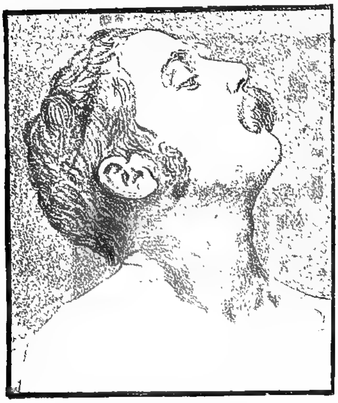


Torticollis due to spasm in the left sterno-mastoid.

Equal contraction of the trapezii and splenii on both sides causes a backward movement of the head.

The effects of the chief combinations are as follows: The association of the sterno-mastoid and the trapezius of the same side greatly increases the inclination of the head toward the shoulder, and this increase in the inclination prevents any increase in rotation. The association of the sterno-mastoid of one side with the trapezius of the opposite side prevents the inclination of the head, and does not appreciably lessen the rotation caused by the former muscle, the trapezius being a feeble rotator. Hence the rotation of the head is often very great. The combination of one sterno-mastoid and the opposite splenius causes extreme rotation of the head, so that the face, as in Fig. 320, may be turned toward the shoulder. I have not seen any case in which one sterno-mastoid was associated with the splenius of the same side, but the resulting movement would probably be similar to that produced by the combination with the trapezius of the same side, a strong inclination of the head toward the shoulder. In the case shown in Fig. 321 both splenius muscles were involved (the right most), as well as the right sterno-mastoid, and a strongly backward inclination with rotation was the result.

FIG. 321.



Torticollis. Spasm, chiefly tonic, in the right sterno-mastoid, and especially in the right splenius, with slighter spasm in the left splenius. The patient was a man, aged twenty-six, in whom the spasm had existed for six months; it commenced gradually after a period of great mental anxiety.

Bilateral spasm, equal on the two sides, always causes a backward movement of the head, because both sterno-mastoids scarcely ever contract alone, and the spasm in the muscles at the back of the neck draws the head backward, and then the inclination is increased by the sterno-mastoids. It is doubtful whether there is ever a forward movement in any case properly belonging to the group now under consideration. The retroflexion of the neck is sometimes very great; the face may even be horizontal, turned directly upward, as in one case that I have seen. Such extreme spasm is, however, rare; more often the contraction is moderate in degree. An interesting fact about this retrocollic spasm is that contraction in the frontales muscles is almost always associated with the spasm in the back of the neck. The association is a physiological one; in the act of looking upward, the head is bent back, and the eyebrows are raised by the frontales. If the spasm is clonic, the contraction in the forehead is synchronous with that in the muscles of the back of the neck.

The spasm in all forms of torticollis may be either tonic or clonic, and often both kinds of spasm occur together; clonic spasm may become tonic as it becomes intense, or, more commonly, tonic spasm, as it increases, becomes clonic and jerky. It usually varies in intensity from time to time, and either form may be intermittent. The patient is able to keep the head still for a time, but every now and then it is drawn to one side, either steadily or in jerks. The spasm is seldom absent for more than a few minutes. As it comes on, the affected muscles stand out conspicuously in their contraction. Occasionally the spasm, either tonic or clonic, is practically continuous. The same variations are seen in the retrocollic variety, but clonic spasm is relatively more common, and intermissions are less common. The contractions are often moderate in degree, and frequent in recurrence, so that the spasm bears more resemblance to tremor than in the ordinary forms.

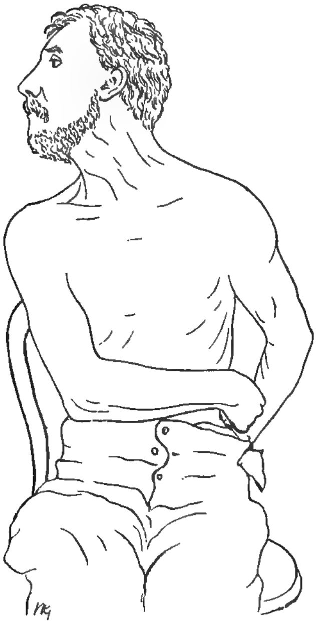
In most cases there is a strong tendency, in the course of time, for the spasm to spread and to involve other muscles in addition to that in which it commenced. Even when the contractions are limited, if the movement of the head is forcibly prevented, the spasm often temporarily spreads to other muscles. Thus in a case in which one splenius alone habitually contracted, if the movement was prevented, the sterno-mastoid began to contract.

In some cases, the spasm is not limited to the muscles of the neck; it involves either the arm or the face or the muscles of mastication. The face is seldom constantly involved, but muscles occasionally contract during a severe paroxysm. In one instance, with clonic spasm in one sterno-mastoid and trapezius, at the height of the attack there was often twitching of the eyelids and pouting of the lips. The spasm in the face may occur on the side on which the sterno-mastoid contracts, or contracts most strongly. In one instance, in which there was continuous clonic spasm in the right sterno-mastoid, and very little in the left, there were almost constant contractions in the right side of the face. Rarely the spasm is equal on the two sides of the face, when the spasm in the neck is unequal, as in a case just mentioned. In retrocollic spasm there may be some bilateral contraction in the lower facial muscles, especially in the elevators of the upper lip, at the height of a paroxysm, in addition to the constant spasm in the frontales, already described. The masseters are involved in true torticollic spasm rarely, and only at the height of a paroxysm. In chronic retrocollic spasm they are sometimes constantly affected.

When the arm is involved, there is always unequal spasm in the two sides of the neck. The sterno-mastoid that is most affected may be on the same side as the arm, or on the opposite side. In the cases that I have seen in which the arm was affected after the neck, it was the arm on the side opposite to the most affected sterno-mastoid. When the arm is affected first, either sterno-mastoid may be the seat of the chief spasm, and the head may thus be turned from, or inclined toward, the arm that is affected. In one case of clonic spasm in the right sterno-mastoid there was also spasm in the left deltoid, but in no other muscle. Another instance of this relation is presented by the patient shown in Fig. 322. In him the spasm commenced in the arm and spread to the neck, where it involved the right sterno-mastoid, the left trapezius and splenius, and the right platysma. It became continuous in both arm and neck, so that the upper arm was in constant strong abduction, and the forearm was carried in front of the body, while the face was turned toward the shoulder.

Considerable spasm always occasions discomfort to the patient, partly due to fatigue in the muscles. There is not commonly any actual cramp-like pain in them. The neuralgic pain that is often complained of at the onset usually ceases after a time, but sometimes continues in varying degree. When there is spasm in the arm, the limb is often the seat of considerable pain, and I have known severe pain in both arms to attend the attacks of spasm in the neck. It is said that there are occasionally tender points in the course of the cervical nerves, but they are not common. Another rare sensory symptom is tingling and numbness in the arm, from the compression of the nerves of the brachial plexus by the scaleni.

FIG. 322.



Spasm, chiefly tonic, in the left sterno-mastoid, right trapezius, and the whole of the right arm, which was strongly abducted by spasm in the pectoralis. The patient was a man aged forty-six, in whom the spasm had existed for two years. It commenced gradually in the hand.

The muscles that are the seat of considerable spasm undergo, in time, some hypertrophy, and may become very distinctly larger than those on the other side. They never waste. The electrical irritability is generally normal; sometimes, indeed, it is abnormally great, so that a very weak current of either kind suffices to put the muscles in a state of strong contraction.

The course of the disease varies in different cases. Often the spasm slowly increases in intensity and widens in range, but ceases to be progressive after a few years, and may remain stationary for the rest of life. In other cases, however, it remains slight, sometimes confined to a single muscle, and may not undergo perceptible increase during many years. Less commonly it diminishes after it has lasted for a few months or years, and may pass away, either spontaneously or as the result of treatment. In some instances it does not disappear altogether, although it becomes slight and occasional. In a case recorded by Brodie, the spasm ceased entirely during an attack of insanity, and returned when the mental state become normal. The disease does not develop to any disorder of more serious character, nor has it any tendency to shorten life, although it may lessen very much the pleasure of existence.

PATHOLOGY.—In no case of torticollis has a lesion been found that can be regarded as an indication of the morbid process to which the spasm is due. The pathology of the disease is, therefore, a matter of inference from its symptoms, its causes, and from what we know of the nature of allied diseases, especially of facial spasm (see p. 660). It is almost superfluous to state that the morbid process cannot be one of the muscles themselves. The facts that many muscles are involved, and that when the spasm commences in a single muscle, it usually spreads to others, as well as the general pathology of spasm, make it probable that the muscular contractions depends on the over-action of nerve cells, and not on any irritation of nerve fibres. But we do not know what nerve cells are primarily deranged, whether they are the cells of the lower spinal centres, or those of the higher cortical centres. The symptoms suggest that in some cases the lower, and in other cases the higher centre is that from which the discharge originates, because the distribution of the spasm indicates that the functional level, so to speak, of the discharge is not always the same. In some, the affected muscles are those that act together in producing a given movement, although they are supplied by different nerves. It seems probable that such a spasm depends on the over-action of a centre in which movements, rather than muscles, are represented. On the other hand, when the spasm is of a single muscle, or of muscles that are not physiologically associated, it seems more probable that it depends on a lower centre, such as the spinal or bulbar gray matter from which the nerves proceed. The physiological association of muscles is specially noticeable in the contraction of the frontales in retrocollic spasm. These muscles are innervated from the facial nucleus in the pons, while the muscles at the back of the neck are for the most part innervated from the spinal cord. It seems far more probable that such an associated spasm depends on the cortical centres than that it is due to the lower centres. So, too, in the cases in which there is spasm in those muscles on both sides of the neck that are concerned in turning the head to one side. On the other hand, such an association as that of the muscles which incline the head toward one shoulder, or strong spasm limited to one sterno-mastoid and the opposite deltoid muscles, seems more intelligible on the assumption that the over-acting nerve cells are those of the lower centres; since these muscles do not commonly act together without others. We have seen reason to believe that the over-action that causes facial spasm is sometimes in the cortical centres, and sometimes in the lower centres. At the same time we must not lay too much weight on the significance of functional association. It is probable that the connection of the cells of the lower centres is determined by this association, and the manner in which the spasm spreads among the muscles of both sides is often more intelligible on the assumption that the over-acting cells are those of lower centres. It may be noted, moreover, that there is reason to believe that structures in the lower part of the pons Varolii influence the associated action of rotation of the head (see p. 611). There is one important difference between the spasm in torticollis and that which occurs in the same muscles as a consequence of cortical disease. In the latter a conjugate deviation of the eyes is usually associated with that of the head, and this is true, also, of the spasm that results from irritation of the structures in the pons. In torticollis, however widely the spasm spreads, the muscles of the eyeball are never implicated. From these considerations it seems doubtful whether we have at present sufficient evidence to justify us in coming to any conclusion regarding the seat of the disease.

The nature of the morbid process is also involved in as much obscurity as is that of all other similar spasmodic diseases. That there is an unnatural state of the nerve cells, in consequence of which nerve force is spontaneously liberated, is scarcely more than a statement of the observed facts, and leaves us still ignorant of the condition on which the phenomena depend. There is at present no evidence that the over-action of the cells depends on any morbid process outside them, on any lesions that could be detected by naked-eye or microscopical examination. The fact that neurotic heredity can often be traced as a predisposing cause, suggests that the over-action has its origin in a primary derangement of the nerve elements themselves. It is to be noted that this disease, in common with other spasmodic affections of the same class, comes on usually in adult life, and seems to depend on a local failure in the stability of function that has been long developed, and cannot therefore be ascribed either to an imperfect establishment of function on the one hand, or to actual senile changes on the other. In their occurrence at the time when nerve action might be expected to possess its full stability and precision, and also in their local character, these affections constitute an enigma to which we have as yet no solution.

Diagnosis.—The diagnosis of spasmodic torticollis seldom presents any difficulty. The condition that has been termed "false torticollis," in which there is a deviation of the head from some other cause than muscular contraction, is readily distinguished from the variety that is due to shortening of one sterno-mastoid (with which alone it is likely to be confounded) by noting the relation between the position of the head and the side on which the muscle is tense. In the spurious form the sterno-mastoid is tense on the side toward which the face is turned; in the true form the tension is of the opposite muscle. Retrocollic spasm, when the movements are small in range, is apt to be mistaken for simple tremor, but the muscular contractions can be felt to be more violent than they ever are in simple tremor, and the associated spasm in the frontales, which has been conspicuous in all the cases I have seen, supplies an absolute distinction between the two affections. Another occasional difficulty is the distinction of true torticollis from hysterical spasm of torticollic type. The facts of etiology show that under thirty true torticollis seldom occurs in females, and therefore at that period there will always be a presumption that the affection is hysterical, but after thirty the mere fact of sex must not be allowed weight. As a rule, hysterical spasm tends to spread from the neck to the trunk, which becomes affected by writhing movements. In all the cases that I have seen in which the conditions of onset raised a suspicion of the hysterical nature of the case, and the spasm was limited to the neck, and was typical in form, the affection turned out to be the true variety.

Prognosis.—The facts of the course of torticollis, already mentioned, show that the prognosis must be grave in every developed case. The more severe the spasm the wider its extent, and the longer its duration the smaller is the prospect that considerable relief will be obtained.

Treatment.—The removal of any influences that can be regarded as having helped to induce the disease is, of course, of the first importance. Cases do not often come under treatment in the early stage, but it is desirable that any stiffness due to cold, that has lasted for an unusually long time, should be treated by rest and hot applications. Unfortunately, it is very rare for causal treatment to have any appreciable influence on the disorder. The same may be said of tonic treatment in general, necessary as it is to give tonics whenever they are indicated. Drugs that may be called "stimulant nervine tonics," such as valerianate of zinc and asafœtida, sometimes have a marked influence on the spasm, even in cases in which it is certainly not hysterical in nature. In one, the administration of these two drugs, without other treatment, reduced severe spasm to a very trifling degree of intensity, and the improvement continued for some months, but the spasm then increased again and resisted all remedies. Sedatives frequently lessen the spasm while their action on the system continues, especially opium, morphia, chloral, succus conii in large doses (gradually increased to two ounces), bromide of potassium, and Indian hemp. But the discontinuance of the drug is usually followed by a relapse to the former degree of severity, although occasionally this does not take place for some weeks or even months. I have only once known great and permanent improvement to result from the internal administration of sedatives, which in this case consisted of the combination of bromide of potassium and cannabis indica. There is one mode of treatment that has in many cases had a more lasting influence, but it is a remedy that has to be carefully weighed in the balance against the disease—the hypodermic injection of morphia. Continued for several months, in doses increased gradually to half a grain or a grain a day, it has entirely removed the spasm. But the patients so treated find the discontinuance of the drug extremely difficult, not only on account of the craving for it that is established but also because (what is perhaps part of the craving) they feel, or imagine they feel, a tendency to the return of the spasm if the drug is discontinued. In one very severe and distressing case of retrocollic spasm, in a man aged forty-five, all muscular contractions ceased under the influence of the drug, and the patient has now been free for ten years, but has never left off the use of morphia, although he has not increased the dose above that named. A patient so treated should undergo a subsequent course for the eradication of the morphia habit, before he passes from the hands of the practitioner, and the effect of the withdrawal of the morphia should be carefully observed. Cocain may be used, but for a time only, as the morphia is being withdrawn. Unless these measures are adopted, the treatment is the substitution of one morbid state for another, and that which is substituted may be in the end the more harmful of the two. The patient above mentioned, however, has had ten years of perfect comfort in exchange for a condition that was most distressing; the spasm was so severe that he could never enter a public conveyance on account of the painful observation he excited. Reynolds has also described favorable results from the treatment.* The addition of arsenic to the injected morphia has been advocated by Radcliffe, but arsenic alone, either by the mouth or by hypodermic injection, seldom has much influence,* and it is therefore probable that the influence of the combination is due chiefly to the morphia.

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* "System of Med.," vol. ii, p. 797.

Electricity has been used in various ways in the treatment of the disease. The method that has generally been employed is the application of the voltaic current to over-acting muscles, a weak current being used, in which the current has some sedative influence. The positive pole may be placed just below the occiput or on the highest accessible part of the nerve, and the negative on each contracting muscle, for five or ten minutes. Thus employed, it has been said to do good in some cases; in one, of severe bilateral spasm, recorded by Poore, the affection almost ceased under the treatment. In general, however, it fails to do more than produce a slight and transient diminution in the spasm; I have not myself seen any permanent good result from electricity, even when long continued. Faradization of the antagonists of the over-acting muscles has also been recommended, but this method is as destitute of rational foundation as it is of practical effect. The disease probably never results from the weakness of the opponents, and cannot be lessened by increasing their activity. I have known a blister over the contracting muscles to lessen the spasm for a time in very marked degree, but the effect is seldom as permanent as it often is in hysterical spasm.

Mechanical supports afford occasionally some relief, which depends on the fact that the greater the movement and muscular contraction, the greater is the discomfort produced. Many patients are comparatively easy when they are sitting in a chair with a high back, against which they can fix the head. The best support is one in which a rigid rod is carried up to the back of the head, and from its top a small pad projects on the side toward which the occiput is turned by the spasm, so that the patient is able to press the head against the pad. The instrument needs careful contrivance, having to be specially adapted to each individual case, and it is only when the spasm is moderate in degree that the expedient is fairly successful. Instruments that are designed to fix the head rigidly, and prevent all movement, can never be endured.

A few cases are on record in which division of the spinal accessory nerve was followed by a cure, but these cases have differed in some particulars from the common form. One instance is the case recorded by Annandale, mentioned at p. 1017, note.† The nerve has also been stretched in many cases. Both division and stretching have been unsuccessful in most of the cases in which these measures have been adopted. The spasm has returned when the effect of the stretching had passed away,‡ and when the nerve has been divided, the spasm has often spread to other muscles. I have seen one case, in which the spasm commenced in the hand and spread up the arm to the neck, in which the brachial plexus was stretched, but the operation was followed by no improvement in the arm, and by a great increase in the spasm in the neck. Should nerve stretching be employed in any future case,* it is desirable that the operation alone should not be trusted to, but that its influence should be supplemented by that of the hypodermic injection of morphia.

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* A case of clonic spasm which recovered under the administration of arsenic, recorded by Buzzard, was certainly of hysterical nature (*Brit. Med. Journal, 1881*, p. 937).

† Another successful case has been recorded by DeMorgan (*Med.-Chir. Rev.*, July, 1866).

‡ The only case in which great improvement followed stretching was very unusual, in the fact that the patient was a boy of fourteen, and the spasm affected also the muscles of the back and both arms. It was probably more closely allied to the hysterical than to the ordinary form. (Southam, *Lancet, 1881*, ii, p. 369.)

The most useless procedure that has been employed is the division of the tendon of the contracting muscle. This has been sometimes done apparently because the operation cures the totally different fixed wry-neck. In active spasm it is worse than useless, because the division of the tendon permits the muscle to shorten, while it does not check the spasm, and the greater the contraction of the muscular fibres the greater is the pain occasioned by the contraction. In one case that came under my notice some time after the operation had been performed, the patient's suffering was much increased, and, probably in consequence of the increased pain, the spasm had spread to other muscles.

TETANUS.

Tetanus is a disease of the nervous system characterized by persistent tonic spasm, with violent brief exacerbations. The spasm almost always commences in the muscles of the neck and jaw, causing closure of the jaws (*trismus, lockjaw*) and involves the muscles of the trunk more than those of the limbs. It is always acute in onset, and a very large proportion of those who are attacked die. The disease is usually the result of a wound (*traumatic tetanus*), but sometimes occurs without external injury, especially from exposure to cold (*idiopathic* or *rheumatic tetanus*). It occurs, also, in newly-born children (*tetanus neonatorum, trismus nascentium*), and, rarely, after childbirth or abortion (*puerperal tetanus*).

Traumatic tetanus has been divided into two forms, *acute* and *chronic*, as it commences before or after the tenth day from the injury. But this is a misuse of words, since all cases are acute or subacute at the onset, and all cases that recover pass through a chronic stage.

ETIOLOGY.—It is convenient to consider separately the causes of the disease in puerperal women and newly-born children. The ordinary forms of the disease are far more common in males than in females. In traumatic cases the proportion between the sexes is nearly as six to one.† The idiopathic form also occurs in males more frequently than in females, although the disproportion is not quite so great as in the traumatic form. Of 46 idiopathic cases which I have collected, 37 were males and 9 females, a proportion of four to one. Doubtless the chief cause of the different liability of the sexes is to be found in the increased exposure to the immediate causes that is involved in the occupations of men.

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* Details of the operation will be found in the paper of Southam.

† The published series of cases at Glasgow (Lawrie, *Glasgow Med. Journal, 1853*, vol. i, p. 339), and at Guy's Hospital (Poland, *Guy's Hosp. Rep., 1857*; F. Taylor, *Guy's Hosp. Rep., 1878*), idiopathic cases excluded, comprise 160, of which 138 were in males and 22 in females. Consecutive series of cases such as these give more accurate statistics than collections of cases which have been published separately (often on account of some special feature), and also than the more massive but undifferentiated statistics of the general mortality.

Tetanus occurs at every period of life. The distribution of the 160 traumatic cases and of 46 idiopathic cases is shown in the tables below:—

TRAUMATIC	1–9	10–19	20–29	30–39	40–49	50–59	60 +
Males	5	36	32	29	20	11	5
Females	4	7	4	5	2	0	0
Total	9	43	36	34	22	11	5
Per cent.	5.5	27	22.5	21	14	7	3

IDIOPATHIC	1–9	10–19	20–29	30–39	40–49	50–59	60 +
Males	2	5	9	12	4	0	3
Females	1	4	0	2	2	0	0
Total	3	9	9	14	6	0	3

During the first five years of life (the first month excepted) there is an almost complete immunity from tetanus, the cases under ten occurring after five. The second decade of life yields the largest proportion, rather more than a quarter; the third and fourth decades, each rather less than a quarter, so that, in the thirty years of life from 10 to 40, about three-quarters of the total number of cases occur. The disease continues, with decreasing frequency, up to old age, and has been met with as late as 89 (Yandell). The few cases in childhood (5 to 10 years) occur in each sex with almost equal frequency; from 10 to 40 the proportion between the sexes is near the average (one to six); between 40 and 50 women suffer less frequently, and after fifty cases in women are very rare.

Dark-skinned races have been observed to suffer from all forms of tetanus more frequently than Europeans, in countries where both are exposed to the same influences. In hot countries, *e.g.*, India and the West Indies, tetanus is far more common than in temperate regions. Even in the tropics, it is chiefly prevalent at certain periods and in certain places. In temperate climates, however, its occurrence does not appear to be influenced by weather, or by the season of the year.

It is very doubtful whether previous health exerts any influence on the occurrence of the disease. It is met with in the robust as well as in the weakly. Erichsen believes that depressing emotions predispose to it, and there is some reason to think that fear of the disease may influence its occurrence.*

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* A curious case bearing on this point has been communicated to me by Mr. A. C. Roper, of Exeter. A highly nervous girl was thrown from her horse, and sustained a lacerated wound of one hand, the abductor pollicis being cut through by a sharp stone. She was much afraid

The *immediate* causes of tetanus are chiefly two—injuries and exposure to cold. Both may coexist. The traumatic cause usually involves an actual wound of the surface. The wound may be in any position, of any character, and of any degree of severity. Cases have been recorded in which tetanus has followed the most trifling injuries (the stings of bees and wasps, the prick of a thorn, the removal of a foreign body from the eye), every kind of incised, punctured, contused and lacerated wounds, and almost every form of surgical operation, from the extraction of a tooth to ovariotomy. But it is far more common after punctured, contused and lacerated wounds and extensive burns, than after incised wounds. Hence it is comparatively rare after surgical operations. Injuries of nerves have been supposed to give rise to it with especial frequency, but those of small nerves seem to be more effective than those of large nerve trunks. The order of frequency * with which the several parts have been the seat of the wound that caused tetanus is (1) hand, (2) leg, (3) foot, (4) head and neck, (5) arm, (6) trunk. It is uncertain what influence the condition of the wound exerts; it has sometimes been in an unhealthy, irritated condition; more frequently it has been in a perfectly healthy state, and sometimes cicatrization was advanced, or even complete. Spontaneous disease, ulcers, for instance, may also cause tetanus, although far less frequently than wounds. In rare instances, the disease has followed injuries which involved no breach of surface, as flogging, and falls on the back of the head and spine.

The interval between the infliction of the wound and the appearance of the first symptoms of tetanus, is usually from five to fourteen days (in two-thirds of the cases), but the malady may set in earlier or later. Many cases have been recorded which commenced within forty-eight hours of the injury, a few within twelve hours, and one or two in which tetanus came on immediately. In Robison's often-quoted case, a negro, who cut his hand with a piece of porcelain, was dead in fifteen minutes. On the other hand,

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of "lockjaw," but her fears were removed, and all went on well for four days, when her dread of the disease was revived by a friend's expression of anxiety for her on this account. She immediately procured "Chambers's Encyclopædia," and read the account of the disease given there. In the evening of the same day she declared that she could not open her mouth, and that her neck was stiff. There was distinct rigidity, which was lessened by a hypodermic injection of morphia, but continued on and off for five days, without any constitutional disturbance. Her mind was constantly dwelling on the subject. On the sixth day the spasm increased in intensity, and on the seventh the symptoms of tetanus were so distinct that the hand was amputated; but the spasms continued, and she died on the eighth day from the onset of the trismus, and the twelfth after the accident.

* According to 395 cases tabulated by Thamhayn (*Schmidt's Jahrbucher*, vol. cxii, 1861). This does not show its absolute frequency in wounds of each part; for the determination of this no adequate statistics at present exist. It is said by some to be very seldom caused by wounds of the head and neck. Of 505 cases of tetanus tabulated in the records of the American civil war, only twenty-one were due to injuries of those parts. Nevertheless, one peculiar form of tetanus (cephalic tetanus) seems to be produced chiefly by injury in the region of the fifth nerve (see p. 1035).

tetanus occasionally commences during the third or fourth week after the injury; scarcely ever later than a month.

Idiopathic tetanus is usually excited by exposure to cold, especially to wet cold when the body is perspiring. In many cases the exposure has been repeated and prolonged. When due to a single exposure, so that the interval could be ascertained, it has rarely exceeded two days, and has frequently been only a few hours. In many cases classed as traumatic tetanus the disease has been excited by exposure to cold after the receipt of the wound. This is probably the chief cause of the special frequency of tetanus in soldiers wounded in battle. The influence of cold is also to be traced in the causation of some cases of puerperal and neonatal tetanus.

In two recorded cases tetanus followed alcoholic intoxication without other cause that could be discovered. In two others exposure to the hot sun apparently excited the disease, and in one case the malady (rapidly fatal) immediately followed a violent emotion. Very rarely an internal inflammation has been the apparent cause of tetanus. The inflammation has been of a serous membrane, pleura, pericardium, or peritoneum in most cases, and has been produced by cold. Intestinal worms have occasionally been found in the bodies of those who have died of the disease, but it is uncertain whether the malady was due to the irritation thus produced, or whether the coincidence was accidental. Lastly, idiopathic tetanus has occasionally come on without any condition being discovered to which it could be ascribed.

Puerperal tetanus is frequent in hot countries, and is perhaps the most terrible form of the disease. It is said that in Bombay, in three years, 232 women died from this cause. Fortunately, it is very rare except in the tropics. Of fifty cases in temperate countries, of which I have been able to find some particulars,* it occurred in eighteen after abortion; in thirty-two after labor at or near the full time. The cases were spread over the child-bearing period of life, the youngest patient being twenty-two, the oldest forty-eight. Those after abortion occurred from the second to the fourth month of pregnancy. In most of the cases there was severe hemorrhage, and in seven it is expressly stated that the vagina was plugged, a procedure that has been thought to influence the occurrence of the disease. The interval between the abortion and the first symptoms varied from five to thirteen days, and in two-thirds of the cases was from five to ten days; in no case less than five. In two cases instruments had been used to procure abortion, and metritis had resulted. In only one case was the tetanus apparently excited by exposure to cold, on the tenth day; the first symptoms occurred on the eleventh.

Of the cases after labor, the particulars of the labor are recorded in twenty-six cases, and in only three was there no abnormal condition during or after parturition. The most frequent accident was adhesion of the placenta, which existed in nearly half the cases (twelve). Metritis occurred alone in two, and in association with pharyngeal diphtheria in one. In only one case was the forceps used; in one there was placenta prævia and turning was necessary. In four cases the disease was apparently excited by a subsequent exposure to cold; and in one it succeeded a secondary hemorrhage, two weeks after delivery, for which the vagina was plugged. The interval before the appearance of the symptoms (noted in twenty-seven cases) varied more than in the cases after abortion, but in nearly half (twelve cases) it was from five to seven days (inclusive). In one-third (nine cases) it came on during the second week.

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* Including sixteen of those collected by Simpson (*Edin. Med. Journ., 1854*). In several cases it has followed the Cæsarean section, but these are not included.

In three cases, which occurred after fourteen days, a secondary exciting cause could be traced (diphtheria and metritis, cold, secondary hemorrhage). On the other hand, the interval was once only three days and once four days, and in one exceptional case (without albuminuria) symptoms of tetanus came on during labor, ceased for three hours after delivery, then recurred with such severity as to cause death in an hour.*

Tetanus neonatorum is fortunately a rare disease in this country, but, like other forms, it is exceedingly common in certain tropical countries, especially among the colored races. In Demerara, at one period, one-half of the negro children died from this cause. But it is endemic, also, in some localities far removed from the tropics, as in the notorious instance of Heimaey, an island near Iceland, where the population at one time was kept up only by immigration, almost all the children dying from this cause. In these circumstances, it is probable that the disease is ultimately due to insanitary conditions, since the employment of well-arranged lying-in hospitals has caused the affection, formerly prevalent, to disappear.† The liability to tetanus in newly-born children seems to be uninfluenced by sex; boys suffer only a little more frequently than girls.‡ The disease is usually ascribed to the influence of the umbilical wound, which is frequently inflamed; sometimes arteritis extends from it along the cord.§ The navel may, however, be healthy in appearance, both during life and after death. In hot climates (*e. g.* India, according to Wallace) tetanus often results from circumcision, being far more common during the second and third week of life among Jews and Mohammedans than among Christians.|| Occasionally (as in other traumatic cases) an exposure to cold apparently excites the disease. Tetanus

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* Curtis Smith, *Philadelphia Med. Reports, 1873.*

† Of three successive children of the same mother, the first two, born in one house, died of tetanus; the third, born in a different house, did not suffer (Salzmann, *Wurt. Cor.-bl., 1885*). Of course the change of residence may have involved many differences.

‡ In Ireland, of 371 deaths from tetanus, under five years, between 1841 and 1851, 219 were of boys and 152 of girls. It is probable that all these cases were of tetanus neonatorum, since, as already stated, the disease is otherwise practically unknown under five years.

§ Two cases, due apparently to umbilical ulceration, one in a child three weeks old, are described by Godlee and Williams, *Med. Times and Gaz.*, December 27th, 1884.

|| Wallace, *Lancet, 1882,* August 12th.

neonatorum usually begins between the third and seventh day, occasionally not till the second week, very rarely later.*

SYMPTOMS.—Whatever be the cause of tetanus, the symptoms, as a rule, are the same. Very rarely vague pains in the head, the epigastrium, or, in traumatic cases, at the seat of the wound, have preceded the onset. The first symptom is usually some sense of stiffness on movement of the neck or jaw, sometime difficulty in swallowing, or stiffness in the tongue. The patient thinks that he has got a stiff neck from sitting in a draught, and if, as is often the case, the symptoms have actually followed on exposure to cold, the patient's impression may be shared by his medical attendant. But in the course of a few hours, or at most of a day or two, the difficulty in separating the jaw becomes greater, and is clearly due to increasing rigidity of the masseters. With this there is also more stiffness in the neck, and the head is slightly bent backward from the preponderance of the spasm in the extensor muscles. As the rigidity in the neck increases it passes down the spinal muscles, until the vertebral column becomes over-extended in what is called "opisthotonos." The legs may also become extended and rigid, but the arms are little affected. Sometimes opisthotonos comes on at the same time as the rigidity in the jaw.

As the rigidity increases it involves the facial muscles. Apparently all the facial muscles are affected, but those which are strongest overcome the others, and impress on the countenance the effects of their contraction. Thus the eyebrows are raised by the frontalis, while the ocular fissure is lessened by the orbicularis. The angles of the mouth are drawn outward and a little downward, and the upper lip is pressed against the teeth, in what has been termed the "risus sardonicus."†

The tonic spasm or rigidity occasions at first little discomfort beyond a feeling of stiffness, but as it increases a pain is felt in the rigid muscles. Soon, however, paroxysms of spasm occur, at first slight, but gradually increasing in severity, and cause distressing cramp-like pain. The paroxysms are usually brief, lasting from five to fifteen seconds, and the spasm is greatest in those muscles which are the seat of the tonic rigidity. Thus the posture produced by the latter is increased during the attacks. The head is drawn back, the spine is arched, the legs rigid, and the feet extended, so that the patient, in a severe paroxysm, may rest on the head and heels. The peculiar expression of the face is exaggerated during the attacks; the jaw is firmly closed, and sometimes the tongue is bitten from being caught between the teeth when the paroxysm comes on. Other muscles, however, are involved, although in less degree. The thorax may be fixed and the glottis closed, while the face becomes livid, in consequence of the interference with respiration. The abdominal muscles are also contracted and hard, like boards, while the arms, though free from rigidity at the elbow and hand, may be fixed to the thorax during the paroxysms. In addition to the cramp-like pain in the muscles, severe and very distressing pain is frequently felt in the epigastric region, darting through to the back. It may be an early symptom, and is supposed to be due to spasm in the diaphragm. During the paroxysms this pain is intensified. From the violence of the muscular action the surface is covered with sweat, and during the paroxysms perspiration may pour in streams from the body.

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* Tetanoid spasms, commencing within twenty-four hours of birth, are probably due to meningeal hemorrhage (see p. 801). Marion Sims maintained that tetanus may be produced by displacement forward of the occipital bone, irritating the medulla. This displacement, he said, is physiological during birth, and is maintained afterward, in these cases, by the child lying on its back, with the head resting on the arm of the nurse; the symptoms may be removed by keeping it on its side, first one, then the other, for a few hours (*American Journal of Medical Sciences*, April, 1846; July and October, 1848). Evidence in favor of this opinion has been brought forward by Wilhite (*ibid.*, 1875, p. 375), and by Hartigan (*ibid.*, Jan., 1884); but these authors are manifestly in error in believing all cases of tetanus neonatorum to be thus produced, and thus remediable. Indeed, the correct statement of the facts, if their view is correct, is not that tetanus is thus produced, but that tetanus neonatorum is less common than is supposed, and is simulated by the effects of compression of the brain.

† From a plant Sardonia, so called because it grows in Sardinia.

Among the variations in the symptoms it may be noted that in extremely rare cases the spasm commences in the limb injured. Occasionally the stiffness in the throat precedes that in the jaw, and may be described as "sore throat." It has been known to continue for a week before the rigidity of the masseters was sufficient to prevent inspection of the throat. Usually the spasm is equal in the two masseters, but in one recorded case it commenced on the side that had been exposed to a draught (Harris and Doran). The spasm is always more extensive than the posture suggests; the muscles which are most powerful determine the attitude, but their weaker opponents participate (perhaps equally) in the spasm. Thus the muscles that depress the jaw become rigid, as well as those that raise it. The jaw is sometimes fixed by both sets of muscles without being quite closed, and in one case the jaw remained widely open during a paroxysm.

Opisthotonic spasm is the rule, to which the exceptions are few. Rarely the trunk is bent forward, from predominant cramp in the abdominal muscles and other flexors of the spine—"emprosthotonos." Still more rarely there is slight lateral flexion, "pleurothotonos," or the trunk and neck are rigid in a straight line, "orthotonos." These irregular forms seem to be rather more frequent in cases of idiopathic tetanus than in the traumatic form. In a case recorded by Treves, considerable pain in the abdomen accompanied the emprosthotonos and shifted to one side as the spasm became pleurothotonic.

The tonic spasm, although constant, usually varies in degree, and is occasionally, in slight cases, intermittent. Rarely there are no paroxysmal exacerbations, still more rarely there are attacks of spasm but no continuous rigidity. The paroxysms are sometimes very brief and frequently repeated, so as to resemble sudden shocks or a slow clonic spasm.

The muscles of respiration, in many cases, share the tonic spasm; the respiratory movements are then limited in range, and the breathing is short and hurried. During severe paroxysms the glottis may be also closed, respiration is arrested, and death from asphyxia sometimes results. It is said that the spasm is inspiratory in some cases, in others expiratory. The former is probably more frequent.*

During sleep, whether spontaneous or induced by chloroform, the spasm usually ceases, but it returns in undiminished force when the patient awakes. It has been thought, indeed, that after prolonged sleep from chloroform the violence of the spasms is increased in proportion to the length of time they have been kept in abeyance. Consciousness is unaffected, even to the last.

The pulse is increased in frequency, especially during the paroxysms, and is often very small. There is some reason to believe that the small size of the pulse is due to general vasomotor spasm. The temperature varies much in different cases. It is often normal, or nearly so, during the whole course of the disease, although in fatal cases there is usually a moderate rise (2° or 3°) toward the end. In other cases there is a continuous elevation of 3° to 5°, without evening remission, although frequent measurements may show a slight rise at each paroxysm, and a slight fall at each interval, so that the chart presents a serrated tracing (H. C. Wood). In other cases irregular variations occur, without corresponding variations in the symptoms. Lastly, in some instances, the temperature rises toward the end to an extreme degree, 108° or 110°, and, as Wunderlich first showed, the elevation may continue for a few hours after death, and even reach 114°. Some of the increase in body-heat may be due to the increased muscular work, but this cannot be regarded as the chief source of the pyrexia. Verneuil has pointed out that the cases in which the muscular spasm is greatest are rarely those in which the temperature is highest. Most of the fever is apparently of nervous origin. The hyperpyrexia is comparable to that met with in some cases of disease of the pons Varolii and upper part of the spinal cord, and may be associated with the fact that the earliest symptoms of tetanus proceed from this region.

The urine is usually scanty and high colored, perhaps partly in consequence of the loss of water by the skin. The amount of nitrogenous matter excreted (urea, kreatinin, etc.) is not increased, even in pyrexial cases, a striking difference from most febrile diseases (Senator). Micturition may be interfered with by the spasm; sometimes there may be actual retention. The bowels also are usually confined, in some cases in very obstinate degree.

VARIETIES.—In their developed symptoms, cases of tetanus present little variety, but the early aspect of the case differs according to the seat of the spasm. There is, however, one form that presents considerable variation from ordinary type; on account of the distribution and character of the symptoms, and also on account of its cause, it has been distinguished by Rose as " cephalic tetanus " (Kopftetanus). It results from wounds on the head, chiefly in the region of the fifth nerve. In many the forehead has been the part injured.* The chief peculiarity is that initial trismus is associated with paralysis of the face on the same side as the injury. In many cases there is also a peculiar pharyngeal spasm on swallowing, often accompanied by respiratory spasm. This symptom resembles the spasm that occurs in hydrophobia, and hence the variety has also been termed "tetanus hydrophobicus." The facial palsy involves all parts, just as in disease of the facial nerve, but its cause is unknown. It is often the first symptom. Occasionally there is slight contraction in the face, especially in the orbicularis palpebrarum, or there may be a slight sense of " numbness " or tingling in some parts of the face, sometimes on both sides. There is no degenerative reaction during life,† and no disease of the nerve has been found after death ; hence it is presumed to be of reflex origin. We have seen (p. 620) that irritation of the fifth nerve may cause paralytic ptosis, and it is worthy of note that in one recorded case ptosis formed part of the symptoms of this disease.‡ Of seventeen recorded cases, five have recovered, between the ages of 2½ and 25. All the cases hitherto met with over 25 (which have occurred at various ages up to 52) have been fatal. In the cases that have recovered the tetanic spasm and facial palsy have very slowly passed away, the symptoms continuing altogether several weeks. Death has usually occurred in the course of the second or third week.

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* According to Richet (*Société de Biologie*, March 4th, 1876), in the inspiratory spasm the glottis is open, in the expiratory spasm it is closed, and the latter entails more interference with circulation, and more danger of death from asphyxia, than the former. But in the case recorded by Harris and Doran (*Path. Trans.*, vol. xxxi) there were severe paroxysms of inspiratory spasm, and the patient died asphyxiated at the end of the second day.

Other varieties depend on differences in the course of the disease and in the distribution of the early symptoms. In the most severe cases, the spasm in the trunk may come on with that in the jaw ; paroxysms occur almost from the first, and the patient may die in two or three days, sometimes in a few hours. On the other hand, the rigidity in the jaw and neck may exist alone, for several days, even for a week, and the tetanic spasms on the trunk slowly supervene. In extremely rare cases transient spasm of the neck and jaw may be the only manifestation of the disease, the *abortive form* of Kussmaul.

In cases that recover, the disease always ends gradually, passing into a chronic stage. The paroxysms of spasm, at the end of one, two or three weeks, become slighter and less frequent, and ultimately cease, while the tonic rigidity continues, slowly lessening in degree. It disappears first in the parts affected last, and endures longest where it commenced, in the neck and jaw. At last it is felt only occasionally, and finally ceases. Very rarely the tonic spasm passes off, while the paroxysms continue. It does not appear that tetanus leaves behind it any special tendency to recurrence. At any rate, second attacks are at present unknown.* Nor have sequelæ (paralysis, etc.), been hitherto observed.

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* A few instances have been recorded in this country, as by C. J. Bond (*Brit. Med. Jour.*, Nov. 10th, 1883) and Nankivell (*Lancet*, July 14th, 1883).

† Bernhardt, Remak.

‡ Sereins, *L'Un. Méd.*, No. 173, 1886. The case is evidently an instance of this disease, although it is not described as such.

The *duration*, in cases that recover, varies from two or three days in the most trifling and, unhappily, most rare form, to six weeks or two months in those that are as severe as is consistent with life; it is usually from three to five weeks. In fatal cases the limits of duration also vary, although less widely, from a few hours to four weeks. Most cases, however, end in less than a fortnight, and if this period is over-lived, the patient's chance of recovery is fairly good.

The *mortality* of tetanus is extremely high, rivaled by few acute diseases, and excelled among its congeners only by hydrophobia. In traumatic tetanus the mortality is nearly 90 per cent., and seems to be rather greater in women than in men.† The influence of age on mortality is uncertain. Adequate statistics for its determination do not at present exist. It is greater after severe injuries than after those that were slight. When the symptoms do not commence until after ten days from the receipt of the injury, the proportion of recoveries is much greater than in those that commence during the first ten days, and seems to increase the longer the interval. When the disease begins within ten days, recoveries are extremely rare, and the mortality seems not to be influenced by the date of onset. Still, cases have been known to recover in which the disease commenced within three days from the time of the injury. Traumatic tetanus, although more common, does not appear to be more fatal, in hot climates. At Calcutta it is said to be about 83 per cent. (Wallace).

At present there are no adequate statistics from which to ascertain the mortality in idiopathic tetanus. In the cases I have collected, the mortality was 50 per cent. Although this may be below the actual mortality, the discrepancy is probably less than it would be in a similar collection of traumatic cases, because a larger number of cases are published merely because they are idiopathic, not because the patients have recovered.‡ In hot climates, idiopathic tetanus is usually stated to be more fatal than the traumatic variety, but the statistics of Wallace give a mortality among males of 56 per cent.,§ nearly the same as that just given for temperate climates.

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* The case recorded by Ogle (*British and Foreign Med.-Chir. Rev.*, *1868*, Oct., p. 488) is inconclusive.

† It has been strangely understated by those who, disregarding the caution given long ago by Lawrie, have estimated the mortality from collections of cases which have been separately published, most of them *because* the patients recovered. For instance, the total mortality was estimated by Yandell (*Brain*, *1879*), from a collection of 449 cases, to be 46 per cent.! The 164 cases of traumatic tetanus contained in the consecutive series of Lawrie, and of Poland and Taylor, contain 140 males, of whom 122 died (87 per cent.), and 24 females, of whom 22 died (91 per cent.).

‡ Moreover, this conclusion agrees with that of the idiopathic cases in the series of Lawrie, Poland and Taylor. Of these, 11 in number, 6 died.

§ Wallace, statistics of tetanus in the College Hospital, Calcutta (*Indian Med. Gazette*, Jan. 1st, 1881). Of 93 males, 53 died. The statistics regarding females are not available, because puerperal cases are included in the idiopathic form.

The mortality in tetanus neonatorum is extremely high, probably at least as high as in traumatic tetanus. Of all forms, however, puerperal tetanus is that which is most frequently fatal. When it occurs after abortion, recovery is practically unknown. The only recorded case which had not ended in death was still in progress at the time of the report.* After labor, all the cases in which the disease could be referred only to the process of parturition have been fatal. Of three cases that recovered, in one eclampsia occurred before childbirth, and in the other two the tetanus was apparently excited by exposure to cold, in one four days, in the other three weeks, after parturition.

Causes of Death.—In about two-thirds of the fatal cases of tetanus, death occurs during a paroxysm, either from failure of the heart, or from asphyxia in consequence of the prolonged arrest of respiration. The cause of cardiac failure is supposed to be the increase of the intra-vascular pressure (partly by the compression of vessels by the contracted muscles, partly by vasomotor spasm) to such a point that the enfeebled heart is unable to contract. The severity of the strain to which the heart is exposed is shown by one case in which a degenerated wall gave way during a violent paroxysm.† In the remaining cases death occurs from exhaustion, or from some accidental complication.‡

PATHOLOGICAL ANATOMY.—Rigor mortis usually sets in early, and is well marked. It is indeed said that the spasm during life may pass into the rigidity of death, but in most cases brief relaxation occurs.

The lungs are usually congested, and there may be œdema, hypostatic pneumonia, local emphysema, and sub-pleural extravasation—direct or indirect results of the interference with the pulmonary circulation by the frequent and violent spasm. The heart is sometimes relaxed; more frequently it is firmly contracted, probably only from rigor mortis. The liver and spleen are usually anæmic. The kidneys may be pale or congested; sometimes they contain extravasations. The difference in the amount of congestion depends chiefly on the mode of death, whether from exhaustion or during a spasm which arrests the movement of the blood.

The muscles often contain small extravasations. Rupture of individual fibres may be found on microscopical examination, and occasionally a large muscle may be torn across by the violence of the spasm. Such rupture had only been seen in the flexors of the trunk and hip (rectus abdominis frequently, psoas rarely§), which are contracted and at the same time are stretched by the more powerful spasm in the extensors. The fibres, under the microscope, usually present a normal appearance, but occasionally granular degeneration is seen, or there is a tendency to split up longitudinally (Bowman). Chemical analysis has shown that they contain more water than normal, and less albuminous material, but the alcoholic extract contains more nitrogenous matter and a substance containing phosphorus—lethicin (Danilewsky).

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* A case described by Lawrie in a letter to Simpson, and published by the latter, loc. cit.

† Duclaux, *Bull. de Thérapeutique, 1877*, Sept. 30th.

‡ *e.g.* in a case recorded by Pitram (*Wien. med. Presse*, Nov. 1st, 1886) death was due to pulmonary embolism from a clot in the hypogastric vein, the formation of which was supposed to be due to the compression of the vein by the muscular spasm.

§ Whynne Foote, Earle.

In traumatic cases, the wound may be in almost any condition, healthy or unhealthy, in process of normal cicatrization, or even actually healed. Perfect cicatrization is necessarily rare, on account of the period at which tetanus usually commences. Otherwise no state of wound is sufficiently frequent to be of significance. The nerves of the part injured have presented, in the majority of cases, no abnormal condition that could be recognized even by the microscope. In some cases, however, distinct evidence of inflammation has been found, the nerve being swollen and reddened, and, in a few instances, ascending neuritis has been traced up the nerve, even as high as the cord—reddened nodular swellings, usually separated by normal intervals. Increase of interstitial tissue, with degenerative changes in the nerve fibres, is found in such cases. It is certain, however, that local neuritis is no constant part of the morbid appearances in tetanus. In puerperal tetanus there is no constant abnormal appearance in the uterus, and its condition is usually perfectly natural. In some cases metritis, and decomposing remnants of the placenta, have been discovered.

In newly-born children, as we have seen, the umbilicus may be inflamed, and an arteritis umbilicalis may be traced along the cord within the abdomen. The adjacent peritoneum is often also inflamed. In some cases, however, these parts are normal.

In the brain and spinal cord the only constant changes are distention of vessels, large and small, and the presence of minute hemorrhage, such as is met with in all cases of death from convulsion, and probably the rupture of the vessels occurs chiefly in the act of death. In the cerebral hemispheres no morbid appearance is, as a rule, discovered on microscopical examination. In the medulla oblongata and spinal cord frequently the microscope has shown no other changes than those to be seen with the naked eye, and this in cases most carefully examined with the aid of modern methods of investigation.* In other cases slight changes have been found, but these have varied much. Some, however, are unquestionably of no significance, being frequent apart from all symptoms of disease. Such are an unusual amount of interstitial tissue in the white columns, spaces around the vessels, yellow pigmentation of the ganglion cells, amorphous exudation at the bottom of the fissures and an increase of nuclei around the central canal, sometimes obliterating it or extending laterally in the posterior commissure.

The microscopical changes which have been described,† and are possibly or probably connected with the disease (besides the vascular distention and minute extravasations) are, increase in the nucleus-like corpuscles in the interstitial tissue and around the vessels, chiefly in the gray substance; areas of "granular disintegration," sometimes amounting to actual cavities containing amorphous or finely granular material, chiefly found in the gray matter; irregular areas of carmine-staining amorphous "exudation" in the gray matter and posterior columns, and various changes in the large ganglion cells, swelling with indistinctness of the outline of the cell and of the nucleus; shrinking of the cells; apparent destruction of the processes. Similar changes have been found in a few cases in the medulla oblongata, but slighter in degree. These changes when found have presented no uniformity in character or distribution, nor can any relation be traced between their position and the seat of the wound.

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* As in four cases examined by F. Schultze (*Neurologisches Centralblatt, 1882*, No. 6), two by Hadden (*Brain*, Oct., 1885), and five by Bowlby (*St. Bart. Hosp. Rep., 1884*).

† Chiefly by Lockhart Clarke, Clifford Allbutt, Ross, Doran, Harris, Dickinson, and Aufrecht.

Microörganisms have been searched for by many investigators, for the most part without success, although in some instances bacilli have been found.*

PATHOLOGY.—Anatomy, coarse and minute, affords us thus only negative help in an inquiry into the nature of tetanus. It is true that the morbid appearances in a few cases have been made the foundation for certain theories; one of these is, that tetanus is due to myelitis (Michaud, Hammond), a theory that is inconsistent alike with the usual pathological changes, with the clinical history of the disease, and with the symptoms that are known to be produced by actual inflammation of the spinal cord. The changes which have been thought to indicate inflammation are probably the result of the intense disturbance of the function of the cord, of which the symptoms afford sufficient evidence. That actual inflammation may *result* from intense functional activity, the pathological anatomy of hydrophobia seems clearly to prove.

As part of the theory of myelitis it has been supposed that there is a propagation of inflammation along the nerves to the cord, but this theory is irreconcilable, also, with the facts that neuritis is the rare exception, and that the symptoms never (practically) begin in the part of the cord which would be first invaded.

The common relation to wounds is the great fact in the causation of tetanus which any theory of its pathology must embrace. But the difficulty of tracing any relation between its occurrence and the nature, position, or state of the wound, has led a large number of pathologists to look for the cause of the disease in some altered state of the blood. In support of this they point to the interval which usually elapses before the symptoms of tetanus appear, the absence in most cases of nerve irritation, to the similarity of the spasms to those of strychnine poisoning, and to the fact that the disease may come on without external wound. These facts have led to the assumption that the cause of the disease must be some poison or ferment developed in the wound (Richardson), or zymotic germs derived from the atmosphere, and some experimental investigations (to be mentioned presently) have been thought to give support to this theory. There are, however, many difficulties in the way of this theory. The rapid development of tetanus in some cases, and the fact that it may occur when a wound has healed, seem to preclude the assumption that it is the result of a poison developed in the wound itself, while for idiopathic tetanus such an explanation has no place. The isolated occurrence of the disease, at any rate in this country (one patient in a ward, full of wounded, suffers and the rest escape), renders it difficult to conceive that tetanus can depend on any poison derived from without, entering either by the lungs or the wound. Moreover, to any theory that it depends on a blood change is opposed the fact that all attempts to communicate it by inoculation or the transfusion of blood have failed.*

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* By Monastyrski (*Petersb. med. Wochenschrift, 1885*, Nov. 23d and 24th), but in one only of four cases, and by Hare (*Brit. Med. Journ.*, Oct, 4th, 1884), and Rosenbach (*Arch. f. Chir., 1886*, Bd. 34, Heft. 2). On the other hand, Bowlby searched for them in five cases without success (*St. Bart. Hosp. Rep., 1884*).

It may be doubted whether, in the present state of our knowledge, any theory can be framed which shall embrace all the facts of the disease. Nevertheless, it is worth while to consider the probable significance of the chief facts in its history. Of the symptoms of the disease perhaps the most remarkable is the uniform manner in which the malady commences, by slow contraction in the muscles of the neck and jaw, irrespective of the position of the traumatic lesion. The spasm cannot therefore be a simple reflex effect of the peripheral irritation, for such effects are always first manifested in the same region of the body. The definite and uniform grouping of the muscular contractions points to the excitation of some preëxisting nervous mechanism.†

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* Arloing and Trepier, among other experiments, injected blood from a horse suffering from tetanus into the veins of another horse, without result. See also Hare (*British Med. Jour.*, Oct. 4th, 1884). The theory that the disease depends on an organized poison has lately been revived, chiefly in consequence of some experiments of Nicolaier, who succeeded in causing symptoms resembling tetanus in mice, guinea-pigs, and rabbits (but not in dogs), by introducing common garden earth beneath the skin of the back. The symptoms commenced one or two days afterward, and the animals quickly died. Heating the mould to 190° C. destroyed its influence. Bacilli were found in abundance at the inoculation wound and in the spinal cord, but not in the blood or other organs. Inoculation with the pus of the wound often communicated the disease, and a watery extract of the earth was sometimes successful; so were some of the results of culture of the bacilli, although pure culture could not be effected. Nicolaier suggests that the symptoms were probably due to some strychnine-like poison produced by the microörganisms (*Deut. med. Wochenschr.*, *1884*, No. 52). Rosenbach (*Arch. f. Chir.*, *1886*, Bd. 34, Heft 2) found organisms in man something like those described by Nicolaier, but failed to produce any effect by inoculation. The significance of the facts ascertained by Nicolaier does not seem to me so great as is assumed by some writers on the subject. From the fact that certain poisons tetanize, to the proof that tetanus depends on a poison, is a much longer step than at first sight appears.

† It is perhaps worth while to note, in connection with this point, that there is one action, our familiarity with which should not blind us to its singular nature—in which there is brief tonic muscular spasm, distributed nearly as in the early spasm of tetanus. A yawn consists of a slow inspiration and depression of the lower jaw, followed by brief tonic spasm of the

To what part of the nervous system is the disturbance to be referred? The early symptoms are in the region supplied by nerves that arise from the highest part of the spinal cord, medulla oblongata, and pons, and must be referred to centres in this region. The same indication is afforded by the coördinated respiratory spasm that forms so conspicuous a feature in the attacks, by the vasomotor spasm, by the occasional hyperpyrexia, by the phenomena of cephalic tetanus, and by such cases as one recorded by Silbermann, in which a fall on the back of the head was followed in a few hours by symptoms of tetanus, accompanied by striking cyanosis and dyspnœa. Permanent trismus sometimes results from organic disease of the pons Varolii, and general tonic spasm, especially marked in the legs, is not uncommon in hemorrhage in this situation. It is only in the later stage of tetanus that there is evidence (in increased reflex excitability) of a morbid state of the lower spinal centres, which, secondary in point of time, is doubtless secondary in its production. The symptoms point, therefore, to the pons and medulla as the seat of the chief disturbance of nerve function in the disease.

The definite and uniform character of the early symptoms seems to exclude any form of inflammation. This conclusion is, as we have seen, suggested also by the results of microscopical examination. The symptoms point to a primary disturbance of function in a definite or nervous mechanism, and its immediate cause must be sought in the nerve cells concerned, not in any abnormal condition of the vessels, the effects of which are always random and variable, not definite and constant. Modern doctrines would teach us to regard the over-action as the result and expression of a diminished resistance to the evolution of nerve force by the cells.*

How the causes of tetanus bring about this result is a question to which a definite answer seems at present impossible, but we may endeavor to ascertain the probable direction in which the answer is to be looked for.

In spite of its difficulties, the theory that traumatic causes act by nerve irritation remains the most probable, and the probability is emphasized by the occasional presence of neuritis. But the nerve irritation, if it exists,

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muscles of inspiration, of the depressors and elevators of the lower jaw, of some of the facial muscles (so that the angles of the mouth are drawn outward), and of the muscles of the neck, so that the head is drawn a little backward. If the individual is standing the spasm may be felt to pass down the back, and the spine to be slightly arched. The muscles involved are those which are affected in the early stage of tetanus. Moreover, most persons have probably discovered (under the exigencies of social life) that the jaws can be approximated during the proceeding. The resemblance to a tetanic spasm then becomes still closer, and is almost complete if, in "stretching," a tonic spasm extends the legs and arms. In tetanus there is rigidity of the depressors of the jaw, and although it is usually overcome by the spasm in the more powerful elevators, the jaw has been sometimes fixed with a space between the teeth, and in one instance, during a paroxysm, the jaws were as widely separated as in an ordinary yawn. Lastly, in one or two cases, persistent yawning attended the onset of the disease. It would seem, therefore, probable that the nervous arrangements which subserve the act of yawning are concerned in the production of the symptoms of tetanus.

* Handfield Jones, Foster, Ringer and Murrell (see *Med.-Chir. Trans.*, vol. lix, 1876).

must be peculiar in its kind and in its influence. It must be peculiar in its kind, because the ordinary symptom of nerve irritation, pain, is not pronounced, and because persistent painful irritation is common without tetanus. It must be peculiar in its influence because this is not exerted on the proximate centres, but on a distant part (the medulla), and because its effect is gradually to bring the nerve elements into a peculiar condition of unstable equilibrium, which after a time culminates in discharge, at first constant and slight, but as it increases becoming paroxysmal, and excitable by peripheral impressions. Ultimately the instability extends to the lower centres, through which those act which are first affected.

In connection with the theory of nerve irritation a case recorded by Terrier * deserves especial note. An injured toe had become gangrenous, and was amputated. Trismus, absent before, was present a quarter of an hour afterward, and the patient averred that the pain of the operation distinctly made his jaw muscles contract while the amputation was being performed. He died from the tetanus in two days. Here it would seem that the preceding irritation brought the centre into a condition so unstable that its discharge was produced by the additional stimulus. Verneuil has also noted that surgical interference with a wound may apparently excite tetanus. The effect of the operation in such cases seems produced in other traumatic cases by exposure to cold. The irritation of the wound brings the centre into an unstable condition in which a slight degree of surface chill will cause discharge. For instance, in the case of tetanus after ovariotomy, recorded by Harris and Doran, the contraction commenced in the masseter, on the side turned toward a window, the weather being very cold at the time. In other cases again, as that mentioned on p. 1028, it would seem that psychical influences may aid in bringing about the morbid state.

The way in which cold acts is, however, as mysterious as the traumatic influence. It has been supposed to cause congestion of the centres, but this is a pure hypothesis unsupported by any close analogy. That the surface chill causes a peculiar nervous stimulation is more probable, since we know that it causes other effects through the agency of the nervous system, stimulating, for instance, the vasomotor centre to constrict the vessels of the skin, and even the motor centres to produce the muscular spasm of "shivering," in which the muscles of mastication always take a conspicuous part. The action of cold through the nervous system is also in harmony with the probable influence of wounds. It is far more probable that both these causes, so often associated, act in the same manner, than that one acts through the nervous system, and the other through the blood. In most idiopathic cases cold appears alone to be the agent in producing the morbid state of the nerve centres, and it probably acts by the same mechanism as in the cases in which its influence is conjoined with that of a wound. It is noteworthy, however, that whether acting alone or conjointly, the effect of cold is always rapidly produced. Rarely more than twenty-

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* Terrier, *Gaz. des Hôp., 1874.*

four hours pass before the appearance of the first symptoms. It seems, moreover, certain that the effect produced in most cases by wounds and cold may result from a simple concussion of the nerve centres. There is no difficulty in conceiving that a direct mechanical disturbance should bring about the result which in other cases results from peripheral irritation.

Lastly, it may be noted that in tetany we have a disease consisting of tonic spasm, and sometimes caused by cold, in which there can be no question of a toxic cause.

Why the disturbance, however excited, should take a special course and be of such profound intensity, whether there exist other predisposing influences, and what or how the nerve irritation differs from that in other diseases, are questions to which no answers are as yet forthcoming. But in the answers alone can we find the real pathology of the disease.

DIAGNOSIS.—The symptoms of tetanus are so peculiar and so pronounced, that the diagnosis rarely presents any difficulty, except in the earliest stage of cases that commence insidiously. Under such circumstances, the stiffness of the neck may be mistaken for muscular rheumatism, the more readily if the symptom has followed exposure to cold. But the existence of rigidity in the muscles of the jaw, never present in simple rheumatism and rarely absent in tetanus, should at once arouse suspicion. In the rare cases in which difficulty in swallowing precedes trismus, the nature of the case can only be suspected from the fact that there is no local affection to account for the symptom, and no evidence elsewhere of paralysis; and the symptom is the more suspicious if there is also stiffness in the movement of the tongue.

The characters of the developed disease resemble those of strychnine poisoning more than any other condition. A mistake is rare, but has probably been made in one or two recorded instances. In strychnine poisoning the symptoms never commence by trismus; they come on and develop in a more rapid manner than has ever been seen except in traumatic cases, in which there is the wound to assist the diagnosis. The reflex excitability is an early symptom in strychnia poisoning, but is late in tetanus, and the severe epigastric pain of the latter is absent in the former, in which, also, there are often collateral circumstances to suggest poisoning.

In hydrophobia there is no initial rigidity in the jaw or elsewhere. The first paroxysms are of respiratory spasm, excited by attempts to swallow. These may be present, however, in the "hydrophobic" form, and in it swallowing is difficult, but the wound on the head and the facial palsy should prevent error. Cases of hydrophobia in which there are tetanoid spasms in the later stage of the disease have been confused with tetanus, but attention to the initial symptoms will prevent error. These spasms are merely an excessive development of those that are frequently met with in hydrophobia, and do not indicate that the two diseases coexist, as has sometimes been erroneously imagined.

In hysteria, tetanoid spasm is extremely rare except as part of a convulsive attack, and then its nature is sufficiently conspicuous. But trismus, causing persistent closure of the jaws, is common in hysteria. It may succeed a convulsion, and last until another, or it may come on without obvious cause, continue for a few hours or days, and then suddenly vanish. It is prone to recur, and this character, the suddenness of onset, its complete degree, the absence of rigidity in the neck, and the presence of other symptoms of hysteria, will rarely leave any doubt as to its nature. It should be remarked that the symptoms of tetanus, coming on after an injury or exposure to cold, must not be treated lightly because they occur in a hysterical person, as is illustrated by the case mentioned in the note on p. 1028.

In tetany there is widely-spread tonic spasm with paroxysmal exacerbations, but its distribution is characteristically different. The limbs are most affected toward the extremities, the arms more than the legs, the hands most of all, and trismus is a late and not an early symptom. In each of these respects the condition in tetanus is the reverse. Even in the most acute and violent cases of tetany a mistake can hardly be made, especially if attention is paid to the peculiar posture of the hands.

In many cases of tetanus there is considerable difficulty in determining whether it is to be regarded as traumatic or idiopathic. They are the cases in which tetanus is apparently excited by cold in a person who has had a trifling injury, perhaps some time before. The question is fortunately not of any great practical importance, and each case must be judged on its own merits. For scientific purposes we must include such cases in the traumatic variety.*

Prognosis.—The statistics of the mortality in tetanus show how grave the prognosis is in every case, and it is still grave, however slight the initial symptoms may be, or trifling the injury which has produced it. But it is distinctly worse if the injury is severe than if slight. After the compound fracture of a limb, for instance, or after labor, recovery is extremely rare. The chance of recovery is also less if the first symptoms occur before the tenth day from the receipt of the injury. After the first ten days the prognosis is better the longer the interval. It is worse when the spasm quickly extends to the trunk, better if trismus exists alone for several days. After the first four or five days the prognosis improves with the duration of the disease. It is doubtful whether the prospect of recovery is materially influenced by sex or age.† Adequate statistics to determine this point are not at present forthcoming. Previous intemperance lessens the chance of recovery, and so also does inability to swallow, and considerable elevation of temperature. The concurrence of cold with injury as a cause does not influence the prognosis. Under the most favorable circumstances, in cases, for instance, which occur more than a fortnight after the receipt of a trifling injury, the probability of recovery is not more than equal to that of death. On the other hand, no case of traumatic tetanus is absolutely hopeless. Cases occasionally recover under all conditions of cause and character.

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* By this means we run least risk of error. It is certain, however, that some proportion of cases of idiopathic tetanus will have a recent scratch or cut or bruise, without this being concerned in the production of the disease. Cases in which the origin is thus uncertain have been termed both "idiopathic and traumatic," an incompatible designation which would be avoided by calling them "rheumatic and traumatic."

† Regarding sex, compare the mortality given on p. 1036.

The prognosis in idiopathic tetanus is, in temperate climates, a little better than in the traumatic form. In hot climates it is regarded as even more grave. In tetanus neonatorum the prognosis is nearly the same as in the traumatic form in adults (provided the children are not exposed to powerful insanitary conditions), and here, also, the prognosis is better the longer the interval after birth before the symptoms appear. After abortion and labor, the chance of recovery is only appreciable when the disease is distinctly excited by cold.

TREATMENT.—For tetanus, as for most acute diseases, no specific remedy is known. But it is a disease of limited, though variable, duration, and if the patient can be kept alive until it is over, he recovers. Moreover, there is reason to believe that the intensity of the disease can often be lessened by treatment, but the variable severity of the affection renders it very difficult to assess the influence of the remedies employed. There are three elements in the treatment of tetanus: general management, operative treatment in traumatic cases, and the endeavor to relieve the symptoms by the use of drugs.

In general management, rest and food are the two essential measures. All possible sources of peripheral irritation should be avoided. The patient should be kept absolutely still and the room darkened. Liquid nourishment should alone be given, even if the trismus is not complete. In most cases, by patience, a fair quantity can be taken through the closed teeth; sometimes there is a space between the teeth, and a tube can be introduced through which the food may be sucked. When this is impossible, sufficient relaxation of the spasm to permit of food being given may be obtained by the inhalation of chloroform, or liquids may be injected into the œsophagus by a long catheter passed through the nose. If this brings on spasm, it is better to employ peptonized injections into the rectum. A tooth has been extracted to permit feeding through a tube, but the desirability of this is doubtful, for the reason to be mentioned immediately. In infants the catheter passed through the nose has usually been employed.

The surgical measures which have been adopted in traumatic cases comprehend amputation, excision of a cicatrix or wound, neurotomy, and nerve stretching. Of all these it may be said that in most cases in which they have been employed they have apparently exerted no beneficial influence on the course of the disease. Indian experience of these measures is at least as unfavorable as that of England.* A curious case has been described by Reichert, in which stretching of both sciatic nerves was followed by improvement and ultimate recovery. The cause of the tetanus was a bite on the back by a horse, but the patient was an epileptic. The modus operandi of the operation is difficult to understand.†

Whatever influence the local nerve irritation has in causing the disease, the latter, once set up, appears to run an independent course, unmodified by the removal of the former. It must also be remembered that the operation for the removal of the old irritation, in itself constitutes a new traumatic influence, considerable in the case of an amputation, slighter but still present in the operation on nerves. The possible prejudicial influence of a new irritation is illustrated by the case quoted on p. 1042. Hence it is reasonable to conclude that surgical measures are only justifiable where there is evidence of a considerable degree of local irritation, so that it is probable that the new irritation will be less than that which it removes. It may be said generally, however, that when the spasms start from the wounded limb, local treatment should be adopted. Most of the cases in which local surgical treatment has had a distinct and immediate effect have been of this character. Amputation is the only measure when the wound is in a very unhealthy state, with much irritation of the structures, and when the nerves from it are not accessible. Neurotomy and nerve stretching should be employed only when there is evidence of irritation of nerves that are accessible above the wound. In one case, for instance, a small nerve in a wound was tender, and pressure upon it brought on severe tetanic spasms. The nerve was excised, and the spasm ceased. In another case tetanus came on after a wound was healed; the cicatrix was tender, and pressure on it excited the spasm, which ceased after the cicatrix had been excised. For the reasons mentioned above, it seems desirable to avoid, as much as possible, all treatment which may involve local irritation, such as the extraction of a tooth for the purpose of feeding. If hypodermic injections are employed, a fine, sharp, steel needle should be used, and the injections made into the back of the forearm, where the skin is thin, so as to cause as little nerve irritation as possible.

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* Wallace, loc cit.

† *Bayer. Aerztl. Intell.-bl., 1885,* No. 5.

There are few diseases for which so many and such varied drugs have been employed as in the treatment of tetanus, but hitherto not one has been found to exert a powerful influence on the disease, not one that does not usually fail when the disease is severe, and frequently when it is moderate in intensity. Recovery has, in most cases, been due to the character of the attack, not to the treatment employed. Nevertheless, there is reason to believe that drugs have, in many instances, helped recovery, and in some cases have actually turned the scale and saved the patient's life.

Of the agents employed there are some that give temporary relief to the spasm, and are used to give rest or avert death, but which do not influence the course of the disease. The most efficient is the inhalation of chloroform. With complete narcosis the spasm passes off, but it returns when the influence of the chloroform is over. Ether has a similar action, but is less convenient. Nitrite of amyl has been said to relieve spasm more speedily than chloroform, and H. C. Wood has recommended it as a most valuable agent for averting death during a paroxysm, but at Guy's Hospital, when employed in quantities of ♏iij to ♏v, it was found that the spasm became more intense at first, although slighter afterward. The continuous inhalation of chloroform does not modify the course of the disease, nor has any benefit resulted from attempts to charge the air of the room with the vapor of chloroform.*

Sedative drugs have been employed in the treatment of tetanus in two ways, occasionally to procure sleep, and continuously to lessen the severity of the spasm. In severe cases it is rare that a marked effect is produced by any drug, but in cases of moderate severity distinct amelioration has been obtained by many agents. Bromide of potassium, given at frequent intervals in large doses, sometimes has a distinct influence, since the spasm has been observed to increase when it is discontinued, and lessen when it is resumed (Southey). It may be given every one, two, or three hours, but the daily quantity should not be less for an adult than four or six drachms. It may, perhaps with advantage, be injected into the rectum with food. Nothnagel has found that it tends to cause ascending contractions, and thus an injection is carried higher up and is more rapidly and more completely absorbed.

Chloral hydrate has been largely substituted for the administration of chloroform by the mouth, although it does not remove severe spasm so completely as the inhalation of chloroform. It may be given at night to procure sleep, and frequently succeeds, or it may be given continuously, and has thus, in many cases, appeared distinctly beneficial. Indeed, it has been held to have more influence on the disease than any other drug, by a very large number of observers.† Large doses must be employed. Verneuil, for instance, who is a strong advocate of its use, would never give, to an adult, a smaller dose than ʒj nor less, in the day, than ʒiv, and he has sometimes given ʒiij at a dose and ʒvij in the day. It needs to be continued for a considerable time. In two cases which recovered, ℥viij were given in the course of a month. Chloral has been injected into the veins by Oré, but this is a dangerous proceeding, since it has caused extensive thrombosis in several cases (Lannelongue and others). In tetanus neonatorum, it has usually been given in doses of one grain, but this is probably too small. Opium and morphia have been largely used, the latter by subcutaneous injection. It is useful for the purpose of procuring sleep, in doses of a quarter or half a grain, which may be employed in addition to the continuous administration of bromide and chloral. The continuous use of morphia has not, on the whole, been distinctly useful so frequently as that of other sedatives.

Belladonna, atropine, Indian hemp and aconite have also been frequently employed. Under their use (as under that of all other drugs) recoveries have been occasionally observed, but on the whole their utility has been even less frequently recognizable than that of other sedatives. The same may be said of conium, lobelia, nicotin and tobacco, veratrum viride and gelsemium.*

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* Simonin used in this way 22 kilogrammes of chloroform, without effect.

† Schmidt (*Bayer Intell.-bl., 1885*, p. 329) records the recovery of four out of five cases treated with chloral, but so favorable an experience must be regarded as altogether exceptional and perhaps open to suspicion.

Next to bromide and chloral, Calabar bean has received most praise. The extractum physostigmatis has been usually employed subcutaneously, in doses of from one-third of a grain to one or two grains, or by the mouth one to four grains. Still larger doses have been sometimes given by the mouth, *e. g.*, seventy-two grains in twenty-four hours, and 1026 grains in forty-three days, in a case that recovered.† Toxic effects are not easily produced, and the pupil does not contract as it does in health, even when nausea and feebleness of pulse have been produced. Eilert has suggested that local effects may be lessened by the simultaneous injection of atropine. In infants from one-thirtieth to one-third of a grain has been given under the skin. Although a considerable number of recoveries have been recorded, a distinct effect on the severity of the symptom has not often been traced. Jaborandi and pilocarpine have been used in some cases, but their value is doubtful.

The alleged success of curara in hydrophobia has led to its use in tetanus, but seldom with success. Small doses $\frac{1}{100}$ grain do not influence the disease. In large doses, up to half a grain every hour, the muscular spasms become slighter, but life has rarely been saved. In one case it was pushed until the patient passed into a state of collapse and respiration ceased. Prolonged artificial respiration, with faradization of the phrenic nerve, revived the patient; the spasms remained absent for several hours, and then returned in slighter form, and the patient recovered. Spasm is probably lessened by the influence of curara on the intramuscular nerve endings, as it is not known to have any influence on the spinal cord. It appears only to avert death from spasm by bringing the patient equally near to death from paralysis.‡ Among other drugs which have been used, are iodide of potassium, carbonate of iron, arsenic, antimony, mercury, and strychnine. Cases have recovered in which each has been used, even, strange to say, the last, which distinctly increases the spasm.

External applications, chloroform, aconite, etc., to the affected part appear useless, but applied to the epigastrium they sometimes give relief to the severe epigastric pain. Cold to the spine (ice, ether spray) has been used without distinct influence on the spasm. Electricity (the voltaic current from the spine to the muscles) has been observed to lessen the spasm in trifling cases, but probably did not influence the course of the disease. Continuous warm baths have been occasionally employed since the time of Ambrose Paré. Zechmeister kept one patient in a bath for a fortnight, but the treatment, used in eight cases, did not influence the mortality, since seven of the patients died. It seemed, however, to moderate a little the intensity of the spasm. Bleeding was formerly largely used, and, opposed as the measure is to the therapeutic principles of the present day, it must be confessed that cases recovered and the flow of blood appeared in some instances to have a marked effect on the spasm. It has gone out of use too completely for its utility to be assessed. Transfusion of blood has been once employed, but without success (Sakler).

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* Four cases are said to have been successfully treated by veratrum viride and gelsemium, by R. B. Harris (*New York Med. Record, 1884*, July 12th).

† Watson, *Practitioner*, April, 1870. For another successful case, see Dougall, *Glasgow Med. Journ.*, March, 1885.

‡ Karg (*Arch. f. klin. Chirurg.*, xxix, p. 338) relates four cases in which the transient amelioration of the symptoms did not prevent death. He advises the simultaneous use of morphia. One noteworthy lesson from his cases is that if artificial respiration becomes necessary it is *not* facilitated by tracheotomy. Cases successfully treated have been recorded by Berckham (*Berl. kl. Wochenschr., 1884*, No. 48) and Gontermann (*ib.*, 1883, No. 44). In the latter case the tetanus followed an injury to the head. Nine injections were given in the course of a few days, each consisting of $\frac{1}{4}$–$\frac{1}{6}$ grain of curara, dissolved in 40 parts of water and 2 of spirit. He makes the reasonable suggestion that the activity of the specimen employed should always be proved by an injection in an animal.

On the whole, the drugs that seem to deserve most confidence in the treatment of tetanus are bromide of potassium and chloral. They may, with advantage, be combined, the influence of both being kept up continuously, or bromide may be given frequently and chloral in occasional hypnotic doses. If the violence of the spasm threatens death during a paroxysm, this should be, if possible, averted by the inhalation of chloroform or nitrite of amyl, but on the value of the latter for this purpose further observations are necessary. Lastly, it is of great importance, in the event of apparent death during a paroxysm, that an attempt should be made to restore the patient by artificial respiration. It is surprising that this measure has been so rarely adopted. Its importance is illustrated by a case recorded by Farrage.* During a severe paroxysm, heart and respiration stopped, the patient was apparently dead. Artificial respiration was employed and in five minutes the heart's pulsations could be again perceived. For two days the spasm was extremely slight; a most violent paroxysm then occurred, during which the patient died, no medical assistance being at hand. The remarkable freedom from spasm in this case, and one or two others on record, after revival by artificial respiration from apparent death, can only be ascribed to the influence of the arrest of the circulation on the morbid condition of the centre. This fact, and the evidence that the primary disturbance is in the medulla oblongata, suggests the desirability of trying the effect of ligature of the vertebral arteries, which Alexander has introduced for the treatment of epilepsy. The operation is severe, when both vertebrals are simultaneously tied, but in a case in which the danger was extreme, it might be justifiable.

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* *Lancet*, 1860, Sept. 18th.

TETANY.

The condition designated "tetany" is characterized by tonic muscular spasm or "contracture," affecting especially the extremities, symmetrical in distribution, and either paroxysmal or continuous. The spasm is often preceded and accompanied by sensory symptoms, tingling, formication, or pain.

The affection was first described more than fifty years ago, by Steinheim in Germany, and Dance in France, but, like so many other maladies, it was through the study and description of the disease by Trousseau that it became generally known. Trousseau called it "tetanilla," but the name "tetany," by which it is now universally known, was suggested by Lucien Corvisart in 1852.*

CAUSES.—Tetany is rather more frequent in males than in females, the proportion being as seven to six, but this relation does not obtain at all ages. It occurs at all periods of life, from infancy to old age, but is most frequent in early childhood and in early adult life. Of 150 cases that I have collected from various sources (including eight observed by myself) 142 are available for comparison on these points:—

Ages	1–4	5–9	1–9	10–19	20–29	30–39	40–49	50–61	
Males	26	5	31	23	9	4	5	4	= 76 Males.
Females	8	3	11	13	15	19	8	0	= 66 Females.
Total	34	8	42	36	24	23	13	4	= 142

Thus the disease is most frequent in infancy and in the second decade of life. More than half the cases occur during the first twenty years. In early childhood the disease is far more frequent in males than in females, but between the ages of twenty and fifty the liability of the sexes is reversed, and females suffer twice as frequently as males. Over fifty, on the other hand, the only recorded cases have been in males.

A neuropathic heredity can be traced in only a small proportion of cases, but that a family predisposition sometimes exists, is shown by instances recorded by Abercrombie, in which there occurred, at different times, in each of two families four cases, and in another family three cases.

Most of the male cases have occurred in the lower classes, and occupation is influential chiefly as involving exposure to cold and fatigue.

A direct exciting cause of tetany can be traced in at least three-quarters of the cases. The most frequent is diarrhœa, usually long-continued and exhausting, but sometimes acute and brief. It often coexists with other causes. Next in frequency are exposure to cold, especially conjoined with fatigue, acute disease, and lactation. A series of cases in women who were suckling led Trousseau at first to propose for the disease the name of "nurse's contracture." The comparative frequency of the disease in adult women is almost entirely due to the various influences of maternity. The tetany may also come on during pregnancy (usually during the second half) and after confinement. Other occasional causes are simple anæmia, prolonged muscular effort of various kinds, alcoholism, and sexual excess, especially masturbation.

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* Trousseau's account of the disease is accessible to English readers in the volume of lectures translated by Bazire. Other descriptions have been given by Riegel (*Deut. Arch. f. kl. Med.*, xii, 1863, 405); Weiss (*Volkmann's klin. Vort.*, No. 169); Buzzard (*Clin. Lect.*, p. 411); and Abercrombie ("On Tetany in Young Children," London, 1880).

In young children the indications of rickets are rarely absent, and in them tetany is evidently allied to the carpo-pedal contractions so common in that disease, and it is often associated with laryngismus stridulus and convulsions. In three recorded instances the disease was apparently due to intestinal worms (tæniæ), and ceased when these were expelled. It very rarely follows a fright, and seldom an injury, but there is one operation after which it is singularly frequent—excision of the thyroid. Weiss has reported thirteen cases of tetany due to this cause. Wölfler observed seven cases in seventy patients subjected to the operation. The whole thyroid was removed in each case. All the patients have been young females, still in the developmental period of life. The symptoms of tetany set in during the first ten days after the operation.*

In well-marked form, tetany is less frequently associated with hysteria than might be expected, but hysterical contracture sometimes assumes a similar form, and it may indeed be difficult to say whether a given case is to be regarded as tetanoid hysterical contracture, or as true tetany in an hysterical subject. The acute diseases, during or after which tetany has been known to come on, are typhoid fever, cholera, smallpox, rheumatic fever, measles, febricula, catarrh, and pneumonia. It has been met with very frequently in some epidemics of typhoid, usually during the latter part of the disease, after the sixteenth day, or during convalescence; in rare cases during the first week. It has also been met with in Bright's disease, and I have seen one case apparently due to lead poisoning.

Lastly, singular epidemics of tetany have been met with on the Continent. Some of these are apparently analogous to the occasional epidemics of hysterical convulsion. For instance, in a girls' school in France, in 1876, an epidemic occurred in which no less than thirty girls were attacked.† A still more singular epidemic occurred in 1846, in certain Belgian prisons. The prevalence of the disease in some outbreaks of typhoid also suggests its occasional dependence on some obscure epidemic influence.

SYMPTOMS.—As a rule, the peculiar symptoms in the limbs come on without premonitory nervous disturbance, but in rare cases they have been preceded by headache, pain in the spine, and malaise, and even vomiting not due to gastric derangement. The muscular spasm may be the first symptom, but is usually preceded for a few hours or days by sensory disturbance in the extremities, as "tingling" or "burning" sensations. The spasm almost always sets in suddenly in the hands, occasionally in both hands and feet at the same time, rarely in the hands only, and still more rarely in the trunk. The hands feel stiff and cramped, and then become fixed. The usual posture is with the fingers flexed at the metacarpo-phalangeal joints, extended at the others; the thumb is adducted and in contact with the index finger or is flexed beneath it, while the spasm in the thenar and hypothenar muscles renders the palm hollow. The fingers are sometimes pressed together, sometimes inclined toward the ulnar side. The position of the fingers is mainly due to spasm in the interossei muscles. The wrist is usually slightly flexed. The elbow may be free, but is usually in slight flexion. The shoulder muscles are rarely involved, but occasionally there is spasm adducting the arm. In a few cases the spasm in the hand muscles has been chiefly in the long flexor of the fingers, which are then flexed at all joints, and the fist thus closed. Still more rarely the wrist has been extended, and in two recorded cases the fingers were extended at all joints.

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* Weiss, *Anzeig. d. Wiener Gesellsch. Aerzte, 1883*, No. 31; Wölfler, *Verh. der Deut. Ges. f. Chir., 1883*, p. 36.

† Simon; Mattraits, *Thèse de Paris, 1877*.

The feet are extended at the ankle joint, and are inverted, being in the position of talipes equino-varus. The toes are strongly flexed; the knees are usually extended, rarely flexed. The thighs are sometimes adducted, rarely flexed.

In cases of slight or moderate severity the contracture is limited to the hands and feet, not infrequently to the hands. In severe cases the muscles of the trunk and head share in the spasm. The abdominal muscles become rigid, and there may be spasmodic retention of urine. Those of the back are less affected, but occasionally there is a slight degree of opisthotonos. The thorax may be fixed by spasm, which may even involve the diaphragm, and cause some difficulty in breathing, cyanosis, and even transient loss of consciousness. Sometimes the sterno-mastoids are rigid, and the head may be bent forward. More frequently the jaws are closed by spasm in the masseters, the angles of the mouth are drawn out, and the eyelids are half closed. Even the eyeball muscles may be involved, causing strabismus, convergent or divergent. The tongue is sometimes stiff, and articulation may thus be rendered difficult. The affection of the muscles of the face is usually only seen in severe attacks, but has been occasionally met with in attacks which were very slight.

The tonic spasm may render the muscles firm and hard, and fibrillary contractions are sometimes visible in them. Attempts to extend them usually cause much pain. Movement is interfered with by the spasm and in proportion to it, but there is no actual paralysis. When the contracture is slight, movements are merely rendered difficult and awkward; when it is considerable, all voluntary movement may be, for the time, impossible.

Slight contracture may be painless, but when the spasm is considerable there is severe cramp-like pain in the muscles, and sharp pains pass up the limbs, especially in the course of the nerves. When the spasm is continuous, the pain may be paroxysmal, probably in consequence of slight exacerbations of the spasm. The subjective sensations (tingling, etc.) which precede the spasm, may persist during the intervals, and may be accompanied by actual diminution of sensibility to touch or to pain, or both.

The spasm is usually paroxysmal. After continuing for a time, which varies from a few minutes to a few hours, and rarely to a few days, the contracture gradually passes off, to recur after a variable interval of hours or days. A feeling of stiffness may continue for a time. In some cases the spasm continues in slighter degree in the arms, rarely in the legs, in the intervals between the paroxysms. In other cases, again, in both children and adults, the spasm may continue in moderate degree for several days, without exacerbations or with merely an occasional trifling increase. Attacks of spasm may occur and even commence during sleep, and if there is continuous contracture this may persist during sleep, although usually in less degree. Abercrombie observed no change in the continuous spasm during an attack of measles which ended fatally. Attacks are sometimes excited by muscular exertion.

In the intervals between the attacks of spasm there is usually a remarkable increase in the excitability of the nerves and muscles in the parts in which the spasm occurs or is most intense. Percussion of the muscles, and especially of the nerves, causes a conspicuous contraction. This phenomenon may be well observed in the face, where a tap on the nerve will often cause a momentary contraction in all the muscles supplied by it. It is also marked in the limbs, in which an attack of characteristic local spasm may usually be induced by compression of the artery and nerves of the limb, as Trousseau discovered. The spasm comes on a few minutes after the commencement of the pressure. It is probable that the effect is chiefly produced through the nerves, although it is said that sometimes compression of the artery is effective when that of the nerve is not. Sometimes this phenomenon cannot be obtained. In children Abercrombie found the increased irritability invariably present in the face, and not in the limbs, in cases of continuous tetany of young children.

There is also a greatly increased electrical excitability of the nerves to both faradaism and voltaism. I have obtained contraction in the muscles of the face by applying to the nerve the voltaic current from a single cell. The mode of reaction to voltaism is changed, as Erb has pointed out. The earliest contraction occurs with the positive pole when the circuit is closed, and sometimes when it is opened, instead of at the negative pole when the circuit is closed. Moreover, the closing and opening of the circuit causes not only a momentary contraction, as in health, but a prolonged contraction, "tetanus," and this with the positive pole (anode) as well as with the negative (kathode). It is the only condition in which anodal opening tetanus has been observed in man. Thus, instead of the normal reaction (see p. 67) 1, KClC; 2, AClC, KOC; 3, AOC, we have 1, AClC; 2, AOC; 3, KClC or 1, AOC; 2, AClC, KClC, AOTe. This increased irritability, electrical and mechanical, is greatest at the height of the disease, lessens with it, but usually persists for two or three weeks after the spasms have ceased and after pressure has ceased to be effective.

During severe paroxysms there is often copious perspiration, and sometimes local vasomotor disturbance, redness, and even slight œdema in the affected parts; the pulse is quickened, and in some cases a rise in temperature has been observed, rarely exceeding 101° F. In continuous tetany the temperature is usually normal. The urine is usually unchanged, but transient albuminuria has been twice observed (Kussmaul, Nönchen), and in one case glycosuria coincided with the attack (Stich). Tetany has been followed by general muscular atrophy and also by local atrophy of the thenar muscles spreading to the forearm, and accompanied by slight atrophy in one leg. The electrical irritability was lowered in the atrophied muscles.*

Varieties.—We may distinguish three varieties of tetany, according as the spasm is intermittent, remittent, or continuous, but cases are met with that present every gradation between these varieties. Although the spasm is almost always symmetrical, I have once seen it so much greater on one hand than on the other that when slight in degree it was one sided. There is little relation between the course and severity of the spasm, except that in the continuous form the spasm is never so violent as it sometimes is in the paroxysmal varieties. In young children tetany is usually continuous, but the intermittent form is not unknown. In older children and adults the continuous form is less common than that in which the spasms remit or intermit.

It has been said that not only does continuous tetany persist during sleep, but that attacks of spasm may occur, even for the first time, during sleep.

Slight symptoms of this character are, indeed, very common during sleep, especially in adult women who are in somewhat feeble general health. The patient is waked up by tingling in both hands accompanied by a feeling of stiffness, and sometimes by actual rigidity, which may prevent all movement and last for a few minutes or longer, even for an hour or more. The fingers may be in the position characteristic of tetany, or may be flexed at all joints, but never strongly. The tingling usually lasts longer than the spasm. The condition is bilateral and comes on irrespective of posture.† It seems to be a sort of nocturnal tetany. Occasionally the patients who suffer from this nocturnal tetany have similar attacks in the daytime. Now and then the attack is accompanied by some pain in the head.

Course.—In both the continuous and intermitting form of tetany, the total duration of the disease varies much. Cases of either variety may last only a few days; but the continuous form very seldom presents the prolonged duration which is not uncommon when the spasm is intermittent. If the spasm presents no considerable remission it is rare for the disease to last more than a few weeks, whereas in the cases of intermittent tetany the disease often lasts for several months. In the severe cases after excision of the thyroid the duration is sometimes prolonged; in one (recorded by Weiss) the symptoms still continued three years after the operation.

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* Weiss, *Wien Allg. med. Zeit.*, *1885*, No. 31.

† It must not be confounded with the tingling in the region of the ulnar nerve that is due to long flexion of the elbow, or in all the fingers that is caused by pressure on the brachial plexus; in these cases the tingling is always one sided.

Tetany is an affection prone to return on a recurrence of its exciting cause, and sometimes without appreciable cause. A mother suffered from it while suckling each of five successive children (Maccall), and a woman had an attack in each of ten successive winters (Lussana). In other cases, years have intervened between attacks; three, six, and even eleven years (Chvostek). I have seen a case in which a woman became subject to attacks at the age of twenty-two, and was still liable to them at thirty-four; each winter, as the cold weather came on, she suffered from rheumatic pains in the limbs, and these were followed by attacks of tetany, each lasting fifteen or twenty minutes. They occurred every few days (but several times daily at the menstrual periods), until the late spring, and then ceased until the winter.

Termination.—Most cases of tetany end in recovery. Very rarely some weakness of the legs has persisted for a time. When death has occurred, it has been usually due to the cause of the tetany, *e.g.* to diarrhœa. In a few instances the spasms have caused death either directly, by their violence producing fatal exhaustion (Trousseau), or indirectly, by the repeated interference with respiration causing congestion of the lungs, and a low form of pneumonia. The disease, when it follows excision of the thyroid, is much more fatal than it is under other circumstances. Tetany has been known to be immediately followed by chorea (Salomonsen), and, as we have seen, muscular atrophy may supervene, either slight and confined to the hands, or general and severe.

PATHOLOGICAL ANATOMY.—In the few cases in which a post-mortem examination has been made, no change has been found which we can regard as the cause of the disease. The minute alterations that have been detected in the spinal cord in some cases* are probably secondary to its functional overaction. Such, for instance, are capillary hemorrhages, accumulation of lymphoid cells around blood vessels, and even minute foci of myelitis. The ganglion cells have been found somewhat shrunken in aspect, and to contain vacuoles.† Hyperæmia of the upper part of the cord has been observed in some cases, but was absent in others. Softening in the cervical region (Trousseau), and hyperæmia of the membranes in the same part (Bouchut), if not of secondary origin, are at present too isolated facts to deserve significance.

PATHOLOGY.—The great increase in the excitability of the motor nerves shows that there is an alteration in the function (and doubtless, also, in the nutrition) of the motor nerve fibres. We know that changes in the nutrition of these fibres are usually dependent on, and secondary to, a similar change in motor cells of the spinal cord, of which the axis cylinders of the motor nerves are the prolonged processes. The discovery of slight changes in these cells in severe cases, and the fact that slight spinal weakness may follow tetany, also suggests a disturbance of function of the cord, while the bilateral symmetry of the disease and the peculiar and uniform character of the spasm, constitute strong evidence of its central origin. The fact that muscular atrophy has been observed to succeed it is also some evidence of a special disturbance of the motor cells of the cord, including, of course, the cells from which the motor cranial nerves arise. The sensory symptoms which often precede the spasm indicate that the disturbance of function is not confined to motor structures, but involves, also, the sensory nerve elements. Beyond this we are not at present justified in going. There is in this (as Weiss has pointed out) nothing inconsistent with the possibility that the primary disturbance may be still higher up, in the brain, and that the derangement of the cord and nerves may be secondary, although there is, at present, no proof of this. The peculiar form of spasm ("interosseal" flexion of the fingers) is seen in some forms of spasm of brain origin (*e. g.* many cases of epilepsy) but we do not yet know what is its precise significance.

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* By Weiss, Langhans, Ferrario, etc.

† The significance of the vacuoles in nerve cells has been much discussed. I believe that they form during the process of hardening, but only when the cells are in an abnormal state.

Whatever the seat of the disease, the morbid process must, as a rule, be limited to disturbance of function and underlying nutrition, and the occasional origin of the disease in reflex irritation suggests that the disturbance of function may be, in such cases at least, the primary change. The resemblance of the spasm to that seen in ergotism has struck many observers,* and has suggested its dependence on some toxic influence, but the majority of etiological facts are opposed to such a supposition. To explain the paroxysms, Weiss has suggested the occurrence of periodical spasm of the vessels of the gray matter of the cord, but intermission in functional nerve disturbance is too frequent a phenomena to need the hypothesis of a vasomotor mechanism to account for it.

The frequent occurrence of the disease after excision of the thyroid is a pathological fact of great importance, and, doubtless, of great significance, if we could only discern what its significance is. But the mystery that still surrounds both the pathological relations of the thyroid, and also the occasional consequences of its removal, should make us hesitate in drawing any hasty conclusion as to the mechanism by which tetany is caused in these cases. Weiss has suggested that the ligature of the inferior thyroid artery causes a hyperæmic condition of the medulla, and that the ligature of many arteries during the operation, irritating the sympathetic fibres, causes an excitable condition of the gray matter of the cord. But this assumption is purely hypothetical; there is no evidence of either the occurrence of the process or of its adequacy. It should be noted, although we cannot at present connect the two effects of excision of the thyroid, that general myxœdema sometimes follows the operation very quickly. In the monkey,

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* Imbert-Goubeyre (1844), Hasse, Moxon, Eulenburg.

Horsley has observed it as early as thirty-eight days after the excision, and, what is perhaps of some significance, has found at this date commencing atrophy of the cortex of the brain.*

On the whole, our present knowledge of the pathology of the disease points to the nerve cells of the spinal cord and medulla as the parts chiefly deranged, and the way in which the cells in rare cases seem to undergo subsequent atrophy suggests that the disturbance is a primary one of the cells themselves, and is not produced by the agency of any vasomotor mechanism. It is difficult to conceive that symptoms of such definite and uniform character can be the result of any vascular spasm. The occasional wasting, with diminished irritability, is especially important as suggesting that the nutritional changes in the motor cells and fibres, causing the increased excitability, may sometimes go on to structural degeneration.

DIAGNOSIS.—The peculiar character of the spasm, its limitation to certain groups of muscles, its commencement in the extremities, and its bilateral symmetry, are usually sufficient indications of the nature of the case. If present, the intermissions in the spasm, the increased irritability of the nerves, and the excitation of the spasm by their compression, corroborate the diagnosis, but the absence of these symptoms is of little weight. The presence of one of the most common etiological conditions (exposure to cold, diarrhœa, lactation, or pregnancy), is also, in some cases, of diagnostic value. It is important, however, to remember that in slight cases the sensory symptoms may attract more attention than the motor spasm. Whenever chronic tingling in the hands and feet is complained of, careful inquiry should be made for attacks of spasm.

From tetanus the disease is distinguished by the commonly intermitting character of the spasm, by its commencement in the extremities, by the peculiar posture of the hands, and by the fact that the earliest symptom in tetanus, spasm in the masseters, is the latest in tetany. In organic brain disease, contracture is usually permanent and often unilateral; there is paralysis in addition to spasm, and cerebral symptoms are usually present. Hysterical contracture may closely resemble tetany in so far as the form of spasm is concerned, but is almost invariably unilateral, while tetany never is. When such contracture is bilateral, the case should be regarded as one of tetany. In the tonic form of epilepsy bilateral spasm may exist, almost identical with tetany in its character, but distinguished by the extreme brevity of the paroxysms, and by the occurrence of loss of consciousness.

PROGNOSIS.—Tetany involves little danger to life; almost all cases recover. The chief danger is from the cause of the tetany, such as severe diarrhœa. It is only when the paroxysms are very violent, causing considerable and repeated interference with respiration, with signs of œdema of the lungs, that there is any ground for apprehension. The danger to life is much greater in tetany after thyroidectomy than in the common form. The duration of the disease is difficult to foretell. When the attacks are prolonged, lasting several days, the disease is not likely to be of long duration, and continuous tetany, of slight intensity, will certainly soon be over. As long as the irritability of the nerves is increased, the paroxysms are likely to recur. It must be remembered that the subjects of tetany are very liable to future attacks, if exposed to an adequate cause. The tetany of pregnancy sometimes quickly ceases, but often it continues until delivery.

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* *Path. Soc.*, Nov. 4th, 1884; *Brit. Med. Journ.*, *1884*, ii, p. 910.

Treatment.—Whenever the cause of the disease can be traced, the removal of this is the first and most important element in treatment. If resulting from exposure to cold, warm baths or free diaphoresis should be employed. The state of the bowels must be carefully regulated. Lactation should be stopped and strength restored by tonics, especially by iron and quinine.

In most cases the spasm may be relieved most effectually by free doses of bromide of potassium, ʒss, three times a day. Chloral, Indian hemp and hypodermic injections of morphia have also been found useful for the same purpose. The inhalation of chloroform at first increases the spasm, but all contracture usually disappears when the patient is fully under its influence; at the same time chloroform has no influence in preventing a recurrence. Two cases, however (recorded by Hauber), which had resisted other treatment, were quickly cured by a course of chloroform inhalation, with energetic massage during the narcosis. The local application of stimulating liniments (especially chloroform) and of cold to the extremities, also sometimes arrests the spasm. Trousseau observed benefit from the application of ice to the spine. Electrical treatment has been followed by improvement in some cases, and has failed in others. The best results have been obtained with the voltaic current, the negative pole to the spine, the positive on the irritable nerves, or both poles on the spine. Faradaism is contraindicated.

In France, tetany was once invariably treated by bleeding ; the symptoms rapidly disappeared in some cases, especially in men, occasionally, also, in weakly women. The procedure often failed, however, when tonics quickly cured the patient.

The treatment of tetany in children, is, in the main, similar to that in adults. Attention to the underlying diathesis is of paramount importance. In young children, symptoms of rickets are rarely absent, and cod-liver oil and iron are the most effectual remedies, the continuous tetany of early life being less amenable to sedatives than the intermittent tetany of older children and adults. Cheadle found bromide and chloral useless in one case, in which the extract of Calabar bean succeeded, in doses of one thirty-sixth of a grain increased gradually to a third, but cod-liver oil and iron were given at the same time.

For noctural tetany I have found no remedy so useful as a dose of digitalis given at bedtime. Bromide also often removes the symptom, but occasionally fails. Tonics are also needed.

TETANOID CHOREA.

A case was recently under my care that presented symptoms intermediate between those of chorea and of tetany. The disease was fatal and no lesion was found after death. The patient was a boy aged ten. A brother was said to have died from some affection similar to that from which this child was suffering. There was a history of three other relations having suffered from maladies resembling chorea. In the patient the symptoms commenced gradually, seven months before death. They consisted of tonic spasm, which was continuous, and varied by paroxysmal attacks of similar but more intense spasm. The face was involved on both sides, so as to cause a constant peculiar smile. The tongue was pressed back against the palate in such a manner as to impede swallowing and prevent speech. The arms were extended, pronated and rotated inward, so as to bring the back of the forearm outward, while the fingers were generally slightly flexed at all joints, but at times were extended, and slowly moved in the irregular way characteristic of athetosis. The legs were extended at all the joints, the feet being over-extended in talipes equino-varus, and the toes were flexed. At times the spasm at the hip became flexor, so that the extended legs were raised off the bed. The muscles of the trunk were also involved in the spasm. At first, the left side was the more severely affected, but afterward the spasm became equal on the two sides. The electric irritability of the muscles was normal, and there was no mechanical excitability of the nerves. There was considerable pyrexia during the more severe stage of the disease. The boy steadily emaciated, and died from exhaustion. The whole central nervous system appeared normal to the naked eye, and no distinct morbid appearances could be discovered on microscopical examination. I have not been able to find a description of any similar case.

OCCUPATION NEUROSES.

The term "occupation neuroses," adopted from the German ("Beschaftigungs-neurosen"), is a convenient designation for a group of maladies in which certain symptoms are excited by the attempt to perform some often-repeated muscular action, commonly one that is involved in the occupation of the sufferer. The symptoms occur in the part by which the action is effected, and the action is interfered with by them. Other acts (at least in the early stage of the affection) do not excite the symptoms, and are not interfered with. The most frequent symptom is spasm in the part, which disturbs or prevents the due performance of the intended action, and hence the word "cramp" is commonly used in naming them, qualified by the special action, or rather occupation, that excites the disorder, as "writers' cramp," "pianoforte-players' cramp," "telegraphists' cramp," "seamstresses' cramp." The term "cramp" sets forth the muscular spasm which, in most cases, is the chief, but rarely the sole symptom. There is usually not only spasm but pain, which is not the direct result of the spasm, and may be the only obtrusive symptom. It may be referred to the muscles, or to the bones or joints, or to the position of certain nerves. In the latter case, it resembles neuralgia, and the more closely, since certain foci of pain and tender points may be developed, as in ordinary neuralgia, from which it differs in being at first not spontaneous, but excited only by the special action. Hence we must distinguish a motor and a sensory form, a spasmodic and a neuralgic variety of occupation neurosis, but the two forms are very often combined. Although the disturbance is in the first instance confined to the special action, it usually extends, after a time, to other actions, in varying degree and in proportion as the muscular contractions correspond in combination and character to that of the movement first disordered. The extension may ultimately be so wide that spasm, and still more often pain, occurs on any movement. I have even met with cases in which a spasm, at first excited only by a special act, ultimately extended not only to all acts, but occurred spontaneously. Thus, in their ultimate extension, the occupation neuroses are not so well defined as in their earlier stages. Moreover, they are not always well defined in their causation. Local disease or injury may distinctly coöperate in developing the morbid state. In such cases extreme care is necessary in determining the nature of the case, because a local affection is often first *revealed* by the interference with the action performed most frequently and for the longest time. Many forms of disease are thus erroneously supposed to be of the nature of occupation neuroses, when the occupation merely manifests and does not cause the symptoms.

The most common form of occupation neuroses is that of writing, and since this is not only the most common but is typical of the class, it will be most convenient to describe it at length, the conclusions regarding it being applicable to all other forms.

WRITING NEUROSES; WRITERS' CRAMP.

Writers' cramp was first described by Sir Charles Bell in the year 1830. It was called "scriveners' palsy" by Solly, a term still sometimes applied to it.*

ETIOLOGY.—The affection is very much more common in males than in females, especially in its motor form, doubtless because comparatively few women are engaged in occupations that involve a large amount of writing. If all forms are included, males constitute two-thirds of the sufferers, but a still larger proportion, at least five-sixths, of those who present the motor form. It is a disease of the active period of adult life, very rarely commencing under twenty or after fifty, while about five-sixths of the cases begin during the intervening thirty years. The distribution of 135 cases† is as follows:—

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* The most important writings on the disease are those of Berger ("Eulenburg's Real Encyclopædia," art. *Beschaftigungs-neurosen*) and of Poore (*Practitioner, 1878*; "Text-Book of Electricity," and *Med.-Chir. Trans.*, vol. lxi). The latter article contains a large number of carefully observed facts.

† 33 cases described by Poore (*Practitioner, 1878*), 64 collected by Berger (loc. cit.), and

	10–	20–	30–	40–	50–	60–
Cases . .	2	48	44	27	9	5

Thus, about one-third of the cases commence in each of the third and fourth decades of life, and only a fifth in the following ten years.

An hereditary tendency to nervous affections can often be traced. One of my patients had an epileptic uncle; another lost a brother from general paralysis of the insane; three others had each an insane parent, and one an epileptic father. In some instances more than one member of a family has suffered from the special affection. Thus Vance relates the case of a patient who, to rest his hand, went on a visit to a distant brother, and was surprised to find that he also had given up writing from the same cause. I have met with a very characteristic case in a lady whose father also suffered from writers' cramp. In estimating a family tendency it must be remembered that writers' camp is a disease that is easily imagined, especially by those who have witnessed the disorder. The malady is sometimes met with in those who exhibit no other symptom or tendency to nervous derangement, but more frequently the subjects are of distinctly "nervous" temperament, irritable, sensitive, bearing overwork and anxiety badly, and occasionally the neuropathic tendency shows itself in other independent disease. For instance, one patient with characteristic writers' cramp (spasm absolutely confined to the act of writing) subsequently presented the early stage of tabes. Two of Berger's patients were epileptic, and one of my own had some fits in early adult life. Another had suffered from infantile paralysis in one leg. We must distinguish, however, between such cases in which an independent disease shows the existence of a neuropathic tendency, and the cases to be presently considered, in which a nervous affection of the part concerned in writing is one of the causes of the special disease.

Whatever lowers the general tone of the nervous system may doubtless act as a predisposing cause, but no influence is met with so frequently as to deserve special mention, except anxiety. It is remarkable how many patients, at the time of the onset of the affection, were enduring anxiety from family trouble, business worry, or weighty responsibilities. As an instance of the potent influence of anxiety I may mention the case of a clerk in a Government office who had done only a moderate amount of writing, but became affected with writers' cramp during an anxious lawsuit about his wife's property. He took six months' rest, and at the end of two months his handwriting was as good and free as ever. Toward the close of his holiday his wife became seriously ill; while nursing her, and before resuming his work, the writing cramp returned.

The affection sometimes follows local disease or injury. A naval officer sprained his thumb, and before it had recovered, had to do a considerable amount of writing in preparing a report of a surveying expedition, and characteristic writing spasm came on. In many recorded instances some painful

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36 cases of my own; all being rejected in which, from the nature of the symptoms, there was any room for doubt as to the nature of the case.

affection of a finger has preceded the onset, and in one case periostitis of the external condyle of the humerus (from which some of the forearm muscles arise) distinctly coöperated in causing the cramp; pressure on the tender part excited reflex spasm (Runge). In two patients, characteristic writers' cramp followed recovery from a slight attack of right hemiplegia (Vance, Runge). Primary neuralgia and neuritis in the arm may also aid in the causation of the disease, but nerve tenderness, which is common, is probably, in most instances, part of the affection and not its cause, and does not show that there is a primary disease of the nerves.

The chief agent in the production of the malady is the act of writing, which has usually been excessive in degree. Hence the affection occurs chiefly among those who earn their living by writing, and by writing much, and clerks furnish the majority of the cases. But it now and then occurs in persons who have not done an excessive amount of writing; and in such cases a powerful predisposing cause, such as anxiety, may commonly be traced. The occurrence of the disease is influenced less by the amount than by the manner of writing. There are, in writing, two chief elements, the way in which the pen is held, and the way in which the movements are effected. The mode in which the pen is held is comparatively unimportant; it is the mode in which the pen is moved that chiefly determines the occurrence of the disease. The movement may be effected either by the muscles moving the fingers and hand, or by the muscles of the upper arm, and according to the mode adopted does the necessary degree of muscular contraction vary. The smaller the muscles employed, the greater must be the relative degree of contraction to produce a given movement of the pen, the greater is the amount of fatigue produced, and the more readily does cramp occur. Writers' cramp is almost entirely confined to those who write in the first two of the following four modes: (1) The worst mode of writing is with the little finger as a fixed point of support. The pen is then moved up and down by the muscles of the thumb and first two fingers, which are constantly contracted almost to their maximum; the lateral movement is effected by a slight supination of the hand; only a few letters can be written without the movement of the little finger, and before it is moved a considerable strain is necessary to prevent the pen being raised from the paper by the process of supination. Of all possible modes of writing, this is the worst. (2) The most common mode is to write from the wrist as a fixed point. In this method, the up-and-down strokes are made chiefly by the flexion and extension of the fingers and thumb in the manner above described, but the lateral movement of the hand is effected chiefly by the ulnar abductors of the wrist; as these are also flexors and extensors, considerable effort is necessary to keep the pen on the paper during the left-to-right movement. (3) A far better way is to write from the middle of the forearm; the wrist moving with the hand and the lateral movement being effected by adduction of the humerus, the forearm rotating on its point of support as on a pivot. Even in this method, however, since the tendency of the hand is to move in a curve, effort is necessary to keep the words in a straight line, and this has to be effected by increasing flexion of the wrist, which interferes with the action of the flexors of the fingers in moving the phalanges. (4) The best and freest method is to write from the upper arm and shoulder, with no fixation of the arm; the forearm, wrist, and little finger rest on the table so as to take off some of the weight of the limb from the shoulder muscles, but both wrist and forearm move along the table as the writing progresses from left to right. In this way the pen is held very lightly; scarcely any of the movement is effected by the small muscles of the hand; the fingers scarcely alter their position, except when a stroke is carried far above or below the line; and even for this a movement of the fingers is not always necessary.* No style can be considered free unless it is easy to write a whole line across a page of foolscap without once breaking contact between the pen and the paper.

It is probable that the practice of thickening the down-strokes conduces to the occurrence of the affection, as it necessitates frequent increased pressure on the pen. Many writers have suggested that the use of steel pens is one element in the causation of the disease, and it seems probable that this is true, since steel pens have to be grasped more firmly, and adjusted more exactly, than a quill, and also because the disease seems to have become more frequent since the use of steel pens has become almost universal. But writers' cramp occurred when quills were the invariable instruments of writing, and is still seen occasionally among those who never use a steel pen.

Among clerks who suffer, lawyers' clerks constitute an undue proportion. This is no doubt due to the cramped style in which they commonly write. On the other hand, writers' cramp is practically unknown among those who write more, and under higher pressure, than any other class, shorthand writers. The speed required and the style needed for forming shorthand characters compels a very free style of writing, generally from the shoulder, and this is also adopted by them in longhand writing, and the result is that they have an almost complete immunity from the disease.†

SYMPTOMS.—The precise mode in which the act of writing is deranged varies much in different cases. The chief disturbance is usually from spasm, but sometimes it is from pain. Occasionally there is tremor without other spasm, ceasing when the attempt to write is relinquished. More rarely still there is simple inability to write, from apparent weakness, without spasm, although there is no loss of power for other movements. Four forms may thus be distinguished, the spastic, tremulous, neuralgic and paralytic forms, but the last is extremely rare, and I have never met with an example of it.

Motor Symptoms.—The spasmodic form is the most frequent and the most characteristic, but presents considerable variety in its features, depending partly on the mode in which the patient is accustomed to write, and the muscles which he accordingly calls into chief action. The commencement s almost always gradual (some instances of sudden onset will be mentioned presently). After writing for some time the patient finds something unusual about his writing; the pen does not move quite as he intended it to do; a stroke now and again is irregular, extends too high or too low; a slight nvoluntary movement causes an unintended mark. He finds that he is grasping the pen too tightly, and cannot help doing so; that the fingers do not keep in their accustomed place; and the first finger has a tendency to slip off the pen, so that this gets between the first and second finger. He endeavors to mend matters by taking a firmer hold, but this seems to increase the difficulty, and he finds that he writes slowly, as if a weight were attached to the hand. The hand feels strangely tired, and an aching pain in the finger or thumb or first metacarpal bone, or in the wrist or forearm, makes it still more difficult for him to go on writing. The symptoms may continue with only slight impairment of the power of writing, for weeks or months, but they occur after writing for a shorter time; they increase in degree; and now and then there is distinct spasm, which cannot be controlled. The first finger or the thumb tends to become flexed at the middle joint, so that its tip moves up the pen, or, less commonly, the fingers become extended, so that the pen is not pressed against the paper with sufficient force, and may even drop from the hand. The characters of the writing become still more irregular, the down-strokes are too thick, the point of the pen may be driven through the paper, and in its irregularity of form and force the writing "resembles that done in a jolting carriage." Rarely the chief spasm is in the fourth finger, or in the third and fourth fingers, and pain may be felt in the long flexor of these fingers, and the ulnar flexor of the wrist. Sometimes the whole hand seems to get stiff. As the spasm increases in degree, it extends in range, and involves more of the muscles of the forearm. There is a tendency for the wrist to become flexed, or extended, or supinated, and in the effort to prevent the disturbing movement, the opponents contract strongly, until at last all the muscles of the forearm may be in such energetic

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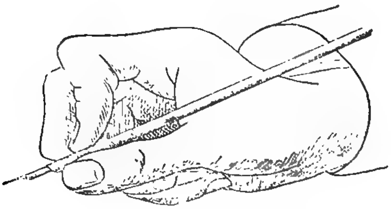
* To some who are accustomed to write with the fingers chiefly, the statement in the text may seem incredible, but I have verified it by a pencil fixed to the forearm, which produced the same characters as were traced by the pen held in the hand.

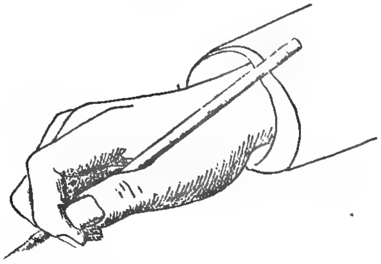
† I have indeed seen one case in a shorthand writer, but it was not quite a pure case, and at the meeting of the recent Shorthand Congress I could not hear of any writer who had ever known of a case of writers' cramp in a member of his profession.

FIG. 323.



Cramped method of holding pen, habitual to a patient who suffered from writers' cramp.

FIG. 324.

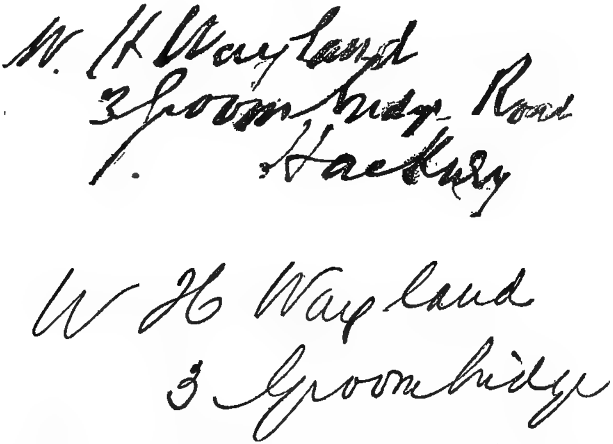


Pen held between first and second fingers, by a patient suffering from difficulty in writing.

spasm as to render movement of the pen impossible. Various devices are at first employed to counteract the spasm. The mode of holding the pen is changed; it is held between the first two fingers, or fixed in a piece of cork, which is grasped with the hand, and the movements in writing are effected by the upper arm; or the patient fixes the right hand by the help of the other; placing, for instance, some fingers of the left hand between the last two fingers of the right. For a time these devices give a little help, but the spasm gradually increases in degree, and overcomes the fixing help, or it spreads to the muscles of the upper arm.

Although the onset is gradual in almost all cases, in very rare instances the affection comes on in an acute manner. In the cases of this character, pain has generally been a permanent symptom, and some slight symptoms have usually preceded the acute onset. One patient, who had noticed a slight "crampy" feeling in his fingers after writing for a long time, one day wrote rapidly for several hours, and then the hand suddenly became so stiff that he could scarcely move it. He gave up, but next day was no better, and when I saw him, ten days later, he could only write two words, and that with extreme slowness and effort, and the attempt caused much pain. Another patient, who had been conscious for some time of a little more fatigue in writing than was usual, sat down one day to write a number of letters on a subject that vexed her much. After writing for about two hours, she felt a sudden pain around the wrist, passing down to the knuckles, and up the forearm to the elbow. The hand then slowly closed in spasm. She forced the fingers back and went on writing for a short time with much pain and difficulty. Subsequently, for some years, as soon as she attempted to write the same pain came on, followed by spasm. Other acts, after a time, also excited the pain, although much less readily than writing.

FIG. 325.



W. H. Wayland
3 Groombridge Road
Hackney

W H Wayland
3 Groombridge

Handwriting of a patient suffering from writers' cramp, and after recovery.

The spasm is almost always tonic in character; and, although it may now and then be varied by a slight start or jerk, there is very seldom actual clonic spasm. I have only seen one case with such clonic spasm ; in this, as soon as the patient attempted to write, the first finger and thumb became flexed at all joints by clonic spasm, and slipped off the pen. The affection had existed for five years, and was at first limited to writing; afterward any action in which the fingers were flexed would bring on the spasm. But tonic spasm is often accompanied by some tremor, and occasionally the tremor is the most conspicuous symptom. The letters are "shaky" and the lines are varied here and there by angular zigzags. As soon as the attempt to write is relinquished, the tremor usually ceases. It is rare to meet with tremor only; in most cases spasm is associated with it; there may be at first simple spasm and afterward some tremor in addition, or tremor may at first occur alone and afterward tonic spasm as well as tremor. Occasionally, in old-standing cases, there is slight tremor in the hand when the patient is not writing, and when this is the case there is often some tremor in the left hand as well as in the right.

The spasm may be limited to the act of writing, and other actions, even such as involve delicate muscular coördination, may be performed without the slightest difficulty. It is not uncommon, for instance, for the patient to be able to shave himself or to play the piano with perfect facility. In slight cases the spasm may be limited even to the act of writing with a pen, and the patient can write with a pencil without difficulty.* I have known a patient to be able to paint without difficulty, although he could scarcely write. A still more curious limitation has been observed; printing characters could be traced with a pen, although an attempt to write in the ordinary manner at once brought on a spasm.

But absolute limitation to the act of writing is seldom met with, except in cases of very slight degree. In most severe cases the patient experiences some difficulty in actions requiring delicate coördination of the same muscles. One patient, for instance, had no difficulty in any other action except in shaving himself. Another, at the end of twelve years, could do everything except draw and scratch out with a penknife. The extent to which the spasm spreads to other actions thus varies much in different cases. It is greatest in cases in which flexor spasm predominates. Occasionally, in such cases, the spasm comes on when any action is attempted, and I have known one case in which spasm, at first confined to the act of writing, ultimately not only extended to all other actions but became spontaneous, so that when the hand was at rest the fingers and wrist gradually became flexed.

Power in the hand may be quite unimpaired. Sometimes the grasp is a little weaker than it should be, and it is not uncommon, as Poore has pointed out, to find definite slight weakness of certain muscles of the hand. Occasionally there is considerable loss of power and inability to sustain effort.

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* Partly because he can press more firmly on the pencil, and thus steady the hand (see Poore, *Med.-Chir. Trans.*, vol. lxi, p. 127).

There may in rare cases be slight wasting of certain muscles, but this is altogether exceptional in cases of true writers' cramp.

The electric irritability of the nerves and muscles may be perfectly normal, or may present a slight change, increase or diminution, chiefly in cases that have lasted for some time. The change is usually the same to faradaism and voltaism, and the degree of irritability is similar in both muscles and nerves. It is often found in all the nerves which are accessible to examination. It is thus to be regarded as a primary change in nerve irritability, not of muscle irritability; the change in the irritability of the muscles depends on the motor nerve endings they contain.* It may be remembered that all three nerves of the arm, radial (musculo-spiral), ulnar, and median, supply muscles employed in writing. Several cases I have seen show that increased irritability is the earlier change of the two and that the diminution of irritability succeeds it.

Sensory symptoms are seldom entirely absent, and are often very prominent. The tonic spasm is accompanied by a painful sense of fatigue in the muscles, and by definite, dull pain, often referred to the bones or joints, very often to the metacarpal bones or to the wrist, soon ceasing when the effort to write is relinquished. Occasionally, though not often, a tingling sensation, "pins and needles," is felt in certain fingers during the attempt to write. Now and then the pain is more severe, and may then be referred to the course of the nerves so as to be distinctly neuralgic in character. At first it occurs only during the act of writing, but after a time it may be produced by any muscular effort in the part. It may be chiefly localized in certain foci along the course of the nerve, and after a time the nerves are found to be tender and the tenderness is greatest at the foci of pain—these tender points thus completing the resemblance to ordinary neuralgia. In many cases some muscular spasm accompanies the pain during the act of writing, and often has preceded the pain in time. I may briefly relate a most instructive case of this character. The patient was a young woman, a lithographic writer, producing exquisite copper-plate writing which could only be executed in a cramped manner, with fixation by the little finger. She found that her hand grasped the pen with undue force, and now and then a stroke did not go quite in the intended direction. The spasmodic symptoms slowly increased, without the slightest pain, for three months, the pen tending to slip between the first and second finger until she was obliged to hold it thus. Then slight pain was felt in the thumb and fingers at the places where they were in contact with the pen. This pain gradually extended to the wrist, and then up the arm to the axilla, and continued until she came under my care two years after the onset. Then, the spasm remained about the same, but the pain was so great as alone to prevent her from writing. It still commenced where the pen was in contact with the thumb and with the contiguous sides of the first two fingers, being felt in all three, and darting up the arm. A focus of greater pain was situated in front of the wrist over the median nerve, and another a little below the elbow; these points were very tender to pressure. There was also some general tenderness of the median and ulnar nerves, and another tender point on the musculo-spiral. The pain never occurred spontaneously, but was now excited by any muscular effort. The nerves presented a distinct increase of electrical excitability. Thus, the prominent trouble at the time the patient came under observation was neuralgic pain, but both the history and the characters of the pain showed that it was secondary to writing and a true "occupation neuralgia." In some cases the pain may become altogether independent of the act of writing. Thus, in one patient it was associated at the onset with well-marked spasm; writing was entirely given up, and the spasm ceased, but the pain continued and became produced by any movement, and ultimately it was felt chiefly in the upper part of the arm and then in both shoulders, and was brought on by any emotion as well as by movements. The sensory symptoms, when they once become prominent, often exhibit a strong tendency to spread, and sometimes acquire a relative independence, as in the case just mentioned. The feeling of "tingling," or "pins and needles," already mentioned as an occasional symptom, may spread, like the tremor, to the other arm.

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* At the same time it would be wrong to lay much weight on this consideration, because it is possible that an abnormal irritability of the muscular fibres may cause them to respond more readily to a given stimulation of the nerve fibres. We can only judge of the stimulation of the motor nerves by the effect on the muscular fibres. It is, however, customary to regard a change in muscular effect of stimulation of the nerves as evidence of a change in the nerve fibres themselves.

Course.—The symptoms continue and usually increase as long as the patient perseveres in the attempt to write, and only become stationary when prudence or sheer inability prevents the effort. The rate of increase varies much in different cases, and is generally rapid in proportion to the badness of the original method of writing. The tendency for the affection to spread to other actions also varies much, and is generally proportioned to the general weakness of the nervous system. As we have seen, the sensory symptoms of neuralgic type exhibit a greater tendency to extension than do the motor symptoms.

The sufferer who finds himself unable to write with one hand often learns to write with the other. After he has acquired the needful facility, and has written with the left hand for a time, similar symptoms may develop in this hand, and they then usually progress more quickly than in the arm first affected. When sensory symptoms are prominent, the left hand is especially apt to suffer early. Fortunately the invasion of the left hand is not invariable; I have even known it to do good work for twelve years, although the affection continued unchanged in the right hand. Indeed, if we exclude the cases of neuralgic type, I think that the chances are nearly equal whether the left hand will suffer or not. If perfect rest is given to the right hand, either by abstinence from writing or by the exclusive use of the left hand the symptoms may slowly lessen, and ultimately pass away, so that writing may be resumed, without any return of the affection. This was the case with the patient whose handwriting is shown in Fig. 325. A more typica case of writers' cramp could not be, and yet the recovery, during two years use of the left hand, was so complete that he has now written, and written much, with the right hand for thirteen years, without any recurrence of the disease. In some cases, however, the symptoms that have been removed by rest, return soon after the attempt to write is made, and in others, even rest fails to effect much improvement, and the symptoms may continue to the end of life. The tendency of neuralgic pain to spread widely has been already mentioned. When tingling is felt in the fingers it is occasionally also felt in the foot of the same side, and I have once known slight cramp in the leg to accompany such tingling and severe spasm in the hand.

PATHOLOGY.—No anatomical changes are known in writers' cramp, and none could be found in one case which I had an opportunity of examining. The pathology of the disease has therefore to be inferred from its symptoms and causation. Three chief theories have been held regarding its nature. According to one, it is essentially a local disease, a weakness in some muscles permits the over-action of their antagonists, which increases to spasm.* A second theory ascribes the spasm to reflex action, the result of the stimulation of the sensory nerves in the act of writing.† A third (held by most writers on the subject) regards the affection as primarily and essentially central, the result of a deranged action in the centres concerned in the act of writing. The first two theories seem inadequate, by themselves, to explain the symptoms. Most observers have failed to find in the early stage of true writers' cramp any evidence of primary weakness. The initial disturbance is spasm; the slight loss of power which may be found in certain muscles after the disease has existed for some time does not explain the spasm, first, because this is far wider in range than the antagonists of the feeble muscle, secondly, because the very muscles that are weak are often involved in the spasm; thirdly, because local muscular weakness does not excite spasm of the character presented by a well-marked case of writers' cramp. The reflex theory is inadmissible because, in the majority of cases, there is no evidence that abnormal sensory impressions precede the spasm. Even in the case related on p. 1068, in which pain was so prominent, it was distinctly secondary in time. Both weakness and pain in the hand are very common without the manifestation of the slightest tendency to the occurrence of writers' cramp. Although neither the theory of local weakness, nor that of reflex spasm, can be regarded as explaining the phenomena of ordinary cases, it is very probable that each influence may take a share, in some instances, in increasing the disease, and occasionally may help to excite it. But the morbid state of the centre may be developed without such aid, and must be regarded as the chief element in the disease.

All forms of occupation spasm affect movements that are purely acquired, and of all these movements that of writing is the most complex and most delicate. The acquisition of the power of performing any action with ease means the "education" of the nervous centres concerned in it, the establishment of a tendency to the associated action of nerve cells, in perfect adjustment of varied order and degree, with the least possible voluntary efforts of excitation and control. This, in the language of modern physiology, means the establishment of lines of lessened resistance in centres, *i. e.* between connected cells, and doubtless, also, modifications in the force and readiness with which nerve energy is liberated in them. This education is effected by the repeated functional action of the centres under the stimulus of the will, at first strong, afterward gradually lessening, as the desired functional mechanism is developed by exercise, and established by the resulting modifications of nutrition. There, in health, the process stops. Readily as nerve energy is evolved, it is always under a voluntary stimulus, and exactly in the degree required. The clearest conception we can form of the pathology of writers' cramp is that this process of lowering of resistance between nerve cells has gone too far, so that the energy evolved is excessive and irregular, although, when the same cells are excited in a different order, the resistance is normal in proportion as the order differs from that involved in the act of writing, and hence, in that degree, other movements escape. No doubt the impairment of function is attended by impairment of nutrition. In some cases the changes go so far that all attempts at movement give rise to cramp, and, at the same time, the total power that can be exerted is considerably below the normal.

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* Dzondi, Meyer, Haupt, Zaradelli. This theory has been ably advocated by Poore.

† Fritz, Romberg.

The mechanism of the morbid lowering of resistance may be the same as that by which the resistance was lowered in the education of the centre, carried in this case to excess. The degree of exertion necessary to produce the effect varies inversely as other influences coöperate and tend to degrade nutrition or to disturb the centre. Some causes, such as anxiety, aid in the former way, by impairing nutrition; others, such as local disease of the limb, in the latter, by deranging the centre. The influence of local pain and local weakness in deranging movement is too familiar to need illustration, but this effect is probably produced through the centre, and can only aid in the production of writers' cramp by disturbing the normal relation of the force evolved, and so aiding in the establishment of the morbid state just described.

The condition assumed is that which used to be described as "irritable weakness." In all parts of the nervous system, when force is too readily evolved, the total amount of nerve force which can be liberated is usually below the normal. Thus there may be weakness as well as spasm, and as the morbid condition may progress in one part of the centre more than another, the weakness may be distinct only in certain muscles. The fact that the affection occurs chiefly in those who write by means of small muscles, is quite compatible with the theory that the disease is essentially central. The difference between the method of writing by small or by large muscles is that in the one case there is extreme activity of comparatively few cells of the cerebral cortex, in the other case there is slight activity of many cells. The morbid state of nutrition which accompanies disturbed function in a motor centre tends to descend the motor fibres, and hence we can understand that a slight change in the electric irritability of the nerves should sometimes be found, just as in chorea.

It has been objected to the central theory, that it assumes the existence of a centre for the coördination of writing, and therefore of every separate action which any one part of the body can perform, and that we know nothing of the existence of such centres. The objection is invalid because no such assumption is made. The theory does not assume that there is a separate centre for writing or other movement, but only that there are varying lines of resistance in the innumerable connections of the motor nerve cells, and that, as each movement is the result of the action of nerve cells in different order and degree, permanent functional relations are developed and may be separately deranged. No separate coördinating centre is assumed, any more than in a complex piece of machinery, in which the bars and bands and wheels that convey the force coördinate it by their arrangement. It must also be remembered that the conception of a physiological centre does not necessarily involve that of a local limitation. Nerve cells act together that are far apart, and those that are adjacent are often independent. Diffused through a mass of gray matter may be many separate mechanisms, not necessarily more in one part than in another, and if we call them "centres" we must not allow mathematical conceptions to govern our physiological ideas.

The tendency, often seen, for the other hand to be affected, affords additional evidence that the disease is essentially central. Many facts show that there is a close functional connection between the similar mechanisms on the two sides. Thus, as already mentioned, a patient, when he tried to write with his left hand, found his right fingers performing slow movements of flexion and extension. On any theory, it is difficult to say why the second side should suffer in some cases and not in others.

One other symptom remains to be discussed—the neuralgic pain, which gives to some cases a special character. The act of writing involves afferent impulses, and sensory impressions, unnoticed till they become increased in fatigue. When cramp occurs, these become excessive. All increased sensation means increased action of the sensory centres, and this once established may assume a morbid independence, as in many cases of ordinary neuralgia (q. v.), and, as also in neuralgia, nerve tenderness may be secondarily developed. There is no justification for regarding these tender points as evidence of neuritis, for even in such a case as that recorded on p. 1068 the symptoms are merely such as are met with in many cases of neuralgia of primarily central origin. This case is especially instructive as showing how the pain, *i. e.* the morbid action of the sensory centre, became gradually developed from the increased afferent impressions caused by the spasm. The pain started from the points at which the nerves of the skin were unduly stimulated by too firm a grasp of the pen. It is scarcely necessary to point out that the same predisposing influences which facilitate the occurrence of that state of motor centres which shows itself in spasm, may also conduce to the development of a like state in a sensory centre, felt as pain.

There is, at present, no direct evidence as to the part of the nervous system in which the primary derangement occurs. That the action of the motor and sensory nerve cells of the spinal cord must be disordered is certain, since it is through them that spasm is produced and pain perceived. But we do not know whether their derangement is primary or whether it is simply the effect of a primary disorder in those cerebral centres in which the movements are arranged. The latter is the more probable, and the central region of the cortex is that to which we should naturally turn as the seat of the primary derangement, since it is from this part of the brain that the spinal cells are directly excited. Analogy suggests that the sensory disturbance is spinal in a larger degree than are the motor symptoms.

Diagnosis.—A well-marked case of writers' neurosis is rarely mistaken for any other affection. The initial limitation of the symptoms, whether spasm or pain, to the act of writing, sufficiently indicates the nature of the case. But the converse error is often made; cases are supposed to be examples of writers' cramp which are of a different nature. Many paralytic and painful affections of the hand are first discovered by the interference they cause with the action of writing, the most delicate action that the hand is called on to perform, and they are supposed to be specially connected with the act, when this merely first reveals their existence. I have many times known hemiplegia of gradual onset to be at first mistaken for writers' cramp, and in one case of commencing insular sclerosis a similar error was made. I have even known paralysis of the musculo-spiral nerve to be ascribed to writing because its onset (in the night) was unnoticed until the patient attempted to write. It is needless to enumerate the various affections that may be thus confused, because the simple rule above given suffices for their distinction—from the first, the symptoms are not limited to the act of writing. In the case of insular sclerosis, for instance, the attempt to write at once brought on clonic spasm in the hand, but every other movement did so likewise. The tremulous form may be mistaken for commencing paralysis agitans, but, at the onset, the tremor is confined to writing in the former case, while in the latter it accompanies all movements, and usually exists, also, when the hand is at rest.

Writers' cramp is a disease that is readily imagined. Most persons who have to write much experience at times some discomfort in the hand after writing, and since such discomfort is one of the symptoms of writers' cramp, they are apt to fancy that they are the subjects of the disease. The most important diagnostic indication is the occurrence of actual spasm, since this is seldom absent, even in the early stage. In the cases in which sensory symptoms are ultimately most prominent they seldom exist alone. If they are isolated, the diagnosis rests on the initial relation to one action. In most cases of the fancied disease, when the patients are reassured, the discomfort quickly ceases to be noticed.

Prognosis.—The disease, when well developed, is one in which the prognosis is always uncertain, and often unfavorable. When the affection occurs in a person whose daily bread depends on the act of writing, the early warnings are neglected, either through ignorance or the stern compulsion of necessity, until the morbid action of the centre has become fixed by nutritional alterations almost as ineradicable as those that render permanent the results of its early education. The prognosis is especially unfavorable if the disease has lasted for some time, and has reached a considerable degree, before the patient comes under treatment; if perfect rest from writing cannot be secured; and, also, if no other cause than the act of writing can be traced. It is better, if any extraneous and removable cause coöperates, such as defective general health, anxiety, and especially if local causes, such as injury, have distinctly aided in the development of the morbid state. Even, however, under the most unfavorable conditions recovery sometimes occurs. The writing shown in Fig. 325 was that of a clerk, aged thirty-four, in whom no cause could be traced but excessive writing. He had averaged ten hours a day for twenty years, and the affection had lasted for twelve months before he came under treatment, and yet, as already mentioned (p. 1069), recovery has been perfect. The prognosis in cases in which the sensory trouble predominates, is good if the patient can take a long and perfect rest, but unless this can be secured, the neuralgic pain, at first produced only by the act of writing, is likely to extend and become spontaneous. The probability that the other hand will be affected is greater in these cases than in those of purely motor spasm.

Treatment.—Writers' cramp is a troublesome and difficult disease to treat, and the question of its prevention is therefore of corresponding importance. The disease might probably be prevented to a very large extent, if not entirely. We have seen that it occurs almost exclusively among those who write in a cramped manner, in the first or second of the the styles mentioned on p. 1062, scarcely ever, and only in slight degree, in those who write in the third, and never in those who write in the fourth. If all persons wrote from the shoulder, writers' cramp would practically cease. But the mode of writing is a matter of education, and prevention of the disease rests, therefore, not with doctors, but with teachers. It is probably impossible to teach writing in the first instance in a free manner, but before a boy is allowed to pass from school, he should be got out of the cramped, and taught the free method—taught, that is, to write from the shoulder. It is also desirable that those who experience any difficulty or discomfort in writing should at once change their style for the freer mode. To make a person realize the characteristics of the free method, I have found the following expedient useful. Let him first draw a line across a sheet of paper; for this, the arm must be moved as a whole from the shoulder. Then let him make the line wavy, next increase the wavy character, and then slightly slope the waves, so as at last to make the line a series of *m*'s—*mmmmmm*. These are letters, and the transition to other letters will then be easy. As I have already said, the object to be aimed at is to write in such a manner that it shall be easy to form an entire line of words without once lifting the pen from the paper. The hand should grasp the pen lightly, and move it as a whole. The comfort and ease experienced when this method is acquired is very remarkable.

For the affection itself, treatment, to be effective, should be early. The commencing symptoms often pass away with a brief rest; a month's abstinence from writing at the onset will do more than a year's rest if the disease has continued for six months. When writing is resumed in such a case, it is important that its style should be changed as just described. The amount of writing should, if possible, also be reduced. It is desirable that a soft quill should be substituted for a steel pen, and that a holder should be employed that is of good size and not smooth on the surface.* Slight symptoms of cramp often pass away at once when this change is made.

In cases in which the disease has become established, rest is still incomparably the most important element in treatment. If writing is continued, every method of treatment fails even to check the progress of the disease. The various mechanical devices which, for a time, aid handwriting are not to be recommended, since they only stave off the impending disability, while the malady is really getting worse, and they thus increase the difficulty of treatment. If, however, prolonged and complete rest from writing is impossible, the patient should learn to write with the left hand. Facility in sinistral writing is acquired with a little perseverance, more readily if the letters are made upright, or if the customary inclination is reversed. In this way complete rest may be given to the affected hand. It is true the left hand may, after a time, suffer in the same way, but the tendency to this has been exaggerated by some writers. If it does, the patient is no worse off than if he had not learned to write with the left hand, while if it does not, he may be able to continue to earn his living, and may afford his right hand a fair chance of recovery. The Government clerk mentioned on p. 1061, fulfilled the duties of his office for twelve years with his left hand, and, although the cramp continued in the right, the left showed no indication of the disease, and at the end of the twelve years he was able to retire on a pension. Besides the case illustrated in Fig. 325, I have met with several others in which practical recovery occurred during the use of the left hand.

For many persons the use of a type-writer offers the means of abstaining from all the use of the hand that has brought on the disease. The depression of the keys of a good type-writer resembles the movement of the fingers in playing on the piano, but is less fatiguing, and most sufferers from writers' cramp are able to use such an instrument, even with the affected hand, without any unpleasant symptoms, while the facility with which some instruments can be worked with the left hand enables the right hand, if necessary, to have absolute rest. Unfortunately, a type-writer cannot often be available for the work of the ordinary clerk, who is so frequently a sufferer. For most others it offers a great advantage; in those who suffer from the neuralgic form, however, the pain is often excited by any movement of the arm, and they may then be unable to use a type-writer.*

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* Quills are too small and too smooth to be held with facility; a quill nib should be used in a holder. The best steel pens are those with turned-up points. A good holder is one that is larger than the average size, and is covered with soft india rubber for about two inches, where the pen is held. Large cork holders are also good. The best small holder is one made by Brandauer, of Birmingham, in which the metal part is roughened.

When impaired nutrition of the nervous system coöperates as a cause of writers' cramp, nervine tonics, especially small doses of strychnia, increase a tendency to recovery, and in all cases may be given as having an influence in the right direction. Sedatives internally, as a rule, effect little in cases of pure cramp, although hypodermic injections of atropine (one-sixtieth of a grain three times a week) have been strongly recommended by Vance. In the neuralgic form sedatives internally and locally are certainly of service. Indian hemp by the mouth, morphia beneath the skin, or inunctions of extract of belladonna and glycerine, or aconite ointment, are the most useful. Probably cocain would also be useful in some cases. When tenderness of the nerves exists, counter-irritation by blisters over the tender points often does considerable good.

Electricity has been extensively used, and strongly recommended, in the treatment of writers' cramp. Faradaism is of no value when there is spasm, although it seems to have been useful occasionally in the rare cases of the paralytic form (Duchenne). In the neuralgic variety it is harmful, while voltaic electricity sometimes gives marked relief, as in other forms of neuralgia. In the spasmodic form Erb recommends the passage of the voltaic current up and down the cervical spine, and from the spine to the muscles. Poore advises the combination of the latter method with gymnastic movements of the hand, and has observed great improvement in some cases from this treatment.† My own experience has led me to assign a low position to electricity in the treatment of the spasmodic form, although I have given it a thorough trial in many cases. Certainly, if the patient goes on writing, it has not the slightest influence on the disease. With rest from writing, there is, in many cases, a tendency to recovery which has not appeared to me to be expedited by galvanism. Gymnastic exercises are often distinctly useful, regular flexion and extension movements of the fingers and hand, for which with advantage a "dumb piano" may be used. Rubbing, and the combination of rubbing and kneading that is called massage, often gives considerable relief to discomfort, and sometimes lessens the tendency to spasm. It has been recently made the subject of public attention, and has been said to give remarkable results, but it is probable that some of these were obtained in cases of the imaginary disorder. While there is no doubt that the treatment often lessens discomfort, in the cases of undoubted writers' cramp that I have known to be treated in this manner the symptoms have not been appreciably influenced.

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* I have given some attention to the relative characteristics of the various forms of type-writer in regard to their usefulness in cases of disease. They may be divided into two classes, the high-priced key machines, which cost twenty guineas, and the lower priced machines, of different construction, that cost five guineas. The key machines are the well-known "Remington," the "Calligraph," and the "Hammond." The two first are similar in construction, but the "Calligraph" is distinctly the better for medical purposes, because there is a separate key for each sign, whereas in the "Remington," the use of "controlling keys" is necessary in the case of figures and capitals, which diminishes the convenience and speed with which the instrument can be worked with one hand. The same characteristic lessens the suitability of the "Hammond," and in it, moreover, the arrangement of the keys over a very wide space, and the greater force needed to depress them, make it less suitable for cases of disease. The cheaper machines are the "Hall," and the "Columbia." Each can be worked with either hand, but the speed attainable is not half that of the key machines. The "Hall" is easy to use, but does not turn out very satisfactory work. The "Columbia" is certainly the best of the cheaper machines, but at first it fatigues the fingers. The fatigue soon ceases to be felt, and it can be worked with various muscular actions of the fingers or wrist, but it is scarcely suitable for a case in which the tendency to pain is considerable, or in which there is a tendency to spasm in the left hand.

† "Handbook of Electricity."

Among other means of treatment which has been recommended is tenotomy (Stromeyer). In one case (Truppert) no less than fifty tendons were successfully divided, and it is said that the patient at the end of the treatment was slightly improved. I have found no good result from rendering the arm for a time immobile by a plaster-of-Paris bandage.

OTHER OCCUPATION NEUROSES.

The general considerations regarding the pathology, causation and treatment of writers' cramp, are applicable, *mutatis mutandis,* to all the other forms of occupation neurosis. In each, pain and spasm may be variously combined, and are at first related solely to the special act.

Pianoforte players' Cramp occurs chiefly among professionals, and more frequently among women than among men. Both the spasmodic and neuralgic varieties are met with. In the former a finger or the thumb does not move with the necessary precision, and may remain extended for a few seconds instead of striking the required note. In the neuralgic form, pain comes on after playing for a short time, and compels the sufferer to desist. Sometimes the pain extends up the arm. Erb has several times met with pain between the scapula and the spine. I have seen one case in which the pain was symmetrical in the outer border of each hand, sometimes extending down the little finger, but never accompanied by cramp, although the patient was liable to the nocturnal cramp in the hands described at p. 1054.

Violin players are liable to a similar affection in either hand, that which fingers the strings or that which moves the bow, and occasionally in both.

Seamstresses' Cramp.—The act of sewing frequently leads to a similar affection in seamstresses and tailors,* usually characterized by rigidity and cramped flexion of the fingers of the right hand, often greatest in the thimble finger. Tingling in the fingers may accompany it, and may cease as soon as the attempt to sew is relinquished. I have met with the same trouble in a sailmaker. In this occupation the needle is held between the finger and thumb, and is pressed through the canvas by a metal plate fixed on the ball of the thumb. The spasm in the fingers caused the needle to slip off the plate, and the attempt to work occasioned pain in the fingers, and in the metacarpo-phalangeal joints.

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* The disease known as tetany was in Germany mistaken for an occupation neurosis of tailors and cobblers, and called "Schusterkrampf." It seems to be rather more frequent in Germany among these workers, although the reason is not clear, since it is very different, in the bilateral distribution and spontaneous onset of the spasm, from any occupation neurosis. A case which is clearly one of tetany is described by Romberg in his account of writers' cramp; every attempt to write brought on the bilateral spasm, but attacks also occurred apart from movement. The patient was a boy, eight years old.

Telegraphists' cramp (first described by Onimus) affects exclusively those who use the Morse machine, which is worked by pressing down a stop with the finger, so as to make a dot or a dash according to the duration of the pressure. The pressure has to be repeated for each sign, and telegraphists have often to send 50,000 separate signs a day. Spasm is set up, which causes the pressure to be too long maintained, and hence dots become transformed to dashes. One patient managed to go on for two years by substituting his thumb for the finger, and then this became unmanageable; afterward he used the middle finger and then the closed fist, but the spasm followed, and he had to give up his occupation. The neuralgic variety is sometimes met with, and I have seen one case in which the pain, for six months excited only by the special work, afterward occurred on other movements. After six months' rest the patient resumed work as a writing clerk, and wrote in comfort for two months. Then the pain returned in connection with writing, rapidly increased, and became independent of muscular action and spontaneous.

Smiths are also, although rarely, liable to cramp. In one case, an artisan who was accustomed to work metal with a hammer and chisel, found that his left hand, grasping the chisel, gradually passed into a state of tremulous cramp, and became supinated in spite of his effort to prevent it. The spasm was worst when the elbow was flexed, and for a time he was able to work with the elbow extended, but at last he had to relinquish his occupation. In another case, a metal-chaser, who worked with a four-pound hammer, found that, after using it for about an hour, clonic spasm came on in the biceps, and interfered with his work.*

Among other occupations which have been known to lead to the development of cramp and have given names to special varieties, are those of painters, harpists, artificial flower makers, turners, watchmakers, knitters, engravers (in using the burin, Hammond), masons (in using the trowel), compositors, enamellers, cigarette makers, shoemakers, milkers, money counters (Beard), and zither players.

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* Some cases of hemiplegia among Sheffield smiths were described by the late Dr. Frank-Smith as due to the use of the hammer, and termed by him "hephæstic hemiplegia" (*Lancet*, *1869*, i, p. 427). But it is quite certain that most of the cases he records were merely cases of organic disease of the left hemisphere occurring in smiths, and it is doubtful whether any one of his cases have any real relation to the use of the hammer. "Hephæstic hemiplegia" is probably a purely fictitious disease.

In rare cases two actions are simultaneously and equally affected—writing and some other. The most common combination is writing and piano playing in ardent amateur musicians; occasionally writing and sewing are both equally deranged.

EPILEPSY.

The term "epilepsy" is applied to a disease in which there are convulsions of a certain type, or sudden loss or impairment of consciousness, but in which the convulsions are not directly due to active organic brain disease, to reflex irritation, or to abnormal blood states, and in which the loss of consciousness is not due to primary failure of the heart's action. In most cases the change in the brain that causes the convulsions is not visible to the naked eye, and hence the condition is often termed "idiopathic epilepsy." There are cases, however, in which similar convulsions occur as the result of old brain disease, such as a spot of softening which has long ceased to cause active irritation. These cases may resemble epilepsy in their course, and sometimes in their symptoms, but they commonly succeed a distinct attack of hemiplegia. Hence they are often termed "post-hemiplegic epilepsy," and are described in a separate section.

The convulsions in epilepsy may consist of tonic or of clonic spasm, or, commonly, of both, but the attitude of movement which results always differs from that which occurs in voluntary movement. On the other hand, in the convulsions of hysteria, the muscular spasm is so arranged as to resemble that which may result from a voluntary action.

Etiology.—After one attack of epilepsy, others usually occur without any immediate discoverable cause. Each fit apparently leaves a change in the nerve centres, facilitating the occurrence of other fits. The immediate or exciting cause of the disease must be sought, therefore, in the cause of the first fit. But when such a cause can be discovered, it is usually manifestly inadequate to produce a convulsion by itself, and can only be effective in consequence of a powerful predisposition. The remote influences to which the disease is due are, therefore, its chief causes. The excitant is no more than the spark which causes a conflagration, rendered possible only by the chemical constitution of the materials ignited.

Remote Causes.—Sex.—Females suffer from epilepsy rather more frequently than males, in the proportion of about six to five.* An inherited tendency (indicated by the presence in ancestors or collateral relations of epilepsy itself, or of insanity) is traceable in rather more than a third of the cases (35 per cent.), and rather less frequently in males than in females (33 per cent. males, 37 per cent. females). There is a family history of epilepsy in two-thirds of the inherited cases; of insanity in one-third; of both in a tenth. When there is an inherited tendency, the females of a family are rather more likely to suffer than the males. This is due to two circumstances; the inheritance is rather more frequently from the mother's side than from the father's, and more females suffer when the heredity is maternal, more males when it is paternal. The inheritance is less frequently from the mother personally than from the father, but this is due to the fact that insanity is much less common in the mother than in the father; epilepsy is equally common in the two. Occasionally many members of a family suffer; in one instance no less than fourteen were epileptic, the cases being distributed through four generations. It is very doubtful whether any inherited morbid state, outside the nervous system, predisposes to epilepsy. A proclivity to phthisis has been thought to do so, but the evidence of statistics shows that this is an erroneous inference, due merely to the great frequency of consumption in the community. Idiopathic epilepsy, however, sometimes occurs in the subjects of inherited syphilis, without other cause being traceable.

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* These statements are based on 1450 cases, the detailed analysis of which is given in a separate work ("Epilepsy and other Chronic Convulsive Diseases," London, 1881).

Age.—Three-quarters of the cases of epilepsy begin under twenty years, and nearly half (46 per cent.) between ten and twenty, the maximum being at fourteen, fifteen and sixteen. One-eighth of all cases commence during the first three years of life. After twenty, the annual number gradually falls, but cases commence even in old age (over seventy). In the cases which commence under thirty, females exceed males, in the first ten years by 6 per cent., in the second by 18, and in the third by 12. After thirty the relation is reversed, and the excess of males gradually increases, until after sixty the malady practically begins in males only. It has been said that heredity can be traced only in the cases which commence under twenty years, but this is altogether erroneous. Most cases of hereditary epilepsy begin before twenty, chiefly because most cases of all kinds commence in this period; the proportion borne by the inherited to the non-inherited cases is considerable throughout life. In the first twenty years of life it is nearly 38 per cent., in the second 34 per cent., and in the cases which commence after forty it is 26 per cent. I have known inherited epilepsy to commence at sixty-five and seventy-one.

Immediate Causes.—We must regard, as the exciting cause of epilepsy, that to which the first fit was apparently due; and its influence, as already stated, is no doubt incomparably smaller than that of the predisposition due to conditions involved in age and heredity. The statements of patients regarding exciting causes have to be received with caution. Excluding the cases in which the relation of the supposed excitant was doubtful, and excluding, also, the cases originating in infantile "dentition convulsions," some exciting cause can be traced in only 37 per cent. of the cases. In males it is met with more frequently than in females, and the difference between the sexes in this respect is least in childhood, and is greatest in the second decade and in the decline of life. In women between ten and forty, exciting causes are relatively infrequent.

Of the cases that commence in infancy, at least three-quarters date from infantile convulsions, ascribed to teething. Such convulsions are known to be the result, in most instances, of the condition of defective development ermed rickets. Whether this condition is entirely preventable or not, it ould certainly not attain the degree in which it causes convulsions, if hildren were properly fed, and thus a considerable number of cases of epiepsy might, without doubt, be prevented.

Mental emotion, fright, excitement, anxiety, is the most potent exciting ause of epilepsy. The most frequent form is fright, which is powerful hiefly in childhood, and at the transition to adult life, while it is very rare fter middle life. As might be anticipated, in childhood it is equally effective n each sex, at puberty it is most effective in females, and after twenty it is eldom traceable in men, but is still a relatively frequent cause in women. he profoundly disturbing effect of alarm on the nervous system, deranging, s it does, almost every function, enables us to understand its frequency as n excitant of epilepsy. There is usually an interval between the fright and he first fit. Fright excites hysteroid as well as epileptic fits, and in most f the cases in which a person is immediately "frightened *into* a fit," the ttacks are hysteroid. The longer the interval, the larger is the proportion f cases in which the attacks are distinctly epileptic. Other forms of mental xcitement occasionally excite the first fit, and most commonly at the period f life, and in the sex, in which fright is chiefly influential, but one emoional cause, prolonged mental anxiety, is effective chiefly in men, and late n life.

Blows and falls on the head, which cause no symptoms of coarse cerebral njury, are sometimes followed by recurring convulsions having all the haracters of idiopathic epilepsy. Such cases are met with at all ages, n childhood equally in the two sexes; but in adult life most frequently in nales, doubtless from the risks of occupation. Exposure to the heat of the un is frequently assigned as the cause of the first fit, but often merely ecause the fit occurred in summer sunshine, and without adequate evidence hat the attack was thus caused. In some cases, chiefly males, its influence s probable, especially when the fits follow other distinct symptoms of suntroke.

Acute disease is an occasional cause of idiopathic epilepsy, but the cases scribed to this influence need careful scrutiny, because "post-hemiplegic pilepsy" often results from a sudden lesion of the brain during an acute pecific disease, probably thrombosis in a surface vein, or of embolism conequent on endocarditis (see p. 840). Excluding such cases, epilepsy is a equel especially to scarlet fever, measles and typhoid fever, in that order of requency, and more cases are consecutive to scarlet fever (apart from the nfluence of nephritis) than to all the other acute diseases put together. his, together with the fact that optic neuritis may follow scarlet fever, ithout any organic change in the brain to cause it, seems to show that the carlet-fever poison has a special action on the nervous system.

Intestinal worms are a cause of reflex convulsions, and these do not always ease when the worms are expelled. They may continue as idiopathic pilepsy, apparently as a result of a change in the nervous system consequent n the primary convulsions. Among other rare causes of the first fit are severe digestive derangement, asphyxia, chronic alcoholism and chronic lead poisoning. In the two latter, the convulsions are primarily due to the toxic influence, but may persist when the latter is lessened or removed by treatment. Analogous convulsions are sometimes met with in chronic renal disease, apart from any acute symptoms of uræmia.

Disturbed or delayed menstruation, at the time of puberty, often coincides with the onset of epilepsy, but it is very difficult to determine the exact relationship between the two. As a rule, when epilepsy is once set up, the establishment of regular menstruation appears to exert no influence on the disease. The fact that so many cases commence at the time of puberty seems due rather to the developmental changes in the nervous system at this epoch than to any direct influence exerted by the organs of generation. No case has come under my notice suggesting that uterine or ovarian disease can be regarded as a cause of epilepsy. Occasionally, the first fit occurs during pregnancy, or after parturition, without any secondary cause to which it could be ascribed.

Masturbation in boys is undoubtedly an occasional cause, but the attacks which result from this influence are more frequently hysteroid than epileptic, or present a hybrid character, so that it is difficult to say in which category they are to be placed.

Syphilis is a frequent cause of chronic convulsions, but these are usually due to organic disease, a meningeal growth or chronic inflammation. Fournier believes that the syphilitic poison, during the secondary period, has a direct action on the nervous system, and may cause pure neuroses, epilepsy among the number. Evidence of this, that will bear a close scrutiny, is scanty. Convulsions may be the only sign of organic changes, which sometimes occur early in the course of syphilis. The occurrence of epilepsy, apparently idiopathic, in the subjects of inherited syphilis, has been already mentioned.

Symptoms.—Epileptic attacks are commonly divided into two classes, "major," or severe, and "minor" or slight. These two forms, although clearly distinguished in their general characters, are not separated by any sharp demarcation. In the major attacks (*grand mal*) there is loss of consciousness and severe muscular spasm. In the minor attacks (*petit mal*) there is usually brief loss of consciousness, often without any muscular spasm, sometimes with slight spasm, and very rarely there is slight spasm or some sudden sensation without loss of consciousness. In severe attacks the patient, if standing, falls to the ground (hence the old English name, "falling sickness;") in slight attacks he may or may not fall. In very severe fits, muscular spasm and loss of consciousness are simultaneous in onset, but in less severe fits the muscular spasm may commence before consciousness is lost; the patient is then aware of the onset. Still more frequently the spasm and loss of consciousness are preceded by some sensation. The sensation, or commencing spasm, which informs the patient of the oncoming attack, constitutes the "warning" or "aura" of the fit.

Major Fits.—At the onset of the severe fit the spasm is tonic in character, rigid, violent, muscular contraction fixing the limbs in some strained position. There is usually deviation of the eyes and rotation of the head toward one side, and this rotation may involve the whole body, and even cause the patient to turn round, sometimes two or three times. The features are distorted; the color of the face, unchanged at first, rapidly becomes pale, then flushed, and ultimately livid, as the fixation of the chest by the spasm stops the movements of respiration. The eyes are open or closed; the conjunctiva is insensitive; the pupils dilate widely, as cyanosis comes on. As the spasm continues, it commonly changes in its relative intensity in different parts, causing slight changes in the position of the limbs. When the cyanosis has become intense, the fixed tetanic contractions of the muscles can be felt to be vibratory, and the vibrations gradually increase until they amount to slight visible remissions. As these become deeper, the muscular contractions become more shock-like in character, and the stage of clonic spasm is reached, in which the limbs, head, face and trunk are jerked with violence. The shocks of spasm effect slight movements of the thorax, so that air is expelled, and saliva is frothed out between the lips. The air that enters the lungs is at first insufficient to lessen the lividity, and the patient may seem to be at the point of death, but as the remissions become deeper, more air enters the chest, and the lividity gradually lessens. In becoming less frequent, the contractions do not become less strong, and the last jerk is often as violent as those which have preceded it. At last the spasm is at an end, and the patient lies senseless and prostrate, sleeps heavily for a time, and then can be roused. Urine frequently, and fæces occasionally, are passed during the fit.

In some cases the spasm is more deliberate in its onset. Instead of commencing simultaneously in all the muscles of the body, it begins in one region, as the face or arm, and thence spreads, first to the limbs on the same side, the head and eyes being turned toward that side, and then, lessening on the side first affected, it invades the limbs on the opposite side, with a corresponding rotation of the head. Such attacks may commence with tonic spasm, or they may commence by, and sometimes consist only of, clonic spasm. This form of convulsion is that which (as we have already seen, p. 524) is most common in organic cerebral disease, such as tumor, but it is also occasionally met with in idiopathic epilepsy. In such cases consciousness is often lost late, so that the patient is aware of the commencing spasm.

Minor Attacks.—The slight attacks of epilepsy may be characterized only by loss of consciousness. The individual suddenly stops in his occupation, looks strange for a moment, and then goes on with what he is doing, may even finish a sentence that he had commenced, and be aware of what has happened only by finding that he has dropped something which was in his hands, or that he is the object of anxious observation. Sometimes there is pallor at the moment of the attack, more frequently immediately after it, and very commonly the face subsequently becomes flushed. Very often, moreover, the slight seizure is heralded by some sensory warning or aura, such as precedes the severe attacks. There may also be slight visible spasm, such as putting the arms forward and bending the head down, or there may be slight convulsion in the part in which the spasm commences in the severe attacks, and, in some cases, the minor attack may consist only of such spasm, without loss of consciousness. After a slight attack is over, the patient may be at once quite well. Often he is stupid and dull for a little time, and sometimes proceeds to perform some action in a dream-like, automatic manner, such as undressing himself, retaining afterward no recollection of what he has done. Occasionally this stage is attended by passion and violence, a brief maniacal condition constituting one form of epileptic mania. After an attack, severe, or more frequently slight, the patient may pass into a state of actual hysteroid convulsion, and in such cases the minor epileptic seizure may be unnoticed, all the obtrusive phenomena of the attack being of a hysteroid character.

SYMPTOMS IN DETAIL.

Precursory symptoms occasionally precede a fit for some hours or days, and indicate to the patient or his friends that an attack is impending. The most frequent of these indications are sudden jerks of the body or limbs, persistent giddiness, flashes of light before the eyes, irritability of temper, or an unnatural degree of appetite.

Mode of Onset; Warning; Aura.—The word *aura* (vapor) originated with the old Greek theory that the fit began by the ascent of a vapor up the vessels of the limb. In later times it was thought that the sensation felt in the periphery does begin there, as a disturbance ascending the nerves, and the opinion was thought to be confirmed by the fact that a ligature around the limb would arrest the ascending aura. But the fact (observed first in the beginning of this century) that the commencing fit may be equally arrested when its cause is a tumor of the brain, has shown that the aura is merely the effect on consciousness of the commencing discharge in the brain, either a direct effect, felt as a sensation and referred to the periphery, or an indirect effect, due to actual spasm at the periphery. But the fact that the aura is the result of the commencing central process renders its study of great importance, since it gives us information of the functional region of the brain in which the process of the fit begins.

Some form of aura is met with, at least occasionally, in about one-half of the cases of epilepsy. In the other half the loss of consciousness always occurs so early that the patient is unaware of the onset of the fit. We may provisionally group the various auræ into certain classes, for convenience of description. The classification must be, in the main, empirical.

(1) The commencement of the attack may be by a motion or sensation in some part of one-half of the body, most frequently in the arm, less frequently in the face or leg, occasionally in the tongue, very rarely in the side of the trunk. Such local commencement is not frequent in idiopathic epilepsy, although it is sometimes met with; it is the common mode of onset in cases of organic brain disease. In any part, the first symptom may be a spasm, or a sensation, or both. In the upper limb, the aura usually commences in the hand. When it is spasm, it generally commences in the hand

as a whole, less commonly in the arm as a whole, rarely in the shoulder. When a sensation, it is usually described as "numbness," "tingling," etc., and commences generally in a definite part of the hand—thumb, forefinger, all the fingers, palm, or wrist; never higher up the arm. Sometimes there is a sensation of motion without actual spasm. The aura, whether motor or sensory, if it commences in the hand, may be felt to ascend the arm and may pass to the head, trunk, or leg, before consciousness is lost. Commencement in the leg is less frequent and presents the same variations in character and seat; the sensation or spasm may pass, before consciousness is lost, up or down the limb (according to the place of commencement), or to the trunk, arm, or head. When the aura passes from the arm to the leg, or *vice versâ*, it may do so by two modes; it may pass from one limb to the other through the trunk and then pass *down* the limb secondarily affected, or it may commence at the extremity of the second limb and thus pass *up* both. In the former case, as a rule, a pure sensation leads; the course of the aura is determined by the discharge in the sensory centre, in which no doubt the representation of the cutaneous nerves is as continuous as their distribution in the skin. Sometimes a purely sensory aura (discharge limited to a sensory centre) may pass up one limb, along the side of the trunk, and down the other limb, and only when it reaches the extremity of this latter is spasm added, which may pass back up the limb, the discharge extending to the related motor centre only when it reaches the limit of the sensory centre.

Fits begin in the face much less frequently by a sensation than by spasm; the latter is generally in the zygomatic muscles, sometimes in the orbicularis. In slight attacks on the right side, the commencement in the face is often associated with inability to speak. An aura in the tongue is usually sensory; it may be associated with movement in the jaw or with nausea, and in these combinations we may trace physiological associations.

(2) Bilateral and general warnings. Fits occasionally commence by a sensation or motion in the limbs on both sides simultaneously. An aura referred to the trunk and not distinctly visceral is rare, and is generally referred to the spine. General tremor or shivering, and general starts or jerks, are occasional warnings, and so are a general sense of malaise, of powerlessness, of heat, and a feeling of faintness.

(3) Visceral and pneumogastric warnings. A frequent form of aura is that which is referred to the internal viscera, and especially to those which are in the region of the pneumogastric nerve, including the respiratory portion of the spinal accessory. The most common is some sensation at the epigastrium, usually vague but sometimes actual pain. Rarely it seems to commence lower in the abdomen; occasionally it is referred to the left of the middle line, scarcely ever to the right. When the sensation is actual pain, it remains at the epigastrium until consciousness is lost; it is sometimes associated with nausea, and seems referable to disturbance in the central relations of the gastric division of the vagus. When the sensation is other than pain, it may also only be felt at the epigastrium, but frequently it seems to ascend through the chest to the throat or head. On reaching

the throat there is a sensation of choking, apparently identical with the globus hystericus. The warning may also be this sensation of choking without any preceding epigastric aura. Now and then other vague feelings are described in the throat or thorax. These auræ, ascending to or felt in the throat, seem to be the expression of a disturbance of the central processes connected with the respiratory function of the pneumogastric. Another organ within the innervation of the vagus is the heart, and various cardiac sensations, such as palpitation, or pain, or vague discomfort, sometimes constitute the aura.

(4) Cephalic sensation. A sensation of giddiness, vague or definite vertigo, is a very frequent aura, and is often associated with actual turning of the head and eyes, seldom of the whole body, more frequently to the left than to the right. In bilateral fits the convulsion is never quite equal on the two sides, and the deviation of the head is apparently the result of the inequality of discharge; the vertigo may be its sensory effect. Giddiness is occasionally associated with nausea and sometimes with loss of sight. Other sensations referred to the head are also common; actual pain, usually indefinite in seat, sometimes a sense of "rushing of blood to the head," or vague "heaviness." A sudden sense of somnolence is also sometimes described.

(5) Psychical auræ occasionally herald attacks, and may consist in an emotion or an idea. The emotion is uniform in the same case, some form or degree of fear. It bears no relation to emotion as a cause of the first fit. The idea is usually too vague or imperfectly remembered to be described in words. Occasionally the psychical aura consists of a vague dreamy state, which Dr. Hughlings Jackson has designated "voluminous;" sometimes it is a sudden sense of strangeness; familiar objects seem unfamiliar. Very rarely there is a sudden sense that whatever is happening is morally wrong.

(6) Special sense auræ are very frequent and important. Olfactory sensations are rare; are usually unpleasant in character. Gustatory auræ, a sour, bitter, or metallic taste, are still more rare. An auditory warning is more common. There may be a sudden loss of hearing—a strange stillness, for a moment, before consciousness is lost. More frequently there is a warning sound, usually of low elaboration—a crash, whizz, a hiss or whistle, and the sound may seem to get louder and nearer until the patient knows nothing more. Very rarely a more elaborate sensation is described, or music or words, and the sensation is usually the same before each fit.

A visual warning is twice as frequent as all the other special sense auræ together. It may be sudden loss of sight; but more frequently it is a visual sensation, a flash of light, or sparks, or flashes of color. Usually many colors are seen, sometimes one only. Red and blue are the most frequent; no other color is seen alone, and both are never absent. Occasionally the visual sensation is much more elaborate, a vague vision of some beautiful place, or a definite image of some object, an old woman with a dress of a certain color, ugly faces, animals, etc. Objects actually before the patient may appear to approach, or more frequently to recede, as the fit is coming on. The recession is apparently due to a slight degree of the inhibition of the visual centre, that, in greater degree, causes the loss of sight. It is noteworthy that lights may be seen after sight is lost, an instance of "discharge" in an inhibited centre, and an important fact in connection with the physiological pathology of the disease. Various special sense auræ may be combined.*

Very rarely an attack is preceded by some coördinated movement. A patient, for instance, may invariably commence running, and after running a few yards, fall down in the fit—the "epilepsia cursiva" of Bootius. I have known a patient always to turn round and retrace his steps immediately before an attack, and another would spring up and jump about the room for a few seconds.

A peculiar sound, the "epileptic cry," is produced at the onset of the attack in some cases. It may be a harsh, weird scream, but more often it is a sort of prolonged laryngeal groan, not very loud, but very characteristic. The sounds are probably due to narrowing of the glottis at the moment when air is expelled from the chest by the tonic spasm. As a rule the patient is not aware of the cry, but sometimes he can hear and remember it, although he cannot prevent it.

The convulsion, as a rule, begins by tonic spasm, which causes rotation of the head and deviation of the eyes and of the mouth to the side on which the convulsion is most intense. The posture of the limbs varies. Commonly the arms are slightly abducted at the shoulder, the elbow and wrist are flexed, and the fingers are flexed at the metacarpo-phalangeal joints, extended at the others, the thumb being adducted into the palm, or pressed against the fingers. The position is thus nearly that seen in tetany. The legs may be extended, but often there is slight flexion at the hip and knee joints. Sometimes initial extension of the legs gives place to strong flexion in the later stages of the fit. Usually the limbs of the two sides do not perfectly correspond in position, but the difference is slight. In other cases, the arms are extended, and in some the fingers are flexed at all joints, the fists being "clenched." In others, again, instead of a combination of flexion and extension, flexion predominates throughout. The head is bent forward, the arms and legs are strongly flexed, so that the fists are in contact with the chest, and the knees with the abdomen. At the commencement of such flexor fits the patient often falls forward. In rare cases the arms are raised up above the head at the onset of the attack, and may be kept in that position throughout, or put straight forward. The neck, in these fits, is often bent backward, the legs may be extended, or one or both may be flexed. The violence of the tonic spasm is often very great; the shoulder may be dislocated by it, and when this accident has once happened it is very apt to recur. It is generally during the stage of clonic spasm that the tongue is bitten; it is pushed by the spasm between the teeth, while the jaws are jerked by the contraction in the muscles of mastication. Very rarely it is bitten during the tonic stage. Now and then, instead of this shock-like clonic spasm, into which the tonic spasm gradually passes, the clonic spasm is finer in range and quicker in time, and is superadded to, instead of superseding, the tonic spasm. The latter continues while the strained rigid limbs are agitated by the coarse tremor. This form of clonic spasm is most common in the cases which present extension in the arms and flexion of all the joints of the fingers.

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* Many illustrations of the different kinds of warning will be found in my treatise on "Epilepsy."

In some epileptic fits there is but one form of spasm, either tonic or clonic. As a rule, those which consist only of tonic spasm are general fits of slight severity. A patient falls unconscious, is rigid for a few moments, and then is better. Severer tonic fits are sometimes seen—the "tetanoid epilepsy" of Pritchard.

The attacks which consist only of clonic spasm are slight fits of partial distribution, beginning in, and often confined to, one limb, especially the hand and arm. They are less common in cases having the character of idiopathic epilepsy than in organic brain disease. Severer fits consisting only of clonic spasm are very rare.

The course of the spasm in fits which begin locally has been alluded to in the account of the modes of onset. The relation of the spasm to the muscles of unilateral and bilateral use is an important point, which has been already considered (p. 524).

The slight convulsion at the onset of fits which begin deliberately seems to cause little pain, but occasionally the pain felt in such cases is very great. In some of these cases it is probable that the pain is due to discharge in the sensory centres.

Other Symptoms of Attacks.—The pupils are said to be occasionally contracted at the onset of a fit, but such contraction is certainly not invariable. As a rule, as soon as the tonic stage is well established, the pupils are dilated, and continue so until the fit is over; the iris does not contract to light. When signs of consciousness can be elicited, the dilatation ceases, and it is not uncommon after the fit for the pupil to present alternate contraction and dilatation every one or two seconds, continuing for a few minutes, a phenomenon first pointed out by Reynolds.

The passage of urine during an attack, already mentioned, is not due merely to the state of the bladder or rectum, or to the loss of consciousness, but is the result of some peculiarity in the convulsion, since it occurs invariably in some patients, never in others. The pulse may be feeble at the onset, but I have never noted an initial failing, although I have several times had my finger on the pulse when a fit came on. A tracing published by Voisin shows that the heart's action may be perfectly normal during the stage of the aura. A stoppage of the pulse has been described by Moxon, but is certainly altogether exceptional. As the muscular spasm becomes considerable the pulse is increased in frequency and in force, but falls when the fit is over. The face is often pale at the onset, but pallor is less constant than is currently asserted. It may come on after and not before the commencement of the tonic spasm, but as this continues the face becomes congested and then cyanotic. The bloated dusky aspect of the face, with features distorted by the spasm, renders the aspect of the patient most alarming to those unaccustomed to the disease. When the clonic spasm becomes completely intermittent, air is changed in the lungs and the cyanosis lessens. During the course of the fit, the surface often becomes covered with sweat. It is doubtful whether there is any change in the retinal vessels until the veins become swollen in the course of the venous congestion. In convulsions which begin locally, I have watched the fundus and have seen that there is no initial alteration.

Excitants of Attacks.—In some patients attacks may be excited by certain influences, but such cases are as rare in epilepsy as they are common in hysteria. The excitants which have come under my notice are emotion, a "startling" noise, a bright light, and voluntary motion after rest. It is excessively rare for cutaneous irritation to excite a fit. Epileptogenic zones, analogous to those of Brown-Séquard's guinea-pigs, are practically unknown.*

Arrest of Fits.—Attacks which begin deliberately can sometimes be cut short, and the means by which this may be effected are of considerable interest, and of some practical importance. Those which commence by a bilateral, visceral, cephalic or special sense aura can rarely be arrested, but now and then they can be stopped by some muscular exertion, as by walking quickly, or by some strong sensory impression, such as the application of ammonia to the nostrils, swallowing a mouthful of common salt, or by the inhalation of nitrite of amyl. The attacks that can most frequently be arrested are those which commence by a motion or sensation in the hand or foot. The oldest and usual method of effecting this is by a ligature around the limb above the seat of the spasm or sensation. The strong peripheral impression apparently raises the resistance in the nerve cells of the affected part of the brain, and so arrests the spread of the discharge. The influence must be exerted first on the sensory centre, and through this on the motor centre. If the commencing fit has got beyond the part to which the ligature has been applied, the attack is not arrested. A ligature acts better than other peripheral impressions, probably because it is most readily applied, and because the cutaneous nerves are stimulated in the entire circumference of the limb, and so influence the entire extent of gray matter in which the discharge is advancing. Occasionally, a more limited cutaneous impression, a pinch or prick, has the same effect. The fits which begin by spasm may sometimes be arrested in another way—by preventing the movement, and forcibly extending the contracting muscles, sometimes by rubbing them. The mechanism is probably the same. Rubbing has a tendency to lessen all forms of spasm, even that of tetanus. Both methods of arrest were known to the ancients.

Conditions after Attacks.—The coma, into which the patient usually passes at the end of an epileptic fit, often continues as heavy sleep, lasting for a quarter of an hour or longer. After the first few minutes, the patient can be roused. The sleep is often followed by a severe headache, enduring for several hours or the remainder of the day. Occasionally the sleep is not followed by headache, but if the patient is roused and is not allowed to sleep, the pain in the head is severe. In some cases there is neither sleep nor headache.

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* See, however, a curious case recorded by Hughlings Jackson (*Proc. Med. Soc., Lond.*), vol. x, 1887, p. 78).

For a few seconds after a severe fit, reflex action in the limbs remains absent. Tickling the sole no longer causes a movement of the leg, and the loss may involve the muscle-reflex action, so that the knee jerk can no longer be obtained, as Westphal first showed. But this stage of loss is succeeded by temporary increase in the myotatic irritability, so that the knee jerk is excessive and the foot clonus may be obtained. In most cases this increase is alone to be detected. It may be present only on the side on which the convulsion was most severe, *i. e.* on the side toward which the head is turned (Beevor). It lasts a few minutes and then passes away. These phenomena are probably the result of temporary exhaustion of the centres concerned—of the muscle-reflex centre, in the loss of the controlling structures (the terminations of the pyramidal fibres),* in the case of excess.

Loss of motor power, paralysis, may succeed a fit of epileptic type—post-convulsive paralysis, as already described (p. 525). It is most distinct after unilateral convulsions (and constitutes the "epileptic hemiplegia" of Todd), but the general prostration after a bilateral convulsion is probably analogous. After a severe fit it may be due to exhaustion of the nerve elements, but the transient palsy that succeeds a very slight fit must be ascribed to inhibition of the motor centres. In many of these cases there is conspicuous sensory discharge, "tingling," etc., passing, for instance, up the arm and down the side to the leg; there may, indeed, be no motor spasm. In such a case of purely sensory discharge, the arm may be for a time almost powerless. Just as such a discharge in the sensory centre may, as we have seen, set up secondary discharge in the motor centre, so it may, when slight, merely inhibit the centre. Loss of speech sometimes occurs after right-sided seizures, and probably has the same inhibitory origin. These forms of transient palsy must, however, be distinguished from palsy due to cerebral hemorrhage caused by the fit (an excessively rare event), and also from the condition in which convulsions merely attend the onset of an acute cerebral lesion.

Automatic action and hysteroid phenomena are very common after slight attacks of epilepsy, and occasionally succeed a severe fit. For instance; a girl aged seventeen had suffered from six years of age from severe epileptic seizures with tongue-biting; at the age of puberty the attacks, without change in character, were succeeded by hysteroid convulsions. In one that was seen, she suddenly fell with a scream; her head was turned to the left, the limbs were rigid, the face cyanotic, clonic spasm succeeded with frothing at the mouth; the tongue was bitten. The fit lasted two minutes and a half.

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* See p. 146.

She then lay unconscious, breathing heavily, and still frothing at the mouth. Then she opened her eyes, looked strange, kicked and threw her arms about, dashed her head, and arched her back for four minutes. She then went to sleep, and slept for an hour.

The temperature may be raised half a degree or a degree by a severe convulsion, or it may present no alteration. When attacks follow each other at intervals of only a few minutes (status epilepticus), the temperature may rise to 105° or 107° (Bourneville). The strain may cause small vessels to rupture, usually in the face and conjunctiva, so that the face may be spattered, as it were, with small hemorrhagic points. After death in a convulsion similar extravasations may be found in internal organs.

Vomiting occurs after fits in some patients. It is a dangerous symptom, on account of the liability of food to get into the larynx in the state of insensibility. Excessive hunger is an occasional symptom after an attack, and a patient who has had a hearty meal before a fit will eat another directly after it. The urine is rarely altered in its constitution by an epileptic seizure. Now and then a trace of albumen or of sugar may be found, but the frequency of this has been greatly exaggerated; each is rare. There is no necessary increase in the amount of urea. It has been said that every fit causes a loss of body weight (Kowalewski), but this is incorrect (Lehmann, Beevor, etc.).

Minor Attacks.—The slight attacks of epilepsy vary much in character, and hence patients speak of them under various designations, such as "sensations," "faints," "losses," "turns," "giddiness." Hence, also, their nature is often not recognized by the patients or their friends. As a rule, there is brief loss of consciousness, and this, as already stated, may constitute the only apparent symptom. Often, however, there is a slight stoop forward, or a slight quivering of the eyelids. The patient may or may not fall. The color of the face, in many cases, undergoes no change; there is scarcely ever pallor at the moment of onset, but in a second or two the face becomes slightly pale in many cases, and when the attack is over, whether there has or has not been pallor, the face commonly becomes a little flushed. In many patients the loss of consciousness is preceded by some warning sensation, and it is from this circumstance that the minor seizures are so often termed "sensations." The patient may know nothing of the loss of consciousness, and be aware only of the sensation. Very often a warning occurs only before the minor seizure, the severe fits coming on so rapidly that loss of consciousness is the earliest symptom. When each kind of attack is preceded by an aura, this is usually similar in character, although, as already mentioned, it may be more elaborate before the slight than before the severe eizures. It is rare for the aura of each to be altogether different.

The following list comprehends the chief characteristics of minor seizures. They are arranged in the order of frequency. In most cases there s, in addition, brief loss of consciousness. The first of the list occurs n one-third of the cases, and the second in a sixth, so that these two re the characteristics of about half the minor epileptic seizures met with.

1. Sudden momentary unconsciousness, or "fainting" or "sleepiness," without warning.
2. Giddiness.
3. Jerks or starts of the limbs, trunk, or head.
4. Visual sensation or loss.
5. Mental state; sudden sense of fear, etc.
6. Unilateral peripheral sensation or spasm.
7. Epigastric sensation.
8. Sudden tremor.
9. Sensations in both hands.
10. Pain or other sensation in the head.
11. Choking sensation in the throat.
12. Sudden scream.
13. Olfactory sensation.
14. Cardiac sensation.
15. Sensation in nose or eyeball.
16. Sudden dyspnœa.
17. General "indescribable" sensations.

While giddiness is thus one of the most common sensations, it is only present in a small proportion of the total number, and it is a mistake to employ the term "epileptic vertigo" (as it has been employed) as a general designation for attacks of minor epilepsy. The sense of vertigo usually involves an apparent movement, sometimes in the patient, sometimes in objects seen; the direction of these apparent movements is usually the same;* the vertigo is sometimes attended with actual rotation. Special-sense auræ are also frequent in minor epilepsy, and present the characters as already described; the visual sensation is the only frequent one. The epigastric sensation is much less common than before severe fits.

Loss of consciousness is the rule, but exceptions are often met with, in which there is merely obscuration of consciousness for a few seconds, and no absolute loss. In the cases in which the minor attacks consist of sudden starts, or of a visual sensation, consciousness may be apparently unaffected.

Urine is often passed during a minor attack; in such cases there is almost always loss of consciousness, but one patient was always aware of it, although unable to prevent it. In some cases micturition is almost invariable, in others it never occurs. The accident is far more common in females than in males; perhaps because the process can occur more quickly on account of the shortness of the urethra.

In some cases the minor attacks may be accompanied with slight convulsive spasm, tonic when general, but usually clonic when it is local. In such cases attacks occur which present every gradation between the slight and severe seizures. The degree which is the maximum in one case may be the minimum in another.

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* The explanation of the fact will be given in the account of vertigo. It may be well to remind the observer that the term "giddiness" or "dizziness" is often applied to any obscuration of consciousness, and the exact character of the sensation should always be ascertained.

Conditions after Minor Attacks.—The return of consciousness to its average degree often occurs slowly; the patient is dazed and stupid for a few minutes, makes random remarks, and occasionally performs, in an automatic manner, some action of which he afterward retains no recollection. Such automatic action was formerly regarded as constituting the minor attack of epilepsy, and these cases were therefore called "masked epilepsy" (Esquirol), or "epilepsia larvata" (Morel). I believe that the old view is not altogether untrue; that in some cases imperfect loss of consciousness, with automatic action, does constitute the minor seizure, without any initial stage more distinctly epileptic. But it is certain that, much more frequently, the automatism succeeds a slight attack, and is really a post-epileptic phenomenon. It has been ascribed to the temporary failure of the highest centres to control those next below them, which consequently act in an automatic manner (Anstie, Thompson-Dickson, Hughlings-Jackson). The condition is not merely of clinical interest, but also of medico-legal importance, since the performances may be complex, and may have all the aspect of deliberate volition; the initial epileptic seizure may be unnoticed by those around, and even unknown to the patient. One of the most common actions is that of undressing, which is occasionally very inconvenient. It is possibly suggested by a feeling of illness, and so likewise may be another occasional action, an attempt to walk up stairs, which is also awkward if (as happened in two cases) the shelves of a dresser, or the dinner table, is mistaken for the staircase. A very common action, still more equivocal in its aspect, is to put in the pocket any object which may be near, irrespective of its ownership. This gave rise at first to grave suspicion in the case of one of my patients, a draper's assistant, who had gone to a new situation. Very complex actions may be performed in this state. I have known a carman, after an attack, to drive through the most crowded parts of London without any object, but also without any accident. Occasionally the automatic action displays emotion, and even anger and violence. One patient struck a friend who was with him a violent blow on the face, and was in consequence taken to the police station. Another, a woman, immediately after a fit, threw her baby down stairs. Without doubt many crimes have been committed in this state, and the point has formed a medico-legal question at many criminal trials.

Instead of presenting such automatic action, some patients pass, as already mentioned, into a state of violent hysteroid convulsion. This sequel occurs chiefly at the age at which hysteria is met with, under thirty-five. It is most common in young women, frequent in boys and girls, occasional in young men. Hence it is evidently the result, not merely of the preceding epileptic fit, but also of the presence of the cerebral state which underlies other manifestations of hysteria. Some patients have similar attacks at other times, as well as after their epileptic fits. In others there are no separate hysterical phenomena, the hysterical tendency being apparently insufficient to lead to independent symptoms, although it manifests itself during the morbid state immediately after an epileptic fit. As an example of these phenomena may be mentioned the case of a girl, aged fourteen, who had had many severe epileptic fits accompanied by tongue biting, and also slighter attacks followed by hysteroid convulsions. I witnessed one of the latter. When speaking to me, she suddenly stopped, bent forward, and remained still for a few seconds; then she suddenly threw her arms about, stamped with her feet, and became stiff and rigid for a few minutes, in a characteristic hysteroid convulsion. It is often said that attacks in childhood were slight, and at the time of puberty became severe, when further inquiry shows that the severe fits consisted only in the addition of hysteroid convulsion to the slight attacks. There is another post-epileptic action which is automatic, and may occur alone or with hysteroid convulsion, and is important on account of its danger—a tendency to turn over on the face. If the patient is in bed, suffocation may easily occur during the comatose sleep that follows, and, without doubt, many epileptics have died from this cause. Some patients never present this tendency, others always do, and their friends should be warned of its danger.

Mental Disturbance in Epileptics.—The occurrence of transient "epileptic mania" as a sequel to attacks, has been already mentioned. It is uncertain whether such an outbreak may take the place of a fit, but we are not at present justified in denying its possibility. In this condition the patient is often violent and homicidal, and, although perfectly sane in the intervals, is one of the most dangerous of lunatics. The mania is usually brief, often lasting only a few minutes, rarely more than an hour. But occasionally, after a fit or a series of fits, mental disturbance may come on and last for several days, a state of dementia, or of mania, with delusions or hallucinations, often with irritability and violence. It may even occur during the temporary cessation of attacks.

The inter-paroxysmal mental state of epileptics often presents grave deterioration, and this is one of the most serious, and most dreaded, effects of the disease. In its slighter degree there is merely defective memory, especially for recent acquisitions. In greater degree the intellect suffers generally, and there is often defective moral control. Mischievous restlessness and irritability in childhood may develop to vicious and even criminal tendencies in adult life. Every grade of defect may be met with, to actual imbecility. The mental state is not, in all cases, entirely the result of the attacks of epilepsy. In some it is, in part at least, the expression of a cerebral imperfection, of which the epilepsy is another manifestation. In such instances mental defect may exist before the occurrence of the first fit, or may follow attacks very rapidly. In other cases the failure is apparently produced solely by the attacks. It succeeds them in time, and may lessen when treatment renders them less frequent.

A comparison of cases with considerable mental defect, and others in which no mental failure was noted, shows that the deterioration cannot be ascribed to any one element in the disease. The most influential agents are the early commencement of the disease, its duration, and the frequency of attacks. The tendency is greater in the cases that commence in childhood than in those which commence between ten and twenty, and greater in those which begin in the first, than in the second, five years of life. The influence of duration is shown by the fact that, of the cases with mental failure, nearly three-quarters, and of those without, less than half, had lasted more than four years. The influence of frequency of attack is shown by the fact that the average interval in the weak-minded cases was fifteen days, in the others twenty-six days. On the other hand, the influence of form of attack (major or minor) of heredity, and of sex, is not great. Considerable failure is often seen in patients who have only minor attacks, but extended observation does not show that it is more common in such cases than in others. That there is no necessary relation between any of these conditions and mental failure, is shown by the fact that each one may be met with, in high degree, without any impairment of the intellect. On the other hand, there is no condition in which mental deterioration does not occasionally occur. The conclusion is that mental failure is determined less by single conditions than by their combinations, and it is probable that a more potent cause than the attacks themselves is a predisposition to suffer under their influence, a predisposition that is related to the ultimate causes of the disease, rather than to its developed characters.

When attacks that have occurred during many years stop suddenly, whether the stoppage is spontaneous or due to the influence of drugs, patients may become dull, forgetful, sometimes irritable, and sometimes half idiotic. The effect is often ascribed to the remedy used, especially if this is bromide, but it seems to be due rather to the general depression of cerebral function by the condition of the nerve cells, which, by repeated discharge, have developed a tendency to over-production of nerve force, and this is no longer released in the fits. We may not know the exact mechanism, but we can understand the effect. A fit may completely remove the state. It is common for patients to say that they feel better when they are having fits than when they are not.

The general health of epileptics may be perfect. Often there is some defect, especially in the functions of the digestive organs. The circulation is sometimes feeble, the pulse small, unduly frequent, and very often slightly irregular. No abnormal state of the pupil or retinal circulation can be found with any special frequency in the intervals between the attacks.

Post-hemiplegic Epilepsy.—In considerable proportion of the cases of epilepsy the convulsions succeed an attack of hemiplegia, sudden in onset, and therefore presumably due to a vascular lesion. The palsy may lessen or disappear, and the convulsions, continuing years after, may resemble those of the idiopathic form, and are often supposed to be of this nature. Hence it is convenient to consider them here.

Recurring convulsions may follow hemiplegia at any age, but are far more frequent after that which occurs in infancy than after hemiplegia in adult life. In two-thirds of the cases the onset is before five years of age, and in nearly half it is during the first two years of life. In the cases that date from infancy, females are twice as numerous as males; after five years of age there is little difference in the frequency with which each sex suffers. The paralysis, in the infantile cases, is more frequently on the left side than on the right, but after the fifth year it is as frequently on one side as on the other. The conditions of onset and the probable nature of the lesion, in the infantile cases, have been already considered (p. 842). In later life, when there is any indication of the cause of the hemiplegia, this also (heart disease, rheumatic fever, constitutional syphilis, the puerperal state) points to vascular obstruction. In softening from this cause, the brain tissue adjacent is usually damaged by the collateral congestion, and it is easy to understand that the nerve cells may suffer a permanent change in their nutrition and function, causing instability, perpetuated by their repeated discharge. Moreover, acute softening often affects the cortex of the brain, where organic changes most frequently cause convulsions. In a few cases the condition is said to date from birth; in most of these the labor was difficult, and then meningeal hemorrhage, with laceration of the cortex, is probable (see p. 801).

In half the cases, convulsions, often repeated and severe, attend the onset of the hemiplegia. The chronic recurring fits date from the onset in about a third of the cases. In the rest there is an interval before the recurring fits commence, which is usually at least a year, and may be fifteen or twenty years. It is not uncommon for the hemiplegia to occur in infancy, and the convulsions to commence at the epileptogenic period of puberty; sometimes a distinct exciting cause can be traced. When the attacks date from the onset, as a rule this was attended with convulsion, but the converse does not hold good. When the hemiplegia came on in adult life, a long interval is rare; the fits usually commence in less than a year.

The degree of the hemiplegia at the onset varies; it may be considerable or slight; in young children, when trifling, it may be overlooked, the more readily if the child is gravely ill. Still more various is the degree of paralysis which persists. The leg has usually recovered, to a large extent or altogether. The arm is more often weak, sometimes powerless and small; occasionally it presents little or no evidence of the initial weakness. In half the cases the hand presents a state of "mobile spasm," in slight or considerable degree (see page 519), and it is important to look for traces of this in doubtful cases, since it may be distinct when the weakness is not.

The convulsive attacks, in the majority of cases, begin in, and are confined to, the paralyzed side. In rare cases in which the attacks are severe, they may be always general. A distinct warning is present in about five-sixths of the cases, far more frequently than in idiopathic epilepsy; and in half the cases it consists in a deliberate commencement of the attack in some part of the paralyzed side. The other less frequent warnings are for the most part similar to those of the idiopathic form. The spasm is usually also of the same character.

Minor attacks are frequent in these cases, sometimes similar to those of idiopathic epilepsy (a faint, sudden giddiness, etc.), but more frequently they consist of the aura of the severe fit, often without loss of consciousness. Hysteroid seizures are very common in these cases, and so also is considerable mental defect.

Course of Epilepsy.—The disease may commence by severe fits or by slight ones. The latter may exist alone for months or years, and their nature may be unsuspected until a severe fit occurs. The interval between the first and second severe fit is sometimes prolonged, and is a point of considerable importance. When a single convulsive attack has occurred, the anxious question—whether it is epilepsy?—can only be answered by the occurrence or absence of other fits. In one-third of the cases the interval is less than a month; in another third it is between one and twelve months; in the remainder it is more than a year, and it is occasionally ten years. Thus the chance of recurrence does not materially lessen until a year has elapsed.

When the disease is established the interval between severe fits varies greatly. It is less than a month in three-quarters of the cases; in about half it does not exceed two weeks; in about a tenth fits occur daily. Usually the intervals vary, and they correspond to our current divisions of time only in the cases (not frequent) in which attacks in women are related to the menstrual periods.

The attacks may be isolated or grouped; the former is more common. When grouped several attacks occur together, and then there is an interval of freedom. The number in each group varies from two or three to twenty. The patient usually recovers consciousness between each. In rare instances a series of fits occurs in which the patient does not recover consciousness; during the coma, one fit after another comes on. This has been termed the *status epilepticus*, and is a very grave condition. In its most severe form, the intervals between the fits become shorter, the coma deepens, the pulse and respiration become very frequent, and the temperature rises, it may be to 105° or 107° (Bourneville). The patient may die in a state of collapse, from the violence of the convulsions, or, the fits ceasing, he may become delirious, and present symptoms of meningitis, with rapid formation of bed-sores, and may die in this stage. At any period the symptoms may lessen and the patient recover. Many cases, however, end fatally, but fortunately the condition is rare.

Minor attacks occur in less than half the cases of epilepsy. They may occur alone, without severe fits, but both kinds are usually associated. The slight seizures are, as a rule, frequent. In more than half the cases in which they occur at all, there are daily attacks, usually from two to twenty each day, and I have even known as many as two hundred to occur every day. When there are no other attacks, the minor fits are usually frequent, and daily. They may also occur daily when there are severe fits, but when the minor attacks are less frequent, the patient usually has also severe fits. When the latter are frequent, minor attacks are often less frequent. Now and then, when the intervals between the severe fits are more than two weeks, the minor attacks occur for a few days before (less commonly after) the severe fits.

Epileptic fits may come on when the patient is asleep or awake, or in both conditions. They occur in both, or in the walking state only. twice as frequently as in the sleeping state only. Very rarely the patient has them only in the act of going to sleep or of waking up. Now and then they are confined to the early morning. When fits that have occurred only during the night, occur during the day, they usually continue during the night, but if they have previously been diurnal only, and commence in the night, they very often cease during the day. Attacks which have occurred both day and night often cease in the day and continue in the night, but very rarely cease at night, and continue during the day.

In women, attacks often occur at the menstrual period, although when closely investigated the relation is not found to obtain in more than about half the cases. The usual relation is for the attack to occur before or (rather less frequently) during the period, rarely after it. The relation does not seem to be connected with any abnormal state of the uterine organs.

Death is a rare consequence of epilepsy, especially when allowance is made for the frequency of the disease and the alarming aspect of the fits. When it does occur it is scarcely ever from the direct effect of the convulsions, except in the "status epilepticus." It usually results from some accident to which the fit leads, such as a fall into the water or a burn. It may also be due to suffocation produced during the state of coma, either by vomited food getting into the larynx or by the patient in bed turning over on the face.

Pathological Anatomy.—The naked-eye appearance of the nerve centres in epilepsy is, for the most part, that of healthy organs. In cases of long duration there is occasionally slight opacity and thickening of the meninges, and after death, in the *status epilepticus*, there may be signs of meningitis, but these are apparently merely secondary changes. If the patient has died in a fit the post-mortem usually shows signs in all organs of the intense venous engorgement which is so conspicuous during life, and small extravasations of blood may be found, such as are met with in all cases of asphyxia. Of the histological changes revealed by the microscope, most of those which are not common, apart from symptoms, are apparently of secondary origin, the result and not the cause of the convulsions; and the various changes that have been described and have been supposed to be related to the origin of the disease have probably no connection with it and do not even deserve enumeration. It is probable that no greater significance is to be ascribed to the induration of the cornu ammonis (pes hippocampi) to which weight has been attached by Meynert. Nor is there at present much likelihood that our knowledge will be more definite. The changes in the nerve centres are probably of that fine kind which is revealed only by altered function and eludes the most minute research.

Pathology.—In the absence of any help from anatomy and histology the pathology of idiopathic epilepsy is a matter of hypothesis, based on the influence of organic disease in causing similar symptoms, on the results of experiments on animals, and on the indications afforded by a study of the symptoms in the light of cerebral physiology.

It may be premised, as admitting of no question, that the muscular spasm is to be regarded as the result of the sudden over-action ("discharge") of nerve cells, the violent liberation of nerve force, and that the sensations which, in some cases, the patient experiences before losing consciousness, must be due, directly or indirectly, to the same cause (see p. 1083). The problem is how this over-action is brought about and where it occurs.

What is the primary seat of discharge? Experiment teaches that irritation of both the cortex cerebri * and the medulla oblongata † may cause convulsion. The teaching of pathology is, that disease which excites convulsions is most frequently at the cortex, and that whenever organic disease causes convulsions that begin locally the disease is almost invariably at the cortex. In idiopathic epilepsy the convulsions sometimes begin in this manner, and this suggests very strongly that in such cases the change occurs in the cortex.

A further study of the mode of onset throws some light on the question, because the character of the aura indicates the function of the part in which the discharge commences. The auræ that consist in a disturbance of the function of the special sense centres indicate that the discharge commences in those centres, that is, in the case of smell and vision, at least, within the cerebral hemispheres. In all cases in which organic disease causes convulsions so commencing the disease has been in or close to the cortex (see p. 525). The warnings which consist in an intellectual process or an emotion, also point to the cortex as the seat of the primary discharge. Thus, the significance of all the facts regarding the modes of onset that we can interpret is that the discharge in epilepsy begins in the cortex of the cerebral hemispheres. The conclusion is confirmed by rare cases in which a lesion interrupting the internal capsule occurs in a person subject to general epileptic fits. The effect is that the fits, which before were general, almost cease on the paralyzed side.‡ Such cases show that the discharge causing the general convulsions occurred above the internal capsule, *i. e.*, in the cortex of the brain. If there is evidence that so many cases of idiopathic epilepsy depend on discharge of the gray matter of the cortex, it is probable that the conclusion is true of all cases. The wide variety of auræ seems to show that the primary discharge may be seated in various parts of the cortex. The fact that the first discharge may be uniform in character in the same case—may be such, for instance, as to cause a definite and even complex sensation—can only be explained by the theory that the derangement of function in the nerve cells is inherent in them, and it is not the result of any random influence outside them.

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* Ferrier, Luciani, Bartholow, Horsely, etc. † Brown-Séquard, Kussmaul, Nothnagel.

‡ Oebeke has recorded such a case, and I have met with a similar instance, although unconfirmed by autopsy.

All parts of the brain are intimately connected, and the *sudden, i. e.*, instantaneous, derangement of the function of any part appears incompatible with the integrity of consciousness. This is shown by many facts of organic disease. Hence it is intelligible that sudden discharge, wherever it occurs, may cause loss of consciousness. But if there is reason to believe that sudden discharge may occur in any gray matter of the cortex, it may occur in that which subserves directly the phenomena of consciousness, and a sudden alteration in the functional state of these structures may well be, as Hughlings Jackson has suggested, the most probable explanation of the attacks which consist only of such momentary loss.

It is necessary, however, to allude briefly to other theories which have been and are still current regarding the nature of epilepsy. It has been widely held that the morbid action in the brain is excited by arterial spasm causing cerebral anæmia, and also that the convulsions originate from the primary discharge of a convulsive centre in the medulla, but that the loss of consciousness is produced by vasomotor spasm in the brain. In each theory it is assumed that the vasomotor spasm is due to the sudden over-action of the vasomotor centre in the medulla. The evidence on which these theories are based is that cerebral anæmia will cause loss of consciousness and convulsion,* and that at the onset of epileptic fits there is always pallor of the face. But the latter statement is, as we have seen, incorrect. If it were true it would not justify the conclusion drawn from it. We do not infer that the brain is congested whenever the face blushes, and why should we assume that the brain is anæmic when the face becomes pale? There is no necessary correspondence between the condition of vessels in the skin and in the viscera beneath, and probably even less in the case of the brain than of other organs. It is far more probable that the spasm of the vessels of the face is the *result* of the cerebral discharge, just as the arteries of a frog's foot will contract when its brain is irritated. In rare cases in which an early failure of the heart occurs, this may also be the effect of the discharge, which, as it often influences consciousness first through the central connection of the pneumogastric (see p. 1085), may also affect the periphery by the same channel. That sudden anæmia of the brain will cause convulsions is no proof that the fits of idiopathic epilepsy are so produced. Such a theory can give no explanation of the complex character of the commencing discharge, and its uniformity in the same patient.

Thus the vasomotor theory of epilepsy is alike unneeded, unproved, and inadequate. The phenomena indicate that there is discharge of gray matter, and there is nothing to warrant us in going beyond the gray matter concerned in our search for the cause of the discharge; it is certain that this may commence in various parts of the cerebral hemispheres, even in the cortex, and possible that it may begin in lower centres, even the medulla. Epilepsy must then be regarded as a disease of gray matter, most frequently of the gray matter of the cortex.

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* Kussmaul and Tenner.

Can we form any opinion as to the nature of the change in the gray matter which permits the sudden liberation of nerve force? It is necessary t remember that we have direct evidence only of *liberation* of force, but w must recognize, in all nerve cells, a function by which the liberation o energy is restrained, a resistance to action as well as a capacity for action.[*] Of the nature of this resistance we can form no idea, but we cannot conceive of the function of cells without it, and the idea has underlain suc expressions as "nerve tension" and the like, which have been lon employed. The phenomena of epilepsy suggest that the instability of th gray matter, its tendency to discharge, depends on instability of resistance rather than on any primary change in the energy-producing action of th cells, although the latter may be secondarily augmented by the increase demand. Analogy suggests that the internal resistance to action is a highe function of the cells than the production of force. Hence the fact tha there is over-action is consistent with the evidence that there is imperfec nutrition. Moreover, the view that it is the resistance which is unstable enables us to understand the phenomena of inhibition which sometime occur as part of the attack. In a slight degree of disturbance, the resistanc may be suddenly raised instead of being lowered. It is possible that th attacks in which there is loss of consciousness only, may be thus produce —may be analogous to the fits in which there is sudden darkness rather tha to those in which there is a flash of light.

DIAGNOSIS.—The first point in the diagnosis of epilepsy is the recognitio of the occurrence of attacks. Convulsive attacks, which occur in the daytime, are never overlooked, but if an attack occurs during sleep, and it onset does not awaken the patient, he may sleep on when it is over, and b unaware of its occurrence. Often there are some subsequent indications o what has happened. The tongue may be sore, there may be a little bloo on the pillow, an extravasation beneath the conjunctiva, ecchymoses on th face, or a severe headache. These (except the last) are of unequivocal significance, but the patient may be unaware of this, and I have known nocturnal attacks to occur for twenty years, without the patient or his friend suspecting the fact. Minor seizures are also often unrecognized, not becaus they are unnoticed, but because it does not occur to the patient or hi friends that they are of any significance. Those that consist only in a subjective sensation may not be mentioned by the patient. It is usually sufficient to be aware of their common forms, and to inquire for these, in orde to ascertain their occurrence.

Certain forms of minor attack may, however, be confounded with othe paroxysmal symptoms of different nature. The simplest form, in whic there is merely brief unconsciousness, is not only called a "faint," but i often supposed to be actually syncopal in nature. The distinction fro syncope rests, first, on the absence of obvious exciting influences, such a cause ordinary fainting. Epileptic faints often occur when the patient i sitting still, in a cool room, under no excitement. Consciousness is lost more suddenly in epilepsy than in syncope. The latter is usually preceded by a sensation of faintness, but so also, sometimes, are the minor seizures of epilepsy. The presence of this, and of vague "dizziness," palpitation of the heart, nausea, and cephalic sensation other than pain, is of little diagnostic significance. On the other hand, the absence of any warning sensation, or the occurrence of a distinct warning other than those just mentioned, is in favor of the epileptic nature of the attack. A sudden return of normal consciousness is in favor of epilepsy. If a normal condition is slowly regained, there is physical prostration in syncope, mental confusion in epilepsy, and in the former the pulse at the wrist is often scarcely perceptible. The passage of urine during the attack, muscular spasm, however slight, and automatic action or mental dullness after the attack, are certain proof of its epileptic character. In many cases the patient has had other more severe seizures, which assist the diagnosis.

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[*] Handfield Jones, Michael Foster, Ringer, etc.

The attacks characterized by vertigo have to be distinguished from other forms of sudden giddiness. The latter are scarcely ever attended by loss of consciousness, and are usually followed by long-continued vertigo, which persists while the patient is recovering, and slowly passes away. In epilepsy there is generally loss of consciousness, and the patient is either quickly well, or presents some mental dullness after the attack; the vertigo may be associated with some other warning sensation, and there is often micturition. The vertigo most likely to be confounded with epilepsy is that connected with a morbid action of the auditory nerve, "labyrinthine vertigo." In addition to the diagnostic indications just mentioned, there is usually, in this form, slight persistent giddiness in the intervals, persistent tinnitus aurium, and some deafness. In epileptic vertigo there may be an auditory sensation, with giddiness, as the aura of the attack, but there is no persistent tinnitus. It must not be forgotten that auditory vertigo and epilepsy may be associated. I have seen several instances of this.

If a patient suffers from distinct convulsion, the next question is, are the attacks epileptic or hysteroid? If an attack can be witnessed, the nature of the convulsion is usually obvious. The violent tonic spasm and shock-like clonic spasm of the typical epileptic fit, with complete unconsciousness and with cyanosis, and also the brief duration of the attack, are wholly unlike the prolonged tonic contraction, opisthotonos, wild, coördinated movements, quick clonic spasm, perverted mental state, talking, biting, and convergent strabismus of the hysteroid seizure. More difficulty, however, may be presented by the untypical forms of epileptic fits, especially by those which consist only of tonic spasm. Evidence of their epileptic nature is afforded by the brevity of the attacks, their suddenness of onset, their occurrence apart from emotion and when the patient is alone, and the absence of hysteroid symptoms. When an attack has not been witnessed, and the diagnosis has to be made from the description of the patient or friends, it is a much less easy task. The chief diagnostic indications between pure epileptic and pure hysteroid fits, are given, in tabular form, as follows:—

	Epileptic.	Hysteroid.
Apparent cause	none	emotion.
Warning	any, but especially unilateral or epigastric auræ	palpitation, malaise, choking, bilateral foot aura.
Onset	always sudden	often gradual.
Scream	at onset	during course.
Convulsion	rigidity followed by "jerking," rarely rigidity alone	rigidity or "struggling," throwing about of limbs or head, arching of back.
Biting	tongue	lips, hands, or other people and things.
Micturition	frequent	never.
Defecation	occasional	never.
Talking	never	frequent.
Duration	a few minutes	more than ten minutes, often much longer.
Restraint necessary	to prevent accident	to control violence.
Termination	spontaneous	spontaneous or induced (water, etc.).

Especial care should be taken, in asking about the character of the convulsion, to avoid a leading question, and if suggestions are indispensable to put them alternatively, thus, "Should you say that the patient *struggle* or *jerked* during the fit?"

If there is evidence that the visible convulsion is hysteroid in character we have still to ascertain whether it is primary or is consecutive to an epileptic seizure, and this is often most difficult. When the initial epilepti fit is severe, its occurrence can usually be ascertained, and tongue-biting alone may be taken as establishing its occurrence.* It is when the epilepti attack is of the minor form that the diagnostic difficulty arises, because the initial stage of many hysteroid fits has a pseudo-epileptic aspect. The mos important guide is the fact that most patients in whom the hysteroid attacks are post-epileptic, have at other times, or have had in the past, epileptic fits major or minor, without this sequel, and the character of these correspond to the commencement of the compound fit. In many cases the character of the initial stage include some feature, such as micturition, which is conclusively epileptic. In most patients who have had attacks at short interval during several years, the attacks are epileptic, and the hysteroid convulsion is secondary. The fact that the patient presents other symptoms of hysteria should be allowed no weight until all indications of epilepsy have been excluded, because, as already stated, it is only in those who are in some degree the subjects of hysteria that the combination occurs. It must not be forgotten, also, that separate hysterical and epileptic attacks sometimes occur in the same individual.

If the attacks are recognized to be epileptic in character, before we can refer them to idiopathic epilepsy, we have to ascertain that they are not due to reflex irritation, to toxæmia, or to organic brain disease. The irritation of dentition, intestinal worms, and, occasionally, indigestible food, are the most frequent causes of fits that may be confounded with those of epilepsy.

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* It is said that French hysterics bite their tongues during the attacks. In this country tongue-biting is practically confined to epilepsy.

In all cases in which the convulsions are of recent origin, these causes should be sought for and excluded, if necessary, by treatment. There is no other way of avoiding error, since the indications derived from the character of the attacks is an uncertain criterion. It must be remembered that fits may be at first of reflex origin, and may persist, when the peripheral irritation is removed, as idiopathic epilepsy. It is most unlikely that fits that have continued for more than a year are still due to reflex influences.

Convulsions that are due to blood states (alcohol, lead, uræmia) are rarely confounded with those of epilepsy, because they are merely part of a group of symptoms of obtrusive significance. The importance of an examination of the urine in all doubtful cases need not be insisted on. The recurring fits, which may occur in chronic Bright's disease without other signs of uræmia, and in some cases of lead poisoning, may resemble epilepsy very closely, and are to be distinguished only by the discovery of the underlying condition.

The problem of the diagnosis of epilepsy from the convulsions of organic brain disease presents many aspects, and is of great importance. Those convulsions which attend a sudden acute cerebral lesion are not likely to be mistaken for epilepsy, nor is the converse mistake probable, except during the transient "post-convulsive" weakness after a first unilateral fit. Chronic brain disease, however, may cause convulsions readily mistaken for those of epilepsy. Those convulsions usually begin locally, and may be partial in range. This character, while it suggests, does not prove, that they have this origin, since the convulsions of idiopathic epilepsy may commence in a similar manner, and moreover the convulsions of organic disease may not begin locally, but may be at once general. For the diagnosis a careful search must be made for other symptoms of organic disease, persistent headache, permanent hemiplegic weakness (not merely after an attack), paralysis of cranial nerves (especially diplopia), vomiting, and optic neuritis. The importance of an ophthalmoscopic examination in such cases cannot be too strongly insisted on. In any case of doubt, causal influences may be allowed weight. A history of syphilis, which so often causes cortical disease and convulsions, on the one hand, or a family history of epilepsy or insanity on the other, may rightly turn the diagnostic scale.

Convulsions may be due not only to active brain disease, but to the influence of an old cerebral lesion, an atrophied tumor, and especially a spot of old softening. In the former case the diagnosis rests on the history of the early symptoms. The latter are the cases of post-hemiplegic epilepsy already described. The distinction of these from ordinary epilepsy is easy, if distinct hemiplegia persists, but may be difficult if this has passed away. The difficulty arises only in the cases which date from childhood. In all such cases, if the fits are unilateral, careful search should be made for signs of weakness or of spasmodic over-action. Slight traces of the latter are significant. The circumstances of origin should also be ascertained. In infancy trifling hemiplegia is readily overlooked, and if the first convulsions were severe and unilateral, corresponding in seat to those which still occur, the case is probably post-hemiplegic, even though no hemiplegia was noted, an although an interval of years elapsed between the initial convulsions an their recurrence. Epileptoid fits may be an early symptom of genera paralysis of the insane, but the tremor of lips and tongue, the unequal pupils and the mental change render the diagnosis easy in most cases.

The simulation of an epileptic fit is on the whole rare, and the pretende fit never closely resembles a genuine seizure. In all cases the perfect reaction of the pupil to light throughout the fit will be conclusive evidence. To much weight must not be placed on the untypical character of the attack because true epileptic fits may deviate much from the usual type.

Prognosis.—The risk to life in epilepsy is not great. The mere violenc of the fit, appalling as may be its aspect, rarely causes death. The dangerous "status epilepticus" is too exceptional to constitute a measurable element in the prognosis. The greatest danger is in the cases in which ther is a tendency to turn on the face, or to vomit after a fit, but even this i slighter than that of the accidents to which the attacks expose the patient Many epileptics die by drowning; the fit not only occasions the fall into th water but prevents any effort to escape, and hence an epileptic has more tha once been drowned in a ditch.

The prospect of a spontaneous cessation of the fits is small; the tendenc of the disease is to self-perpetuation. Occasionally convulsions durin infancy cease at four or five years of age. Attacks which have continue till puberty rarely cease at that epoch. After twenty, spontaneous cessatio does sometimes occur, and I believe that it becomes more frequent as lif advances, but it is too rare to be reckoned on.

The chief question, therefore, is—what is the prospect that the diseas will be cured, or held in abeyance, by treatment? We cannot, at present separate the two points, and the only way known of curing the disease is t keep the fits away for a sufficient length of time to permit the morbid tendency to subside. Hence the question resolves itself into the probability o complete arrest. The prospect of arrest is slightly better in males tha females, better if the disease begins after twenty than before, and better th shorter the duration of the disease, being greatest in the cases in which i has existed for less than a year. It is rather easier to arrest the fits whe there is an hereditary tendency than when there is not—a curious fact whic was also pointed out by Herpin. The presence or absence of an excitin cause for the first fit does not influence the prognosis. A longer interva between the fits increases the prospect of arrest; this is extremely rare if fit occur daily. But this consideration is interfered with by the great difficult of getting patients whose fits occur at long intervals to persevere with treatment. The prognosis is better if the fits occur only during the sleeping o the waking state, than if they occur in both. It is better if there is no considerable mental change and if the attacks are all of the severe variety, tha if there are minor seizures, and better if the attacks are preceded by an aur than if they occur without warning. In cases of post-hemiplegic epileps the prognosis is much less favorable than in the idiopathic cases.

Treatment.—The treatment of epilepsy consists partly in the general management of the patient, and partly in the administration of drugs to influence the attacks—to arrest their occurrence, or, failing this, to render them less frequent and less severe. Unfortunately, the influence of all drugs is transient, and has to be repeatedly renewed. No means is known of suddenly curing the disease, of suddenly effecting such a change in the nerve centres that the attacks do not again occur, nor are there at present any facts which render it likely that such means will be discovered. The only method of producing such a change is by the continued administration of drugs for a long time, so as to prevent the occurrence of the nervous discharge and thus to produce such a change in the cells that the medicine may ultimately be discontinued without a recurrence of attacks. It is of great importance that this need for prolonged treatment should be made clear to all patients, and its reason to all those who can understand it.

Since the introduction of bromide salts for the treatment of epilepsy they have superseded other drugs to a large, and some think too great, extent. In the majority of cases their influence is incomparably greater than that of any other remedies, but in a minority of cases they fail, and in some of these other agents are more powerful. They only do permanent good by continued administration. The absence of a permanent effect from a short course of treatment is, however, equally conspicuous in the case of other remedies. Bromides are said to cause contraction of the small arteries of the brain, but it is exceedingly doubtful whether any part of their influence in epilepsy is due to this action. They lower reflex action in the spinal cord, and this effect shows that they have a direct action on the nerve cells. On any theory of epilepsy we must ascribe it ultimately to the disturbed action of nerve cells in some situation, and it is unnecessary to go beyond this influence of bromide on nerve cells to explain its action. If we regard the morbid state in epilepsy as an instability in the resistance of nerve cells, it seems probable that the effect of bromide is to increase the stability of that resistance.

The bromides of potassium, sodium and ammonium are extensively used, and bromide of lithium is occasionally employed; some authorities express a preference for one, some for another, some for a combination. My own experience has been that the influence of each on the disease is nearly the same, but that the bromides of sodium and lithium are rather less effective than the others, and that the bromide of potassium is a little better borne than the bromide of ammonium. It is probable that very little, if any, of the bromide salt is decomposed in the system, and that the alkali does not exert its own isolated action. The administration of free bromine has been suggested, but it is difficult to give it in sufficient quantity, and it must be transformed into bromide as soon as it enters the alkaline blood. This is true also of hydrobromic acid. No salt of bromine has much less tendency than another to produce acne, and this can always be prevented or rendered extremely slight by giving arsenic at the same time.

Bromide is usually given continuously in the smallest doses which will arrest the fits, or, failing this, in such doses as produce the most marked effect upon them. When the fits occur at a certain time one daily dose may be given two or three hours earlier. If the attacks occur at various times the bromide must be taken two or three times a day. The total daily quantity may vary between fifteen grains and two drachms, according to the age of the patient and the effect of the drug. Few patients are able to bear more than a drachm and a half a day without becoming what is termed "bromised," lethargic and dull, physically and mentally, with cold extremities, and a feeble pulse; the best results are usually obtained with not more than a drachm a day. If this does not arrest the attacks, larger doses rarely succeed, and combinations of bromide with other drugs are more useful. The effect of bromide is sometimes immediate: after the first dose the attacks may cease; often, however, its influence is gradually produced. When the attacks have ceased under its administration, they too often relapse if it is discontinued, and then are less easily arrested than at first. Relapse usually occurs within a few weeks (sometimes within a few days) of a too early cessation of treatment; after a year of freedom without treatment it is probable (though not certain) that the disease will not recur. As a rule, bromide should be continued, without any diminution of the dose, for two years after the last fit. It should not then be suddenly discontinued, but the daily dose should be gradually lessened through another year. The continued use of bromide in moderate doses has no necessary influence on the general health or intellectual energy. At first, however, some cerebral depression may often follow the arrest of fits (see p. 1094), and this is often erroneously regarded as the effect of the bromide alone. If moderate in degree it is better to combat it by tonics than to reduce the bromide. Occasionally the depression becomes so alarming that it is necessary to stop or lessen the medicine and allow a fit to occur, and to go on with smaller doses.

In order to facilitate the occurrence of the change in the nutrition of the nerve cells which we must assume to underlie the cure of epilepsy by bromide, I have found it well, in cases in which a moderate dose stops the fits to administer a series of large doses at increasing intervals, beginning with two drachms every second morning, and increasing to three drachms every third morning, and four every fourth;* the dose and interval are then reduced in the reverse order, so as to spread the course over about six weeks The dose should be given after breakfast, in about half a pint of water After such a course, if the bromide is discontinued, patients remain free from fits much longer than after bromide has been given for the same time in ordinary doses, showing that more effect on the nerve elements has been produced. But a permanent result is seldom obtained from such a course alone; it is still necessary to continue small doses for a year or more. believe, however, that the prospect of cure is increased by this treatment.

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* I have many times gone up to ounce doses every five days (more is usually vomited) but slight mental derangement, lasting for a few weeks, is apt to be set up. The chief immediate effect of a dose of four to eight drachms is headache.

When bromide alone fails, it may succeed when combined with certain other drugs, most of which have by themselves some influence on the disease. One of these is digitalis, a popular remedy for epilepsy in the west of England two centuries ago. The combination is useful, as might be expected, when there is cardiac dilatation and valvular disease, but it is also specially useful in nocturnal epilepsy, and in some other cases. Digitalis probably has an action on the central nervous system, as well as on that of the heart and vessels, although some part of its influence may be due to the regulation of the blood supply. Five or seven minims of the tincture may be given with each dose of bromide. Another combination of value is that with belladonna, which is also an old remedy, and, although very rarely successful alone, it is a useful adjuvant, in doses of five or ten minims of the tincture. Atropine may be given instead, one or two drops of the B. P. solution.

Cannabis indica is occasionally beneficial, both alone and in combination with bromide; the combination is most useful in cases with a good deal of persistent headache. Opium and its alkaloid morphia are of little service in epilepsy. The hypodermic injection of morphia in full doses is attended with considerable danger. If an attack occurs after the injection has been given, and the post-epileptic coma coincides with the narcotism, the patient's life may be in great danger, and I have known death to occur, apparently from this cause.

Zinc has long been held in repute, and with some reason. It is far inferior to bromide in most cases, but now and then succeeds when bromide fails. The lactate of zinc, introduced by Herpin, is the most convenient form; it is the most soluble of the less irritant salts of zinc, and if given after meals, it can generally be increased to eight or ten or even fifteen grains twice or three times a day, without producing nausea. The oxide of zinc may be given, if the lactate is not accessible, but the limits of toleration are sooner reached. The citrate answers almost as well as the lactate. The bromide of zinc is far inferior in utility. The lactate may be combined with belladonna or with bromide, and the combination of the three sometimes succeeds when each alone fails.

Iron, in the opinion of some distinguished authorities, should not be given to epileptics, because it is thought to increase the frequency and severity of the fits. This opinion, as a general principle, is certainly erroneous. I have given iron to several hundred epileptics, and instances of apparent aggravation of attacks are extremely rare. In most cases it may be given without any ill effect on the disease, and in some its use is distinctly beneficial. I have known attacks to cease entirely when iron was added to bromide, and, in rare cases, when iron was substituted for bromide. Care must, of course, be taken not to ascribe to the iron any effect of the discontinuance of the bromide. Iron seems to have a direct action on the nerve centres, analogous to that of zinc.

In some cases of inveterate epilepsy, in which bromide had no influence, I have found borax distinctly useful. From fifteen to thirty grains may be given after food three times a day, and it may be continued for years without any ill effect beyond a possible eruption of psoriasis, amenable to arsenic. A little gastro-enteric disturbance may occur at the commencement of administration, but quickly ceases if the dose is lessened. Of course, the influence of borax is not comparable to that of bromide in cases in which this is effective.

Nitroglycerine has been lately praised as a remedy for the minor attacks (Hammond). It may be given in doses of $\frac{1}{150}$ of a grain to begin with, increased gradually to $\frac{1}{25}$. In most cases it fails, but in a few it has a distinct influence. In one case, with rather severe fits, these became fewer as the dose was increased, and finally ceased when the patient was taking $\frac{1}{80}$ grain, and did not again recur. Among other drugs which I have tried without seeing benefit, other than slight and rare, from their use, are aconite, hydrocyanic acid, bromide of camphor, nitrite of soda, nitrite of amyl (by the mouth), chloral hydrate, paraldehyde, benzoate of soda, Piscidia erythrina, Calabar bean, ergot, sclerotic acid, codeia, and cocculus indicus. It is singular that the latter, injected beneath the skin, will infallibly produce a fit in an epileptic patient. From nitrate of silver I have seen little benefit, and I have had among my patients several who were discolored by it, in the prebromidic days, without any alteration in the severity of the attacks.

The treatment of minor attacks is on the whole the same as of the severer seizures. They are often arrested by bromide, but it is far more common for bromide to have no influence in the case of *petit mal* than in convulsive attacks. The latter may be arrested and the former may continue or even become more frequent. The other drugs above mentioned are sometimes effective when bromide fails, especially the salts of zinc, belladonna and Indian hemp.

Arrest of Attacks.—The means by which commencing fits may be arrested have been already mentioned. In attacks commencing in one extremity, the ligature is often successful. The most convenient method of applying it is for the patient to double a piece of tape, and pass it round the arm above the elbow, with the ends through the loop formed by the doubled part, and brought down to the lower part of the sleeve so as to be accessible, and easily pulled tight, as soon as the warning is felt. Now and then the repeated arrest of fits produces a permanent effect; in one patient, for instance, the aura ultimately stopped spontaneously at the place where it had been many times arrested by the ligature. In cases of this kind it has been proposed to produce a more permanent effect by a blister around the limb.* It is occasionally successful. But the arrest of the fit, by the ligature or a blister, sometimes causes so much giddiness and distress, that patients may consider the remedy worse than the disease.

Of methods of arresting fits which begin in other ways, inhalation of nitrite of amyl is the most frequently (though not invariably) successful; it doubtless acts by flooding the brain with arterial blood, a potent agent for modifying the action of the nerve elements.

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* An old method of treatment, revived by Brown-Séquard and Buzzard.

During an attack little treatment is necessary. In patients who bite the tongue, a cork or, better, a small piece of india rubber, placed between the teeth may prevent this accident. The patient should be laid down, for obvious reasons; it is doubtful, however, whether posture influences the duration or severity of the attacks. It is necessary to see that the clothes are loose about the neck; if they are tight when the neck becomes turgid and swollen, the resistance to the return of blood is increased, and the extravasations into the skin and conjunctivæ are more probable. After the attack the patient should be allowed to sleep, if inclined to do so, for at least half an hour.

In the *status epilepticus* bromide often fails. The inhalation of chloroform usually effects only a transient amelioration. Nitrite of amyl has been recommended by Crichton-Browne. In the cases I have seen, most influence has been exerted by chloral (gr. xv every three or four hours), subcutaneous injections of morphia (gr. $\frac{1}{10}$), and the application of ice to the spine.

General Management.—It has been recommended, on theoretical grounds, that the diet of epileptics should contain little or no animal food. The evidence of experience, as far as I have seen, is opposed to this opinion. I have known the exclusion of meat from diet to cause a great increase in the severity of the fits, which became slighter when meat was again given; and I believe that patients do best if a moderate quantity of animal food is given twice a day, care being taken to avoid that which is indigestible. I have known one case, however, in which the patient could never take beef without bringing on an attack, although he could take other kinds of meat with impunity. Stimulants should be taken sparingly; young persons can do better without alcohol. In all cases it is most important that the regular action of the bowels should be secured.

Moderate exercise of body and mind does good, but severe and exhausting exertion is undesirable, and the excitement of competitive and other examinations should be avoided. The education of children should not be entirely neglected. It must be remembered that there are many positions in life for which epilepsy constitutes no insuperable disqualification and for which they may be trained should the disease not be arrested. In this choice of an occupation, so much depends upon personal opportunities that it is difficult to lay down general rules. But there is one consideration to which all others must be subordinate—the calling must be one which involves no risk of life from the occurrence of an attack while the patient is at work. An outdoor life is better than a sedentary occupation, but the choice of the latter is so much larger that in most cases a sedentary calling has to be selected.

The question of marriage presents itself under two aspects, as regards the individual and as regards the possible offspring. Marriage has no influence on the disease, beneficial or the reverse, except so far as it may involve deleterious sexual excess. But with reference to the offspring the question is very important. There is no certainty that the taint will be transmitted; on the contrary, as regards any individual child there is a probability that it will escape. But the probability is also against the escape of all the offspring from diseases of the nervous system, especially if the disease in the parents is inherited.

Surgical Treatment.—Counter-irritation in the neck or scalp has been often employed, the usual method being by a seton in the neck. That it occasionally does good is undeniable, and the same is true of an extensive accidental burn. In most cases the effect is temporary only. Trephining, an old remedy, has been lately brought into fresh prominence. The mere formation of a hole in the skull, without interference with the brain, has been employed in many cases of idiopathic epilepsy, but the results obtained are little better than those of a seton in the neck, and it is doubtful whether the operation has other influence than that of an energetic counter-irritation. But the local commencement of the fit in one limb means local excessive instability in the corresponding cortical centre, and in some cases it means also old organic disease. (When there is active disease there is probably a tumor, and such cases have been already considered.) The question comes, Is the removal of such disease justifiable? The answer must depend on the character of the fits. If many of these are local and partial, and there is reason to believe that there is disease that can be completely removed, an operation is justifiable. If, on the other hand, the fits generally spread through the whole of one side, and, still more, if they often spread to the other side, the probability of benefit is too small to justify the very considerable risk of the operation. Apparently the repeated discharges lead to so wide a deficiency in the stability of the nerve cells that the discharge starts from other regions if the primary lesion is removed. Hence the operation should never be thought of in a case of idiopathic epilepsy in which the fits begin locally, because it is certain that the discharging tendency is widespread and almost certainly adequate, in other parts, to cause fits.

Ligature of the carotid artery has been performed without result. Ligature of one or both vertebrals has been practiced by Dr. Alexander, o Liverpool, as a cure for epilepsy, but the results obtained are not commensurate with the risk.

CONVULSIONS: ECLAMPSIA.

Convulsions, resembling more or less closely those of epilepsy, may occur from various causes. In epilepsy, however the disease was originally excited, the recurring convulsions are the result solely of the tendency o the brain to "discharge," and no causal influence outside the brain can be discovered. When such fits are due to some other cause, they are called simply "convulsions." But this term has also a wider application; it is applied to the fits of epilepsy and also to those that are produced by organic brain disease. Hence the word "eclampsia" has come to be used as a name for the condition in which convulsions occur from other causes than primary states of the brain. In epilepsy the convulsions themselves are the sole evidence of their cause; in eclampsia, the cause of the fits manifests itself by other symptoms, often by symptoms outside the nervous system.

The term "eclampsia" is, however, chiefly used as a designation for convulsions that recur. It is not applied to the single fit that a child may have at the onset of an acute fever, or in consequence of an indigestible meal. Moreover, the convulsive attacks that form part of the manifestations of hysteria are also excluded from the meaning of the term. There are three special varieties of eclampsia—infantile, puerperal, and uræmic.

INFANTILE CONVULSIONS: INFANTILE ECLAMPSIA.

Convulsions occur in young children with great readiness, and have many causes. The special liability of infants is probably due to the condition of development of the nervous system. At the time of birth, only parts of it are structually complete. Extensive tracts of fibres have not yet acquired their white medullary substance, and until the axis cylinders are thus clothed, the fibres have but little conducting power, although it is probable that such power is not altogether absent. But the lower centres are further advanced than the higher ones, and are, in consequence, imperfectly controlled. This is probably the chief reason why reflex disturbance so readily occurs in early childhood. By far the most potent cause of convulsions in children is the constitutional condition termed "rickets." The essential element in rickets is defective development; the perversion of development that occurs (*e. g.* in the bones) is secondary to, and consequent on, its defect. At the time at which this constitutional state chiefly occurs, the structural development of the nervous system is complete. But it is probable that functional capacity is only fully developed after structural perfection, and the parts last developed may suffer from the general delay in development more than those parts that have been longer perfect and longer in full use. Whether this is the explanation or not, it is certain that, in rickets, there is an excessive activity of the centres of the brain and cord on which reflex spasm and convulsion depend. It is probable that the morbid tendency is exalted by an inherited neurotic disposition.

The period at which rickets chiefly occurs is between the sixth and eighteenth month of life. This corresponds with the most active epoch of dentition. Dentition is delayed, with other developmental processes, and hence the fits have commonly been ascribed to dentition, and have been called "teething fits." It is probable that, in some cases, the process of dentition has an influence; it involves irritation of the sensory nerves, and so may excite the convulsion. But it is certain that this exciting influence is only a small part of the process of causation, and that it was formerly over-estimated, even after the relation of the fits to rickets had been conclusively proved by Sir William Jenner. The convulsions may occur without any discoverable exciting cause, or they may be produced by various peripheral impressions besides those of the teeth, both in children who are rickety and in those who possess only the predisposition inherent in infancy.

Of these causes the most frequent is some irritation of the gastro-intestinal mucous membrane, especially by worms or by some substance in food that is absolutely indigestible, such as the skins of dried fruit, currants, etc. Among worms, lumbrici are chiefly influential; tapeworms are rare in young children, and threadworms scarcely ever cause convulsions, although they are often thought to do so. The influence of irritation of the alimentary membrane in causing convulsions is remarkable; the effect often occurs with very little conscious sensation, while contractions of the intestines that cause acute pain have no corresponding influence.

Convulsions occur in infants in states of general exhaustion, however produced, especially in that caused by diarrhœa. Thus they may form part of the condition called "hydrocephaloid" (p. 761). The predisposition in these cases probably depends on the acute impairment of nutrition of the nerve elements, perhaps, also, on the deficient blood pressure, of which the depressed fontanelle is evidence. On the other hand, they result also from mechanical congestion of the brain, which involves, among other conditions, a deficiency in the supply of arterial blood. Hence paroxysmal cough, of whatever nature, but especially that of pertussis, may lead to a general convulsion. Possibly, in whooping-cough, convulsions may be in part due to the spread in the predisposed brain of the "discharge" causing the cough. It is doubtful whether convulsions ever result from active congestion of the brain, which was once thought to be their common cause. If the condition ever has this effect, it is only in excessively rare cases, or when the congestion is the first stage of inflammation. During the first few days of life convulsions are not uncommon, but they probably always depend on direct injury to the brain during the process of birth (see p. 801).

Symptoms.—Infantile convulsions are often preceded by other signs of irritation of the nervous system. The child is restless and irritable; the aspect of the face often changes; there may be a little twitching of the mouth at times, or some abnormal movement of the eyes. In rickets, there is often inversion of the thumbs and great toes—"carpopedal contractions"—or there have been attacks of "laryngismus stridulus," or more distinct partial convulsions. The actual attack usually comes on suddenly, and may occur during either the walking or the sleeping state. Sometimes a severe attack resembles closely an epileptic fit, but in general the attacks are slighter than typical epileptic convulsions. There is a sudden fixation of the eyes, which are often rolled up or down, or to one side, or may converge or diverge. The face and lips become pale. The limbs and trunk are rigid and stiff, the head turned to one side or retracted. Respiration is interfered with, and the face becomes dusky. After a minute or so, the spasm may relax, or may become clonic as in an epileptic fit. Sometimes the spasm is entirely clonic, but it is then slight, and involves chiefly the face and eyes, or the hands and feet. In other cases, again, a slighter tonic spasm may continue for a considerable time, half an hour or more; it is then insufficient to stop breathing but may interfere with this enough to cause a slight change in the color of the face. In these cases the spasm may not change the position of the limbs, and may not be noticed until the alteration in the color of the face attracts attention, when the rigidity is discovered. Such persistent spasm, however, usually varies from time to time, so that there is really a series of convulsions with imperfect recovery in the intervals. Many slight attacks are accompanied by a scream, and are popularly called "screaming fits;" there may be nothing more than a scream and quick breathing or grinding of the teeth, and brief rigidity of the limbs. Such convulsions are probably always bilateral; it is doubtful whether the fits in true infantile eclampsia are ever confined to one side.

The attacks of laryngeal spasm termed "laryngismus stridulus" are really slight local convulsions and occur under the same conditions as the general convulsions. They consist of sudden spasm stopping the breath and causing the child to seem on the point of death from suffocation. Then the spasm relaxes, and with a loud crowing inspiration the child gets its breath again. Such attacks often occur many times daily, spontaneously, or excited by some sudden alarm, or by some peripheral impression.

The convulsions of rickets may be few, and cease after a few days, or they may recur during several weeks, or even months. Not uncommonly they begin at eight or nine months, and go on through the second year of life, and then cease. They may, however, continue for a still longer time, and the condition must then be regarded as epilepsy. Indeed, whenever attacks continue after their cause has ceased, the condition is inseparable from epilepsy. Some of the children in whom the fits cease, after lasting for a year or so, become epileptic in later childhood or at puberty.

Diagnosis.—The chief point in the diagnosis of the nature of infantile eclampsia is to distinguish from it the convulsions that are due to organic brain disease. Those produced by such processes as tumor or meningitis are soon accompanied by other symptoms of the morbid process. It is important, however, to bear in mind the fact that the commencing morbid process may increase the irritability of the brain before it causes other symptoms, and then some peripheral impression may excite a convulsion. It must be remembered that a fit, even in a young child, generally means some increased central excitability, and the older the child the greater is its significance in this respect. If there is not evidence of a cause of increased excitability (such as prostration or rickets), the possibility that there may be commencing organic disease should always be remembered.

A special difficulty is presented by the cases in which convulsions are due to a sudden cortical lesion and recur (see p. 1095). The distinction of these fits from those of eclampsia rests especially on the fact that they are generally unilateral, and often commence locally in some part of one side. When they are accompanied by distinct evidence of hemiplegia, corresponding in side to the convulsions, the diagnosis is easy. Often, however, the paralysis is slight and unnoticed. Whenever fits are one-sided, and commence with a sudden series of severe convulsions, accompanied by the signs of a cerebral illness, it is exceedingly probable that they are of this nature. If recurring eclamptic convulsions are ever unilateral, they are not constant in seat, but affect sometimes one side, at other times the other side.

In all cases of infantile convulsions a careful search should be made for any peripheral cause of irritation, such as local suppuration and the like.

PROGNOSIS.—There is more danger to life in the eclamptic convulsions of infancy than in the epileptic fits of later life. Frequent convulsions may readily cause fatal exhaustion in a young and feeble child. The actual prognosis must be founded on the severity and frequency of the fits, and on the strength of the patient. If convulsions continue for some months, the prognostic question arises—Will the disease go on to epilepsy? The danger is considerable, and it increases the longer the fits continue. But even after they have lasted for a year, there is a fair chance of arrest.

TREATMENT.—In the treatment of the convulsions of children, the first important measure is to search for their cause, and, if possible, to remove it by treatment. This is especially important in the case of rickets, in which the mere treatment of the attacks may have little influence if their cause is allowed to continue unchecked, whereas the treatment of the underlying diathetic state may alone speedily stop the fits. The attacks themselves, whether general convulsions or the local laryngeal spasm, are generally amenable to bromide of potassium, but it is necessary to give this in adequate doses, three grains to a child under six months; five grains to one between six and sixteen months, and still larger doses to older children. Two or three doses may be given in the day. Severe attacks may need the inhalation of chloroform, which is almost always effective, but may have to be repeated two or three times before the bromide that is given can assert its influence.

It was formerly the custom to treat infantile eclampsia by warm baths and by applications designed to draw the blood to the skin, on the theory that the convulsions were the result of cerebral congestion. Gray powder was given, in frequent doses, to combat the supposed local condition. Indeed, the fact of a patient having suffered from infantile convulsions may still often be correctly surmised from the indications presented by the permanent teeth of the influence of the mercury in early life. The theory was certainly erroneous, and it is doubtful whether the treatment did any good. I remember, when a pupil in the country, spending the greater part of a day watching in vain for the expected effect of repeated warm baths on the almost continuous tonic convulsion from which a rickety child was suffering. Certainly, unless a warm bath has an immediate effect, it is useless to repeat it.

The dentition theory of the origin of the convulsions led to the almost universal adoption of the practice of lancing the gum over a coming tooth to lessen the irritation. As the sole or even the chief element in the treatment the measure is a mistake, since it deals with the least important, and often quite unimportant, element in the causation of the convulsions. It is, however, probable that the measure does sometimes facilitate the eruption of the tooth, and may thus lessen any irritation that exists.

PUERPERAL CONVULSIONS: PUERPERAL ECLAMPSIA.

Etiology.—Toward the end of pregnancy, during labor, and after labor, women sometimes suffer from severe convulsions, and of those thus attacked, many die. Women who are pregnant for the first time, and those who are comparatively young, are the most prone to suffer. A few of the patients have been epileptic, and the fits are simply those to which they have long been liable. In most cases of epilepsy, however, the attacks do not occur during the puerperal period. In some other cases, very rare, there has been evidence of no other causal condition than an over-excitable state of the nervous system, and the convulsions were distinctly excited by the pains of labor, or by the irritation of digital examinations. In the vast majority of cases, however, puerperal convulsions are associated with the presence of a large quantity of albumen in the urine, in which casts also are found, and in fatal cases the kidneys present indications of acute nephritis. These signs of kidney disease are usually accompanied by distinct œdema of the subcutaneous tissue. The association of puerperal convulsions with such conclusive proof of Bright's disease, is too uniform to be without very strong significance, and the balance of evidence is most strongly in favor of the common inference that has been drawn from the association—that puerperal convulsions are really uræmic convulsions. They differ from such convulsions under other circumstances in the common absence of pronounced uræmic coma, but they differ, also, in another fact—which explains to some extent the absence of coma—the presence of a powerful cause of reflex irritation, which, as we have just seen, may excite convulsions apart from any toxæmic influence, and when no other predisposition exists than an undue central excitability of the nervous system. We must remember, also, that the condition of pregnancy itself involves an abnormal state of the nerve centres, as is clearly shown by the occurrence of many functional disorders such as chorea and tetany. Thus the influence of uræmia is exerted on predisposed centres, and is aided by peripheral irritation which is, under the circumstances, peculiarly powerful. Moreover, even when other signs of uræmia are absent, albuminuric retinitis often testifies to the intensity of the influence on the system exerted by the renal disease.

The evidence is so conclusive that it seems hardly worth while to mention the objections that have been raised to the uræmic theory or the hypotheses that have been put forward to take its place. The one and the other are alike feeble. In addition to the absence of other signs of uræmia, which has been already discussed, it has been alleged that albumen is to be found in so large a proportion of pregnant and puerperal women that its coincidence with convulsions has no significance. But it is a question not of the mere presence of albumen but of its amount, and the amount that is common, apart from convulsions, is trifling and without significance.* On the other hand, clinical experience shows that whenever a woman, at or near labor, presents much albumen in the urine, with other signs of nephritis, she is in imminent danger of convulsions. The chief other theory that has been put forward is, that there is anæmia of the brain, the result of a watery state of the blood and of vasomotor spasm, which is itself due to the influence of irritation of the uterine nerves or of the nerves of the sacral plexus. To render this theory tenable it has yet to be proved, first, that there is more vasomotor spasm than frequently exists in various conditions without convulsions, and, secondly, that vasomotor spasm ever yet gave rise to a fit.*

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* This point has been ably and conclusively discussed by Galabin, *Brit. Med. Journ.*, Aug. 30th, 1880.

SYMPTOMS.—Puerperal convulsions occur during, before, or after labor. They may not only be distinctly excited by the local irritation, but are sometimes set up by other causes, as a blow on the head (Engstrom), while the depression of anxiety and fear seems sometimes to aid in their production. In addition to the œdema already mentioned their onset is often heralded by severe headache, præcordial pain, or sudden amaurosis, the latter certainly, and the others probably, uræmic in origin. The onset is usually sudden and the convulsions generally resemble closely those of epilepsy. There is absolute unconsciousness, the tongue is frequently bitten, and the bladder and rectum may be emptied during the fit. One difference from ordinary epileptic fits is, that very frequently the affection of the two sides is not simultaneous but successive. The eyes are often conspicuously involved. In one case in which the character of the fits was very carefully noted † each began with nystagmus, lateral and to the left. Then first the eyes and afterward the head were strongly turned to the left, and clonic spasm came on in both eyelids, with tonic contraction of the left side of the face, and the left arm and leg. First the wrist was extended, then strongly flexed and pronated. Subsequently the eyes and head turned to the right, and the right arm and leg were rigid like the left, so that tonic spasm became universal and the tongue was thrust out of the mouth; then general clonic spasm set in with horizontal nystagmic movement of the eyes. The spasm ceased on the left side sooner than on the right. During the coma that followed, the eyes were rolled upward. All the fits were the same, save that in the earlier attacks there was opisthotonos, and the later fits, as the patient became exhausted, were limited to the face, involving especially the frontales. The patient was a young married woman, aged eighteen, who had an intense dread of parturition. The fits commenced soon after labor set in, and each pain excited a convulsion. She was delivered with the forceps, but the fits continued, with an interval after labor of four hours, and she died forty-eight hours after the onset. The temperature rose to 103° and continued at that point. The urine, when heated, became solid from albumen, and the kidneys after death presented the characteristic signs, naked-eye and microscopic, of acute inflammation. In some cases, in which the convulsions are very severe, the temperature rises to 108° or 109°, as in the *status epilepticus*. The mortality in puerperal convulsions is about 30 per cent.*

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* Some of the framers of other theories have urged, as an objection to the uræmic explanation, that the temperature is often raised after puerperal fits. But it should be generally known that true uræmic convulsions also often raise the temperature, and the objection is absolutely without weight.

† By Mr. Hyde Marriott, of University College Hospital, to whom I am indebted for the report of the case.

TREATMENT.—The treatment is in part obstetrical, and this need not be here discussed. Venesection was formerly employed, and when much blood had flowed, the fits usually ceased, but the consequent exhaustion of the patient, and the discovery of the coincident kidney disease, has led to the abandonment of this practice, especially in view of the fact that inhalations of chloroform are usually successful in arresting each convulsion. As long as the inhalation is kept up, the convulsions may cease, but when it is discontinued they too often return. The treatment of the blood state is necessarily limited under the circumstances of parturition, and yet it is unquestionably more important than any other. Vapor baths have been employed, and so has pilocarpine, sometimes with success. Theoretically, venesection and transfusion might be expected to afford more chance of recovery to a desperate case than any other measure. For further details of treatment, the reader is referred to works on obstetric medicine.

URÆMIC CONVULSIONS.

The nature and precise cause of the convulsions of uræmia are topics beyond the province of this work, but it may be convenient briefly to describe their characters. They occur only when the kidney disease has profoundly affected the system and has altered the state of the blood; in chronic kidney disease there are usually other indications of this effect in hypertrophy of the heart and albuminuric retinitis.

Uræmic convulsions usually set in suddenly, and there may be no preceding symptoms to indicate what is coming. In some cases, however, they are preceded by other symptoms of uræmia, as coma or amaurosis. The fits are seldom isolated; usually several occur in a short time, sometimes in the course of an hour, sometimes at intervals of several hours.

The convulsion usually resembles very closely an epileptic fit, consisting of tonic and clonic spasm. The preponderance of the spasm on one side is generally well marked, and causes a strong deviation of the hand and eyes to one side. Occasionally the convulsion is unilateral, but the affection of one side is not constant—the side involved varies in different fits. Another occasional characteristic is a special affection of the muscles of the face, which may be bilateral, as was conspicuous in the case of puerperal convulsions described above. Nystagmus, as in that case, is sometimes observed, the quick movement being to the side toward which the head deviates. The convulsions often begin locally, now in one part, now in another, but quickly spread. The pupils are usually dilated during the fit, and do not act to light, but I have known them to be contracted during the fit, and to dilate afterward. In severe convulsions, the interference with respiration is very marked, and the tongue may be bitten. Immediately after the fit, the knee jerk may not be obtained. The patient may sleep for a time after the attack as after an epileptic fit, or may pass into a state of coma, or may be merely drowsy.

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* Of 62 cases in *Königsberg Hospital Records*, the percentage mortality of mothers was 29, and of children 38 per cent. (Gettkant, *Diss. Königsb., 1884*).

The treatment of uræmic convulsions is that of the blood state.

VERTIGO.

The term "vertigo" means, by derivation, a "turning," and is used as designation for any movement or sense of movement, either in the individua himself or in external objects, that involves a defect, real or seeming, in th equilibrium of the body. The word "giddiness" is used as a popular synonym for vertigo. An exact and complete appreciation of the relation of th body to its physical surroundings is involved in the mental state that we cal "consciousness." If that appreciation is incorrect in any degree—and it i rendered incorrect by a false sense of movement—to that degree the sense o perfect consciousness is interfered with. Hence vertigo always involves slight interference with consciousness. This fact influences the popular us of the term "giddiness," which is sometimes used to designate a menta state of imperfect consciousness when there is no sense of movement, an nothing that would be termed "vertigo" in the strict medical use of th word. The word "dizziness" is more often used in the sense of imperfec consciousness, but it is also sometimes used as a loose synonym for "giddiness."*

Vertigo is a common symptom of organic brain disease, but in such case it is associated with other distinct symptoms of its cause, and has bee already considered (p. 530). In all cases, indeed, it is merely a sympto of some definite morbid state, but in many conditions it is a symptom th is far more obtrusive than its cause. Hence it has become customary t describe vertigo as if it were a definite disease, and the method has considerable practical convenience, although it is not logically correct.

Before describing the special forms presented by vertigo, it is desirable t consider what is known of the symptom and its origin. The subject involve a very obscure branch of the pathological physiology of the nervous system Vertigo is essentially a sense of defective equilibrium, and it is necessary t consider, first, what we know of the way in which equilibrium is maintaine under normal circumstances.

The maintenance of equilibrium is effected, except when the body is rest, by the activity of the muscles, influenced by the brain. When th body is at rest, the equilibrium is permitted by the absence of muscular contraction. The action of the brain is in some way determined by certa centripetal impressions, which give information to the cerebral centres as to the relation of the body to external objects, as to the manner in which the body is supported, and as to its position. (1) Sensory impressions come from the skin, etc., of the parts on which the body is resting. (2) Centripetal impressions reach the brain from those muscles, the contraction of which determines the posture of the body; the most important of these are from the muscles of the legs and lower part of the trunk. (3) Information as to the position of the eyes and head shows the relation of seen objects to the body; this information, in the case of the eyes, is apparently derived from the sense of the active innervation of the muscles. (The evidence of this will be found at p. 597.) Whether this is also aided by centripetal impressions from the muscles, we do not know, nor do we know what impressions inform the centres as to the position of the head. The innervation of the eye muscles seems to be the more important of the two. It is probable that the innervation of other muscles also exerts an influence, but we cannot distinctly trace this. (4) The impressions from the semicircular canals of the internal ear. These are apparently due to the pressure of the endolymph under the influence of gravitation and of movement (Flourens, Goltz, Cyon,* etc.). We shall have to consider these in greater detail presently.

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* The original meaning of "giddy" was mirthful (a sense still extant), and that of "dizzy was foolish or dull.

Of these various impressions which are doubtless always acting on the intracranial centres, only those of the first class influence consciousness; the impressions from the cutaneous and other nerves, due to pressure, are felt as sensations; of the others we are almost entirely unaware, although we are conscious, indirectly, of the effects of their derangement. They apparently act on some centre through which equilibrium is maintained, and which in some way regulates the outgoing currents of nerve force. The whole of this process goes on, under normal circumstances, independently of consciousness. Many facts suggest that the middle lobe of the cerebellum takes an important part in the process. We can trace a connection between it and many of the afferent paths concerned. It may merely coördinate these impressions, and influence, according to them, the cerebral centres whence the outgoing currents proceed. This conception harmonizes with the various facts better than the assumption that the outgoing impulses proceed directly from the cerebellar centre.

If any one of the centripetal impressions that regulate equilibrial coördination is imperfect, we are chiefly conscious of the effect on the action of the centre, not of the imperfection itself. We feel the effect as vertigo. That vertigo is commonly a disturbance of the action of some centre, and is not a mere consciousness of the defective centripetal impression, is shown by the fact that, when well marked, it is a sensation of a motor process.† There may be merely a sense of movement, but if the sensation is intense, there is actual movement, and this is almost always in the same direction as the sensation of movement. From this it appears that the sensation felt is the result of the process which, in greater degree, causes the movement, *i. e.*, it is due to a motor process. Even when the apparent movement is in external objects, the same correspondence is often observed; if the patient also moves, his movement may be in the same direction as that in which objects appear to move. This is intelligible only on the assumption that the sensation is due to a motor process. If a person actually moves, and an object remains opposite his face (in the centre of his field of vision), he rightly infers that it has moved. The subjective process in vertigo leads to the same inference. Sometimes, however, objects appear to move in the opposite direction to that of the sense of subjective movement; to this we shall presently return.

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* The various experiments on the subject are described in most text-books of physiology. Their details, however interesting, have not such a direct bearing on the medical aspect of vertigo to make it worth while to describe them here.

† This fact has been repeatedly insisted on, as of paramount importance, by Dr. Hughlings

These statements apply to what may be termed typical vertigo of sensory origin. But vertigo may be due to other causes. We have seen that it is common in epilepsy as the warning of severe, and the sensation in slight attacks. We can understand its occurrence in these cases on the theory just described. In epilepsy there is a spontaneous motor discharge, which may be one-sided, or merely greater on one side than on the other; in either case there will be a tendency to lateral movement, which is actually seen in the deviation of the head and eyes, and may give rise to positive rotation. This motor process may be felt as vertigo before it causes movement, or when it is too slight to cause movement. Hence, too, we can understand that many organic lesions of the brain should cause vertigo at the time of their occurrence, just as they may also cause convulsion.

But the sensation is not always thus definite, and does not always correspond to the motor process. It may be merely a vague sense of unsteadiness or uncertainty. It is possible that in some cases it may be a more direct effect of the deranged and discordant centripetal processes, or the vagueness of the sensation may be due to the trifling character of the motor disturbance, or to the circumstance that this is complex.

The mechanism of vertigo is thus so complex, and our knowledge of it is so fragmentary, that we may easily go very far wrong if we endeavor to formulate the precise mode in which any given sensation is produced, or the actual disturbance that exists in any given case. But at the same time we may note the apparent suggestiveness of some of the phenomena, in considering their features. The exact character of the sensation experienced is often very difficult to ascertain; and this is not surprising, for the moment

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Jackson. It was also pointed out, long ago, by Dr. Russell Reynolds: "The feeling o equilibrium results from the harmony of our different sensations among themselves, and witl the motor impulse which is their combined effect. When any one group of the sensoria impressions is distorted or removed, the balance is disturbed, and as these impressions ar themselves the stimuli of muscular action, attempts are made for its restoration . . . . producing vertiginous or allied movements."—Russell Reynolds, "Vertigo," London, Churchill 1854—a paper in which are collected all the older facts and opinions on the subject.

is one of intense mental confusion, in which accurate observation is most difficult. But the common uniformity of the sensation in the same case at different times, compensates, in some degree, for its disturbing character, and enables the patient, after a time, to give a description that is trustworthy, as far as it goes.

The apparent movement of the individual himself, the subjective vertigo, as we have seen, varies much in different cases. When it is definite in character, its direction is often emphasized by the fact that the patient falls or tends to fall in the same direction. There may be a tendency to incline to the right or left, or to turn toward right or left, but more often this is combined with a movement forward or backward, and often the latter is all that the patient is able to realize. More common, however, than any other single movement, is a sense of going downward, of sinking. One patient described it as if there were a powerful magnet in the earth, drawing him toward its centre; another, as if there were a huge weight on his head pressing him downward. A sense of falling from a height is also common, and is probably familiar to most persons as a sleep sensation, the cause of which will be considered presently. It is noteworthy that the sense of going downward toward the earth may have the same character, whether the patient is standing or lying. This sensation is occasionally felt when there is disease of the semicircular canals, and it therefore appears as if the precise direction of the false sensation were determined by the true sensations engendered by the posture of the body. The patient who feels as if he were sinking into the earth when he is standing, feels as if he were sinking through the bed when he is lying. The opposite sensation, that of rising, is extremely rare.

The apparent movement of objects, objective vertigo, may be an impression that they are passing before the patient in a given manner, lateral, upward, or downward. As we have already seen, the direction of the objective movement is often the same as that in which the patient seems to move; this may be regarded as the typical condition; at any rate it is that which is most easily understood. In many instances, however, the direction in which objects seem to move is opposite to that of the subjective movement. The explanation of this is difficult, and may be not always the same. In some cases of this kind the vertiginous disturbance may not involve the visual centres in such a way as to give rise to the error of inference which causes the apparent movement of objects in the same direction as the subjective movement. If a person in health turns round, objects seem to move in the opposite direction, because their images move across the retina; in some cases of vertigo there may be a false impression of such a reverse movement, although there is no movement of the images on the retina.* In the same way, to a patient who fell forward, it seemed that the ground rose up to meet him, while to one who, in bed, felt as if rolling over to the left, the left side of the bed on which he was lying seemed to be rising up. This condition, in which objects seem to move as they would seem if the movement were actual, is perhaps a less complete form of vertigo than that in which objects seem to move *with* the patient.

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* Objects are not *involved* in the false inference, as in the vertigo described as typical, but there is, as it were, a subsidiary false inference of their movement in the opposite direction. Another possible explanation is that the apparent movement of objects is the result of compensatory processes to maintain the disturbed equilibrium.

In other cases, again, objects before the patient seem to move in a manner that has no exact parallel in the subjective vertigo. Every object may seem to be rotating on its own axis, or all the objects may seem to rotate around a fixed point in front of the patient. In these cases the subjective movement may be a simple one to right or left, or there may be no subjective vertigo; the apparent rotation of external objects may be the only sensation of which the patient is conscious, and this in cases of labyrinthine vertigo.

There seems to be a special relation between the labyrinthine impressions and the movements of the eyes, and in labyrinthine vertigo there may be an actual involuntary movement of the eyes. This is probably the slightest degree of the actual movement which, as we have seen, occurs in many cases, and involves the whole body; whenever there is actual rotation, the eyes and head lead the way. This point will be considered in connection with this form of giddiness.

The sensation of movement in external objects increases very much the patient's discomfort. He is often compelled to keep the eyes closed, because, as soon as he opens them, the rapid movement or whirling rotation of the objects before him, including the walls of the room in which he is lying, may be unendurable. The vertigo may also be greatly increased by movement, especially by sudden movement of the head.

There is a close association between vertigo and vomiting. Intense giddiness almost always causes vomiting. It is probable that the gastric fibres of the vagus have a connection with the middle lobe of the cerebellum, disease of which so frequently causes vomiting. It is more probable that the symptom is thus produced than that it is due to a direct connection between the auditory and vagal nuclei. It has, also, an obverse side in the influence of gastric derangement in causing vertigo, which we shall consider presently.*

OCULAR VERTIGO.

Ocular vertigo is a rare form; it is due to weakness of an ocular muscle, and depends on the erroneous projection of the visual field (see p. 597). There is an error in the unconscious inference of the relation of the body to seen objects, and this element in the impressions that influence the equilibrial centre is at variance with others, and either the discord or the disturbance of the centre is felt as giddiness. The giddiness is seldom intense, and is not independently paroxysmal; it occurs only when the affected muscle is put in action. In most cases it is very slight and transient, as the cerebral centres learn to neglect the error. Now and then it continues for a long time. It is chiefly important in respect to diagnosis. The treatment of the ocular palsy soon removes the vertigo, but if its cause is not discovered the patient may be put through long courses of useless treatment.

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* It probably underlies the pathology of sea sickness, in which the vomiting may be simply a reflex effect of the oscillations of the endolymph in the semicircular canals.

AURAL VERTIGO; LABYRINTHINE VERTIGO; MENIÈRE'S DISEASE.

The vertigo that depends on disease of the semicircular canals is sometimes termed "aural" or "auditory vertigo," sometimes "labyrinthine vertigo," sometimes "Menière's disease." "Auditory" the vertigo is not—it is generally "aural" or "labyrinthine;" it always depends on derangement of the auditory nerve, commonly on disease of the nerve endings in the labyrinth, rarely on disease of the fibres of the nerve in its trunk or origin. Attention was first drawn to this cause of vertigo by a description given by Menière* of some cases in which very violent disturbance was produced by a sudden lesion of the labyrinth. Such cases came to be termed "Menière's disease"—the great frequency with which slight disease causes slighter symptoms was not at first recognized. The name has been since applied by some to all forms, slight and severe; by others it has been restricted to the severer cases; hence there has been some confusion as to what is, and is not, "Menière's disease."

The labyrinthine form is by far the commonest of all varieties of vertigo. This is shown by the statistics given below. In nine cases out of ten in which there is definite giddiness, not epileptic in nature or obviously due to organic brain disease, it is due to a morbid state of the labyrinth or auditory nerve endings. It has been doubted by some whether this opinion is justified, whether undue significance is not given to the association of slight deafness with the vertigo, but the evidence, considered as a whole, is very strong. In the first place, we have the undoubted fact that violent vertigo may result from acute lesions of the labyrinth. This is proved by the severe cases described by Menière and others. Secondly, progressive disease of the labyrinth may be attended with severe vertigo, which may cease when the progress of the disease has produced complete deafness. These two facts show that the vertigo is the result rather of the irritation of the nerve than of its loss of function. Thirdly, in the majority of cases of definite vertigo, slight or considerable, tinnitus is present, and is evidence of a process of irritation of the fibres of the nerve. Fourthly, slight loss of hearing, tinnitus and vertigo may be observed not only to come on together, but to pass away together. Lastly, the frequency with which symptoms of labyrinthine disease are found in cases of vertigo is itself a fact of very great significance. Of 106 consecutive cases in which definite vertigo made the patient seek advice, in no less than ninety-four ear symptoms were present, tinnitus or deafness, or more often both; defect of hearing through the bone always existed, and in almost all cases in which it was slight a distinct difference between the two sides emphasized its pathological character. In conjunction with these facts we have the experimental evidence that vertiginous movements result in animals from lesions of the semicircular canals.*

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* Before the Académie de Méd. of Paris, in 1861. The association of tinnitus aurium and vertigo was noted by Burns in 1809 (" Obs. on Dis. of the Heart," p. 75).

The coincidence of auditory symptoms with vertigo depends on the fact that the lining membrane of all parts of the labyrinth is continuous, and so also is its cavity. The two sets of symptoms depend on the affection of adjacent structures, which commonly suffer together, but since they are distinct it is conceivable that labyrinthine vertigo may occur without any auditory symptoms. I have actually seen two or three cases in which definite giddiness existed alone, and in which auditory symptoms came on later. From this consideration it is easy to understand that there is no necessary proportion between the disturbance of hearing and of equilibrium.

The central relations of the two parts of the auditory nerves have been already described (p. 485). The stimulation of the nerve fibres of the semicircular canals is believed to be through the pressure of the endolymph on the hairs within the ampullæ. The three canals are arranged at right angles; the horizontal and transverse canals have their ampullæ at the outer ends, while that on the vertical canal is at its anterior-inferior extremity. If movements stimulate the nerves by increasing the pressure in the ampullæ, the fibres of the transverse canal will be excited by an inclination of the head to the opposite side, as in falling in that direction; those of the horizontal canal by a rotation of the head toward the side of the canal concerned; those of the vertical canal by a forward movement. Irritation of the canals by disease may be expected to give rise to corresponding sensations of movement. But it is probable that, in most cases, the condition is a complex one and the disturbance is seldom the result of the irritation of a single canal.

On account of the frequency with which vertigo seems to the patient himself to preponderate over the auditory symptoms, a large proportion of the sufferers seek the advice of physicians rather than of aural surgeons, and most text-books of diseases of the ear give a very imperfect representation of the malady. The following account is based upon nearly a hundred cases in which the affection was distinct.

Etiology.—Aural vertigo seems to result from almost any of the many morbid processes that involve the labyrinth and the nerve endings it contains. As a chronic symptom, however, it seldom results from inflammation of the middle ear or caries, such as are so common in childhood. It is a well-known fact that in these cases the labyrinth usually escapes.† In most cases, there are no signs of mischief in the middle ear; the affection seems to be confined to the labyrinth. Chronic, slightly irritative disease is that which seems most frequently to cause it, and such disease is most common in the second half of adult life. It is met with very seldom under twenty, occasionally between twenty and thirty, and frequently after thirty. In four-fifths of the cases the symptoms commence between thirty and sixty. For some reason, it is more common in men than in women; of ninety-three consecutive cases, sixty-two were males, and the proportion was about the same in private as in hospital patients.

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* The literature of aural vertigo is exceedingly voluminous. Among the more important papers are those of Knapp and Brunner, "Arch. of Oph. and Otol.," vol. ii; of Hughlings Jackson, *Med. Times and Gaz.*, *1872*, *Med. Record*, vol. ii, and *Lancet*, *1880*, Oct. 2d; of Charcot, *Prog. Méd.*, *1874*.

† In a curious case reported by MacBride the symptoms were caused by disease of the middle ear through the agency of defective pressure in the tympanum, and were at once relieved by inflation. (Quoted by Grainger Stewart, "On Vertigo," 1884, p. 20).

The nature of the change in the labyrinth is necessarily a matter of conjecture. In a few cases the symptoms came on after exposure to cold, and inflammation is the probable lesion. Such inflammation is generally chronic or subacute, but in one of Menière's cases, due to cold, it was so intensely acute that the patient died in a few days, from the severity of the brain disturbance produced. In many cases the mischief seems to be the result of gout, and it is highly probable that the labyrinthine membrane may suffer from this cause, as other membranes do that are connected with bones. I have more than once known all symptoms to pass away under the influence of treatment directed to the gouty state. Occasionally a syphilitic inflammation is suggested by the presence of the constitutional disease, and labyrinthine lesions have been proved to occur from this cause.* In other cases, again, the slow progressive character of the symptoms suggests that they are due to degenerative changes in the membrane, either senile in character or allied to senile changes. Atrophy of the nerve may be a cause, but exists more rarely than symptoms suggest (see p. 671). Very acute inflammation or hemorrhage, such as gave rise to the intense symptoms of the cases described by Menière, is certainly most rare. Another very rare cause is a lesion at the base of the brain, damaging the auditory and facial nerves, but such cases do not come into the class now under consideration, in which the vertigo itself is the chief symptom. (It should be noted that attacks may be excited by almost any depressing influence, in a predisposed subject.) The effect of stomach derangement will be again considered, and, in addition to this, I have known attacks to be brought on by fatigue, by excitement of various kinds, and by pain, such as toothache.

Symptoms.—The vertigo in these cases presents very great variation in its character. It is almost always paroxysmal, but there is often continuous slighter vertigo, sometimes vague, often of the same character as that in the attacks. Less commonly there is no vertigo except during the paroxysms, or for a few hours or a few days afterward. The attacks may occur at intervals of a few days or of several weeks or months; sometimes many attacks occur daily. They may come on spontaneously, or be excited by some sudden movement, occasionally by coughing or sneezing. Some of these patients can never blow the nose without reeling. An attack may come on during sleep, and wake the patient.

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* As in a case of acute deafness and giddiness in a syphilitic subject (recorded by Moos, *Virchow's Archiv*, Bd. 69), in which the whole labyrinth was occupied by semi-solid inflammatory material.

The vertigo may be subjective or objective, or both. When the patient feels as if turning or falling, there is often an actual tendency to turn or fall in the same direction during the severe attacks. A tendency to go forward or backward is very common, with or without a lateral tendency. A sense of movement in external objects is also frequent, and this may present all the varieties described at p. 1121. When both phenomena are combined, it is far more common for the two movements to correspond in their direction than for them to be in opposing directions. The other aural symptoms are often one-sided, or are much greater on one side than on the other; in such cases the sense of movement may be either toward or from the ear most affected. When the subjective and objective movements correspond in direction, they are generally toward the affected ear, rarely from it. When they differ, it is about equally common for the subjective movement to be either toward or from the ear affected, and this is also true of the apparent movement of objects. It must be remembered that when the difference in the degree of the auditory symptoms is slight, the excess does not furnish any strong ground for inferring that the cause of the vertigo is greater on that side, because the derangement of the canals may be greater on the side on which the cochlea is less affected.

The paroxysms of vertigo are generally sufficiently severe to make it difficult or impossible for the patient to stand, and in many instances he occasionally falls. Sometimes, indeed, the sufferer is hurled to the ground with violence, as if by some unseen power, much to his own astonishment and alarm. Occasionally there is a sensation as of a blow behind the ear, but this impression is probably due only to the suddenness and violence of the vertigo. When on the ground, it may be impossible for the patient to rise, so intense is the giddiness. In such cases there is certainly, sometimes, a moment's loss of consciousness, as might be expected from the intense derangement of the functions of the brain. In other cases sight may be indistinct for a few minutes. After severe vertigo has lasted for a short time, nausea comes on, followed by vomiting; the patient becomes pale, and a cold sweat breaks out. The pallor and physical depression are often extreme, and very alarming in aspect. If the giddiness persists, the vomiting may go on for some hours, and after the stomach has been emptied, bile is brought up, as is usual in continued vomiting; this is popularly regarded as conclusive proof that the attack was due to "biliousness." The vertigo is often increased by any movement of the head, and the patient may be unable to raise the head from the pillow without being sick. Gradually, however, the attack passes away, but for a few days the patient experiences more than the ordinary amount of vertigo, and the interference with the functions of the stomach occasions some indigestion, and especially some diarrhœa.

Often the paroxysms are much slighter in character, and are unattended by vomiting, or even by nausea. There may be merely a sudden tendency to fall, or sudden movement of objects, or a tendency to deviate to one side in walking.

Ocular symptoms, secondary in origin, are present in some instances.* In cases of ear disease, an increase of pressure within the ear, as by pressing firmly the antitragus over the opening of the meatus, may cause nystagmus. During paroxysms of vertigo, the patient may be conscious of a jerky movement of objects, a quick motion in one direction and slow return, like that sometimes produced by nystagmus, and I have known it to correspond with intermitting tinnitus. This apparent movement, as well as nystagmus, may sometimes be caused by pressure on the meatus.† I have several times known double vision to occur during or after a paroxysm; in one case of pure aural vertigo, each attack was followed by double vision, jerky movement of objects, and distinct erroneous projection in the direction of the movement, so that, if the patient attempted to touch an object, the hand went too far in that direction. Slight diplopia is sometimes due to nystagmus that is not quite equal in the two eyes.

In most of the cases, the auditory symptoms comprise both tinnitus and deafness. These correspond in side, except in rare cases in which one is bilateral. The deafness may present every degree of intensity, but is generally sufficient to be a source of trouble to the patient, and always involves hearing through the bone (see p. 672). In the cases in which the loss of hearing is slight and unknown to the patient, it is generally one-sided; the watch can be heard loudly through the bone on one side, and not at all on the other, or the notes of Galton's whistle (very high-pitched sounds) are inaudible on one side, and the loss is emphasized by corresponding tinnitus. On the other hand, absolute deafness is scarcely ever met with, perhaps because when all hearing is lost vertigo usually ceases.

Tinnitus is present, at some time, in the vast majority of the cases, and in almost all is persistent. It may present most of the variations described at p. 677. A continuous sound is the most common; pulsating sounds are occasionally described, but are less frequent than in the tinnitus that occurs without vertigo. The intensity of the sound is usually moderate, and sometimes slight, but it often becomes more intense at the onset of a paroxysm of vertigo, and may then become very loud. Thus one patient who habitually heard a noise like a distant waterfall, said that with the vertigo it rapidly increased in loudness, and was like an express train coming past a station, and as it became loudest "it seemed to force on the giddiness." It is exceedingly rare for the noise to be confined to the paroxysms of vertigo. On the other hand, it is not at all uncommon for there to be no increase of the tinnitus on the occurrence of the giddiness.

An attack of vertigo, however severe and however alarming it may be to the sufferer, is attended with little danger. Nevertheless it is possible that, in a patient with a feeble heart, the prostration may go on to fatal syncope. The chief danger, however, is in the cases, fortunately extremely rare, in which the labyrinthine lesion is intensely irritating, and the cerebral disturbance induced is so intense as to lead to fatal exhaustion.

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* According to Cyon, irritation of each of the semicircular canals produces its own special effect on the position of the eyes.

† In *Brain*, vol. iii, Dr. Hughlings Jackson describes a case of aural vertigo in which objects appeared to move from the affected ear in nystagmic jerks, and quotes a case of ear disease in which pressure caused objects to appear to move toward the affected ear. In several cases a similar spontaneous movement has been described to me, and cases are not at all rare in which it can be produced by pressure.

The course of aural vertigo varies according to the nature of the morbid process. If this is steadily progressive, the symptoms may only cease when all hearing power is destroyed. Happily, however, these cases are rare ; in the majority either the changes in the labyrinth are not progressive, and the tendency to giddiness lessens in the course of time—or else the tendency can be kept down by treatment. I have known perfect recovery to occur in many cases in which the attacks of giddiness were most severe.

PATHOLOGY.—The chief facts regarding the pathology of labyrinthine vertigo have been already mentioned, but one or two other points remain for consideration. We must refer the vertigo to changes in the semicircular canals, but how it results from these changes we do not know. Apparently it is by the stimulation of the fibres, rather than by simple diminution of function, that the giddiness is produced. This conclusion is suggested by the results of experiments on animals, and is in harmony with the fact that there may be gradual loss of function, to judge by progressive deafness, without vertigo. Thus the morbid process is more nearly allied to that which causes tinnitus than to that which causes deafness.

It is possible, however, that the irritation of the canal fibres may produce vertigo in more than one way. Sometimes the sensation may be the direct effect of the morbid action, but, as we have seen, it is highly probable that the paroxysms of vertigo are only indirect effects of the labyrinthine irritation. The latter brings the centre for equilibration (or the centre in which the centripetal influences are coördinated) into a state of instability, in which a sudden violent derangement may occur on some slight exciting influence, or even without any excitant that can be traced. This conclusion is indicated by the extreme intensity of many paroxysms, in which the sufferer is hurled to the ground with convulsive violence, without any coincident indication of special aural disturbance. In some cases a slight aural disturbance may perhaps excite the paroxysm, but even when a sudden loud subjective sound occurs as the vertigo comes on, we cannot take this as proof of a labyrinthine irritation of corresponding intensity, because it is possible, and even probable, that the sound is the result of the vertiginous discharge spreading to the centres through which normal tinnitus is perceived, and that the sound is thus part of the attack, rather than an indication of its cause. This view of the nature of the attacks enables us to understand that severe paroxysms may occur when the labyrinthine change is apparently slight, and also the coöperation of stomach disturbance in exciting the paroxysms.

DIAGNOSIS.—The diagnosis of labyrinthine vertigo depends on the coincidence of vertigo of definite character with indications of derangement of the functions of the labyrinth—tinnitus, and deafness not due to impairment of the conduction through the external meatus or middle ear. The indications of this have been already described (p. 672). If the loss of hearing is trifling, its significance is greater if it is one-sided.* It must be remembered that the fact that paroxysms are excited by stomach disturbance does not prove that the vertigo is simply gastric. I know a gentleman who all his life has been liable to attacks of acute dyspepsia, but they were never attended with giddiness until he became deaf; afterward, each dyspeptic attack was accompanied by severe vertigo. The diagnosis of aural vertigo is occasionally helped by the fact that giddiness may be brought on by a sudden movement of the head in one direction and not in another, or by suddenly increasing the pressure in one ear.

Cases of epilepsy, in which the aura is an auditory sensation accompanied by giddiness, may be mistaken for labyrinthine vertigo. The distinction depends on the fact that in the latter there is usually persistent tinnitus, impaired audition, and more or less constant slight vertigo, while loss of consciousness is extremely rare, and is confined to an occasional very violent paroxysm. Such a paroxysm is followed by vomiting and by prolonged giddiness. In epilepsy, loss of consciousness is the rule; there is no subsequent vertigo, and indications of a convulsive seizure can usually be ascertained. It must be remembered that the two diseases sometimes coexist.

It is a very common thing for aural vertigo to be mistaken for a slight attack of organic cerebral disease, congestion, or an actual vascular lesion. The error generally arises from ignorance of the occurrence and frequency of labyrinthine vertigo, and it is then a mistake easily made, because the prostration and pallor that are the consequence of the giddiness are very like those that result from a cerebral lesion. The diagnosis rests on the absence of other symptoms of such a lesion on the one hand, and on the presence of aural symptoms on the other, while in most cases the decision is much helped by the fact that the patient has had other attacks of simple giddiness, which are as significant if they have been slight as if they have been severe. In a cerebral attack, loss of consciousness is out of proportion to the vertigo, whereas in the other it is slight, often imperfect, and clearly subordinate to the intensity of the giddiness.

PROGNOSIS.—The prognosis is distinctly serious chiefly in cases of steadily progressive disease of the labyrinth, in which the symptoms may persist in spite of all treatment, until complete destruction of the nerve endings brings a cessation alike of the giddiness and of the power of hearing. It is still more grave in the case of extreme severity from acute lesions, but these are so rare that they scarcely influence the general prognosis. In most other cases the prognosis, although uncertain, is not definitely bad; improvement is exceedingly common, and in many cases goes on to recovery.

TREATMENT.—In the treatment of aural vertigo we must recognize the double element in the pathology of the disease, the labyrinthine irritation, and the central instability induced by the irritation; the latter may be to some extent in excess of its cause. For the central condition no agent has so much influence as bromide, which almost always lessens the tendency to vertigo, and, when this is slight, may remove it altogether. Twenty grains should be given two or three times a day, and the addition to each dose of a few minims of tincture of belladonna seems to increase its effect. Hydrobromic acid has been recommended, but must be converted into bromide in the blood, and only a quite inadequate dose of bromide can be given in the acid form.

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* In one case, for instance, even the watch could be heard perfectly through the bone on either side, but Galton's whistle was quite inaudible with one ear, and readily heard on the other side. This was absolute proof of changes in the nerve endings.

The local irritability is commonly lessened by counter-irritation, whether the lesion is inflammatory or degenerative. The most effective counter-irritant is a small blister over the mastoid process. It is remarkable how rapid and marked may be the effect of a blister on all the symptoms, especially in recent cases. Drugs, unfortunately, have very little influence over morbid processes in the labyrinth, unless these are of a specific nature. Syphilitic inflammation can be readily removed, and the more common gouty changes can be lessened in a very marked degree by appropriate treatment. In the latter class I have several times known treatment remove not only the vertigo, but the tinnitus and the deafness, so that even the power of hearing through the bone, before completely lost, became normal. Purgatives, alkalies, and colchicum were the effective agents, but it is desirable to employ also counter-irritation, and at first to give bromide also, in order to lessen the morbid irritability of the centre. The bromide of lithium is a convenient salt to use in these cases, if its higher price is not an obstacle. It must be remembered, however, that degenerative changes occur earlier in gouty subjects than in others, and hence, in such patients, the change in the labyrinth does not always prove amenable to the special treatment.

We can get little help from drugs in dealing with other chronic changes in the labyrinth. Charcot first suggested that agents which have a special action on this structure may possibly exert an influence antagonistic to the morbid process. With this object he gave large doses of quinine, so as to produce cinchonism. The patients were worse for the time being, but when the influence of the quinine had passed away, some of them were distinctly better. I have not found the effect of quinine so satisfactory as that of salicylate of soda, and I think that more good is done by giving it in moderate doses, five grains three times a day, than by administering it in such quantities as to produce toxic ear symptoms. In several cases in which other treatment had failed, the moderate doses of salicylate rendered both the vertigo and tinnitus less troublesome.

It is important to treat any morbid influence that may coöperate in exciting the vertigo. The general health must be improved by tonics; exposure of the head to cold should be avoided, and the bowels should be kept open. Of special importance is the treatment of the dyspepsia which often coexists and has a very powerful influence in exciting the giddiness. As far as practicable, patients who are liable to vertigo should avoid stooping, and sudden movements of the head.

Nocturnal Vertigo.—Most persons, perhaps all, have been occasionally disturbed when falling asleep, or just after going to sleep, by a sudden sensation of falling from a height. Sometimes it is accompanied by a dream. I believe that this is really slight labyrinthine vertigo, due to spasmodic contraction of a tympanic muscle, which suddenly changes the pressure within the labyrinth. Those who wake up quickly during this sensation may distinctly hear the peculiar vibratory sound characteristic of intra-aural muscular contraction. It is identical in character with that which many persons may produce at will by contracting the orbicularis palpebrarum, and at the same time turning the eyeballs upward. What muscle contracts is uncertain; it is perhaps the stapedius, which would suddenly lessen the pressure in the labyrinth. As soon as the sound of the contraction ceases, the sense of falling ceases also. These attacks may be prevented by a dose of bromide.

OTHER FORMS OF VERTIGO.

We have seen that it is exceedingly rare for definite vertigo to occur apart from aural symptoms, and it is certain that, in the majority of cases in which such vertigo has been ascribed to other causes, these have only had an exciting influence, and the symptom has been essentially due to the effect of unobtrusive labyrinthine disease, bringing the centre into an unstable condition. This is certainly true of the majority of cases of *gastric vertigo* which was formerly thought to be so common; the mere presence of dyspepsia was regarded as a sufficient explanation of the giddiness. Certainly vertigo of purely gastric origin does not constitute more than five per cent. of the cases in which definite giddiness is the prominent symptom.* The giddiness met with in such cases is similar to that above described as met with in the aural form. The diagnosis rests on the fact that it distinctly follows stomach disturbance and no other cause can be discovered. The treatment needed is, first, that for the gastric disorder, and secondly, the reduction of the central irritability by bromide.

Vertigo occurs, in slight and vague form—a mere sense of unsteadiness—as a symptom of many morbid conditions, in which, however, it is subordinate to other more characteristic symptoms. Thus it is met with in anæmia, in hysteria, and in various conditions of nervous weakness. It is met with, although rarely, as part of an attack of migraine. It occurs, also, in the old whose brains are ill nourished, in consequence, perhaps, of arterial degeneration, and also at the onset of various vascular lesions of the brain, of which it may be a premonitory symptom. It is conspicuous in some cases of intracranial tumor, especially tumors of the cerebellum or of the pons Varolii (see pp. 530 and 718).

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* I do not think it is quite certain that there is such a thing as definite vertigo of purely gastric origin. Thirty years ago 80 per cent. of cases of giddiness were supposed to be due solely to the stomach. But we now know that in 90 per cent. of the cases of definite giddiness a morbid state of the labyrinth is the real cause of the vertigo. It is possible that in the small remainder, of apparently stomach giddiness, there is some other influence that is the real cause, *e. g.* a morbid state of the semicircular canals causing no auditory symptoms, and so not to be detected save by its effects.

Definite vertigo is occasionally met with apart from any recognizable morbid state, aural, gastric, or other, to which it can be ascribed. This form, in ignorance of its nature, has been termed *essential vertigo*. Cases so described were formerly often met with; now they are extremely rare, and it is not unlikely that, with more exact observation, they will disappear altogether. The treatment of such cases is that of vertigo generally.

NEURALGIA.

The word "neuralgia" means simply "nerve pain;" such pain may be due to actual disease of a nerve trunk, by which its fibres are irritated, or it may occur without any organic lesion. It is true that the absence of such morbid change cannot often be proved by actual microscopical examination, but the transient character of the pain, and its migration from one part to another, frequently afford strong confirmation of the opinion that, in a large number of the cases of local nerve pain, the symptoms are not due to actual organic change. Two classes of neuralgias have been distinguished—"symptomatic" in which the pain is a symptom of organic disease of the nerves, and "idiopathic," in which the malady, in the first instance, at least, consists only in functional disturbance. Both of these forms have been called "neuralgia" by some writers. By others, however, the term is restricted to the idiopathic class, and this seems to be the only logical course. It is manifestly unreasonable to describe inflammation of nerves as "neuralgia" when it causes much pain and few other symptoms, and as "neuritis" when other symptoms predominate over pain. But the distinction of the two forms of nerve pain is often very difficult in practice. In some forms described as neuralgia, the pain has certainly been generally the result of neuritis. This is the case, for instance, with sciatica, which, as stated in the account of that disease (page 103), is generally an inflammation of the nerve. Yet, not only has the common form of sciatica been described as a neuralgia, but its symptoms have been allowed to influence the description of neuralgia in general. In ascertaining the clinical history of neuralgia, it is therefore of great importance that only cases should be used in which a primary organic lesion of the nerve trunk or centres can be excluded with reasonable confidence. This limitation does not, however, exclude organic lesions of nerve endings. In many cases some structural irritation of the termination of nerves sets up a neuralgia of wide range, and out of all proportion to its cause. Such cases are classed among neuralgias, and are very different from those in which a primary lesion of a nerve trunk causes local and limited pain.

The subject of neuralgia is a very large one, so numerous are the forms of the disease, and so varied its characters. It will be most instructive to consider the general causes and symptoms of the affection, and also its general pathology, before describing its special varieties.

Etiology.—Neuralgia is essentially a disease of adult life. It is rare before puberty, and is not common in extreme old age, although, when the disease does commence late in life, it is often very severe and intractable. Most cases commence between twenty and sixty years. Children enjoy an almost complete immunity from true neuralgia, although very liable to certain headaches which do not come under this category. Women are more prone to neuralgia than men, but the degree of their liability has often been over-estimated, and the excess of females among the sufferers disappears in the second half of life. Moreover, the relative liability of the sexes is not the same in the several varieties. The tendency to neuralgia is often hereditary, although not so frequently as in the case of migraine. Anstie found evidence of heredity in only one-quarter of his cases. Sometimes the inherited tendency is not special but general, indicated by the occurrence in ancestors or collaterals of epilepsy, insanity, and other neuroses.

The subjects of neuralgia often present a peculiar temperament. They are what is popularly called " nervous "—excitable, often irritable, anxious, worrying over the trifling ills of life, sleeping badly, and are often extremely liable to headaches not distinctly neuralgic in character. In many patients the neuralgic tendency is deeply rooted; they suffer from neuralgia first in one situation and then in another, during the course of years. The disease is more frequent in those of weakly constitution than in the robust, but the latter do not enjoy complete immunity. Among the constitutional relations of neuralgia, those to rheumatism and gout are especially important. The connection with rheumatism is often conspicuous, and is seen in several aspects. Persons who are liable to rheumatism of the fibrous tissues sometimes suffer from pains which have both rheumatic and neuralgic characters—not specially related to the nerves in situation, and yet paroxysmal and unconnected with movement. Such pains are especially frequent in the limbs and back. Women who suffer from rheumatoid arthritis are often also liable to true neuralgias of great severity. A young lady, for instance, suffered for several months from severe paroxysms of pain in one shoulder and the dorsal spine, apparently neuralgic; these ceased, and she was immediately attacked by subacute rheumatoid arthritis. Lastly, both rheumatic affections and neuralgia are certainly sometimes due to gout, and probably, not infrequently, to inherited gout.

Among the exciting causes of neuralgia, as among those that are remote, impairment of general health takes the first place. The affection may be excited by any kind of debilitating influence; overwork of mind or body, over-lactation, prolonged fatigue, and anæmia of every degree and causation, are frequently met with as its immediate antecedents. Certain forms of sensory fatigue, as over-stimulation of the eyes, sometimes seem to have a special influence. Severe emotion, or its physical analogue, mechanical concussion, are also occasional exciting causes; the former is often combined with the latter. As examples of the influence of emotion two cases may be mentioned, one of slight, the other of severe, neuralgia, thus induced. A lady was intensely distressed after parting with her husband, who was going to America. She felt on the point of bursting into tears, and as if the tears would give her relief. Her sister said, " Do not cry: you *shall not* cry." By an effort she succeeded in restraining her tears, but was immediately conscious of a sense of intense pressure above the eyebrows, and a few days later severe supra-orbital neuralgia came on upon the left side, and lasted for several weeks. A girl of eighteen was much startled and alarmed by the unexpected discharge of a gun close beside her. The same evening facial neuralgic pain came on, and continued for five years in most violent paroxysms, sometimes on one side, sometimes on the other. In this connection it may be noted that the fifth nerve is especially related to emotion, both by influencing the secretion of tears and also as the sensory nerve of the chief region of emotional display—the face.

No single actual excitant of neuralgia is so frequent as exposure to cold, sometimes general, sometimes local and affecting the part in which the neuralgia is felt. Valleix found a history of exposure to cold in one-third of his cases. Cold may not only produce neuralgia, it may also excite paroxysms of pain when the neuralgia is due to some other cause. Another frequent cause is the irritation of nerves, especially near their peripheral distribution. The pain often extends far beyond the area supplied by the irritated nerve. A common example is the widespread pain that may result from the irritation of a carious tooth; the pain may extend into other divisions of the fifth nerve, and even into the region of the cervical plexus. Moreover, the pain may be felt only or chiefly in some other region than that in which it is produced. Thus, I have known severe neuralgia confined to the second division of the fifth to be due to a carious tooth in the lower jaw, and cease entirely when this was extracted. Traumatic lesions of nerves constitute another cause, relatively infrequent, but very important on account of the extreme obstinacy of the pain they cause.

Toxic influences often give rise to neuralgia. The most frequent are alcoholism, lead poisoning and the presence of an excess of sugar in the blood. The influence of some of these causes in producing neuralgia is, however, probably less than has been assumed, because we now know that the nerve pains that are caused by them are sometimes the expression of actual neuritis. This is especially the case with the pains of alcoholism. Nevertheless, when all cases are excluded in which there is reason to believe that neuritis exists, there remain many others, which, from their character, must be regarded as neuralgic. It is intelligible that a condition which has such an influence as to produce actual inflammation of nerves, should often irritate them so as to cause neuralgia. This statement is also applicable to gout, the influence of which has been already mentioned. Both neuritis and neuralgia may unquestionably result from acquired and inherited gout. Malaria is a powerful cause of neuralgia, but its influence is seldom seen in this country.

SYMPTOMS GENERALLY.—The great symptom of neuralgia is pain, spontaneous, paroxysmal, and felt in certain regions of nerve distribution. It is usually unilateral; when bilateral it is almost always symmetrical in distribution. Usually the pain is constant in seat for a time, it may be for many years. In other cases it changes, now in one part, now in another. Thus, a girl aged fifteen had suffered for two years from paroxysms of intense pain in various parts—arms, legs, back, different parts of the head—and occasionally universal.

The pain is never constant in degree; there are paroxysms, with intervals of complete freedom, or there is a slight, continuous pain, with intense exacerbations. Continuous pain may be merely a dull ache, but it is generally acute and sharp during the paroxysms, and is described as "darting," "stabbing," "boring," "burning," etc.; often the sufferer can find no words to express its exact character. The sharp pain generally has a darting character. A series of sudden sharp pains occur every few minutes; the series of successive pains constitute a paroxysm, and a series of paroxysms an attack. The intervals between the attacks present extreme variation, and are sometimes remarkably long in proportion to the severity of the pain. Thus, one patient will have attacks daily during several years, while in another (as in an actual instance) intervals of many months separate groups of attacks of most intense pain, each group lasting only a few days. In such paroxysms it is usual for the pains to commence suddenly, but they are rarely as severe at first as they subsequently become. Sometimes a peculiar sensation, such as throbbing, heralds each attack of pain. The attacks gradually increase in intensity, and in each attack the separate paroxysms may present a characteristic augmentation and decrease.

The pain is rarely referred to the skin; usually it is more deeply seated, and often corresponds to the position of a nerve trunk and branches. The throbs of pain sometimes, but seldom, coincide with the arterial pulsations. There is often a darting movement of the pain, usually toward the periphery —centrifugal—less commonly from the periphery—centripetal; still less commonly the pain darts alternately in both directions. Sometimes it seems to be localized in a single point, and then has usually a boring character. When most intense the darting pains seem to radiate to other nerve regions adjacent to that in which it is chiefly felt.

The duration of each attack varies according to the number of paroxysms and their length. Rarely there is a single momentary pain, and it is over for the time. Commonly an attack lasts several minutes, sometimes for hours. When there is freedom from pain, in the intervals there is sometimes a peculiar sensation in the part, not amounting to actual pain. The intervals vary in duration from a few hours to several months. Often an approximate periodicity exists; exact periodicity is met with in malarial cases, in which it may be singularly precise, the pain commencing at the same hour each day. The intervals in such cases rarely exceed four days. Now and then the periodicity is exact in cases that are not malarial. The pain may be worse at the catamenial periods, and I have once known a fifth-nerve neuralgia to occur only at those times. When there is continuous pain in the intervals between the paroxysms, it is moderate in degree, supportable in itself, but often most trying to the patient, as it prevents rest.

The paroxysms and attacks are often induced by certain influences, external or internal, such as by exposure to cold, sometimes by warmth, by movement, posture, or emotion. In some cases there is remarkable sensitiveness to atmospheric influences. When the paroxysms occur at regular intervals, an influence that will induce the pain when it is "due," may be powerless immediately after an attack. During a paroxysm, the influences that will bring it on usually intensify the pain. Movement is especially influential in the neuralgias of the fifth nerve; the slightest motion of the jaws may bring on the pain. A touch on the skin may have the same effect; nevertheless, in many cases, although slight pressure increases the pain, firm pressure gives distinct relief, and even when mere contact with the skin causes an exacerbation, rough rubbing may distinctly relieve the suffering. This difference, however, is not always to be observed. Occasionally alcohol, even in small quantities, invariably intensifies or induces the pain; in other cases it gives relief.*

This increased sensitiveness of the skin is a very common accompaniment of the pain. It may involve all forms of sensation, although thermic impressions much less commonly occasion pain than does a touch. Sometimes the tactile impression seems to be felt as pain; more often it excites an increase in the true neuralgic pain. The hyperæsthesia, or hyperalgesia, is usually limited to the region in which the spontaneous pain is felt. When this pain follows the course of a nerve, it is commonly most intense at certain spots, and at these places pressure may cause a special increase in the suffering. In the intervals between the paroxysms, these spots may remain tender, and pressure upon them may induce a paroxysm. They are not usually present until the disease has lasted for some time. In recent cases, and when the attack occurs only at long intervals, although there is no persistent tenderness, the pain may be increased during the paroxysm by pressure on certain places. The tender points were first studied by Valleix, and hence are often called after him. They are present in about half the cases; when absent there is sometimes diffuse, ill-defined tenderness in certain areas. When the tender points are well marked and definite, they are tolerably uniform in their position, and for the most part correspond either to the place at which a nerve trunk emerges from a bony canal, passes over a hard structure, or passes through a fascia to become superficial, or to the point of division of a nerve trunk, or to an anastomosis of two nerve trunks. Their exact situation will be mentioned in the description of the several varieties of neuralgia.

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* Very curious facts are sometimes met with in regard to the induction of the pain. Thus, in one patient, intense fronto-occipital neuralgic pain was excited by every act of defecation. This action, in some cases, has a very peculiar influence on the nervous system, and so also has micturition, as the familiar shiver shows. I have known micturition to be frequently attended with a moment's loss of consciousness.

There is occasionally tenderness of the vertebral spine corresponding to the origin of the painful nerve, the *point apophysaire* of Trousseau. It is probable, as Anstie pointed out, that the relation of this to neuralgia has been exaggerated. Tenderness of certain vertebral spines is common apart from neuralgia, and there is not always a close correspondence between the position of the spinal tenderness and the seat of the neuralgia. In trigeminal neuralgia, for instance, there may be tenderness of the cervical spines. It is said (by Brenner) that the spots of spinal tenderness, when undiscoverable by pressure, may sometimes be detected by a weak voltaic current, causing, in those positions, distinct pain.

Other sensory disturbances are occasionally observed in neuralgia. It is said that the onset of the pain is sometimes preceded by numbness, tingling, etc., in the affected area, but it is doubtful whether this is true of simple neuralgia. Occasionally the attack of pain is followed by transient anæsthesia. Persistent diminution of sensibility is only met with in cases of "symptomatic neuralgia," in which there are structural changes in the nerves. Increased sensitiveness to pain (hyperalgesia) in the whole area of the neuralgia is not uncommon. Vomiting is rarely associated with simple neuralgia, although in migraine, the pain which terminates in vomiting is often of a neuralgic character, either localized in the temporal branch of the fifth, or more extensively distributed over the cranium. Now and then paroxysms of true neuralgia end in vomiting, an interesting link of association with migraine. I have met with this in two cases of neuralgia; one, bilateral, in the anterior branches of the cervical plexus, and the other in the two upper divisions of one fifth nerve. Severe attacks in women often cause hysterical symptoms as the pain is subsiding.

Muscular spasm may be excited by the acute paroxysms of pain, evidently in a reflex manner. It is usually confined to the motor nerve related to that which is the seat of the pain, but sometimes spreads to adjacent areas, very rarely passing into a general convulsion. In a case of cranio-spinal neuralgia, each paroxysm was attended by opisthotonos so severe that the patient rested on the head and the heels. The exacerbation of the pain by movement may lead to temporary diminution of mobility, partly voluntary, partly of inhibitory origin. Herpes is very rarely, if ever, a consequence of true neuralgia, although pain so often accompanies herpes. The hair of the part may undergo changes; it may lose its pigment, fall off, or very rarely overgrow. Anstie observed temporary grayness of a lock of hair after each attack, followed at last by permanent loss of pigment.*

Vasomotor disturbance often accompanies a paroxysm. The first effect of the pain is usually to cause a constriction of the vessels of the part, but this is often followed by their relaxation; flushing of the skin results, and the throbbing of the arteries may considerably intensify the pain. The arterial dilatation may be general, and be demonstrable by sphygmographic tracings (Anstie). In one case of trigeminal neuralgia all the veins of that side of the face became distended during the paroxysm, and as the pain subsided pallor replaced the flushing. The local vascular disturbance may cause local sweating, or local œdema, or even erythema, sometimes mistaken for erysipelas. The œdema thus produced is occasionally considerable; I have known each attack of cranial neuralgia to be accompanied by great swelling of the whole scalp, due to such œdema, which slowly disappeared some hours after the cessation of the pain. In another curious case, attacks of pain in the tongue and face were attended by swelling of each part, which usually came on during the night, and sometimes occurred with very little pain. Repeated attacks of such vasomotor disturbance may lead to permanent dilatation of the vessels of the surface, and, after a time, to thickening of the cellular tissue, periosteum, and other structures.

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* A very remarkable case has been recorded by Raymond, in which, at the time neuralgic pain in the head was most intense, all the hair of the patient (a woman aged thirty-eight) changed color from black to red, and in a few days to white, and then, in the course of fourteen days, fell off (*Revue de Méd.*, Sept., 1882.)

Pathology.—Few questions have been the subject of more controversy than the pathology of neuralgia. The difference of opinion is largely due to the different senses in which, as we have seen, the word has been used. The problem of pathology is, What is the nature of nerve pain that has no known organic cause?

In neuralgia we have two symptoms, first, spontaneous pain, and, secondly, "hyperæsthesia" (more properly hyperalgesia), *i. e.* the transformation into pain of sensations that are not usually painful (or excitation of pain by them); the former includes the latter, and therefore must be first considered. Spontaneous pain means the action of sensory nerve elements apart from local external stimulation. The pain corresponds to certain peripheral nerve areas, and we must therefore look for its cause to the elements constituting a peripheral nerve structure. These are the nerve fibres, their peripheral end organs, and the central cells with which the fibres are connected. To which of these can we ascribe a functional activity independent of external stimulation? We know nothing of a capacity for such action on the part of nerve fibres. They possess a limited power of transforming external energy into nerve force, which constitutes their "excitablity," and they "conduct" nerve force, but there is no evidence to show that, apart from such external influences, they are capable of the independent evolution of nerve force. Nor are any facts known which would suggest that the peripheral end organs of the sensory nerves are capable of such independent function. We are thus reduced, by exclusion, to the central terminations of the nerve fibres as the source of the pain in idiopathic neuralgia.* The fibres end in nerve cells, and nerve cells, as far as is at present known, are the only elements capable of the independent evolution of nerve force. The conclusion is further corroborated by physiological facts, which show that the sensation of pain depends on a special function of nerve cells. There are separate structures for the sensations of pain and touch; this is proved by the fact that either tactile or painful sensibility may be lost without the other. But nerve fibres are indifferent structures. Even between the fibres which conduct sensation, and those which conduct motion, there is no difference; the one may be experimentally substituted for the other without affecting function (Bidder, Vulpian and others), and it is therefore *a fortiori* probable that there is no difference between the fibres which conduct tactile and painful impressions. The different effect depends on the central connection of the fibres—on their nerve cells. Some of these, when stimulated by the afferent impulse, give rise to tactile sensations, others to pain, and it is probable that the former, when excited in a special manner or degree, may also give rise to pain. We must therefore regard the pain of idiopathic neuralgia as central,* as due to the spontaneous activity of the cells that constitute the central termination of the nerves. When an external cause (*e. g.* an injury of the nerve) causes pain, the sensation is due to the stimulation of these cells, and the pain which results from their spontaneous "discharge" is necessarily referred to the region to which their fibres are distributed.

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* The central theory has been adopted, among others, by Vulpian, Anstie, Clifford Allbutt and Vanlair, although the theories of these authors differ somewhat in their detail.

But the sensory fibres end in two sets of cells, those of the ganglia on the posterior roots, and those within the spinal cord, chiefly in the posterior cornua. There is no direct evidence to show which of these series of cells is concerned in neuralgia. But we know nothing of any sensory function of the ganglia, and we are therefore justified in looking to the nerve cells within the cerebro-spinal axis as the seat of morbid process.

This conclusion is indirectly corroborated by the symptoms of many cases of neuralgia. Especially significant are: (1) the fact that the pain may occupy adjacent parts of several nerve regions. For instance, in one patient the pain extended on both sides from the seventh cervical vertebra, over the whole occiput and vertex to the coronal suture. (2) The phenomenon of radiation of slighter pain into adjacent nerve regions during severe paroxysms. (3) The phenomena of reflex neuralgia, in which the pain is felt in another region than that of the nerve irritated. All these are explicable only on the theory that the morbid action, felt as pain, is in the central cells, which are no doubt connected according to the relations of the surface regions from which they receive impressions, and to which their disturbance is subjectively referred.

The same conclusion (the central nature of neuralgia) is also indirectly corroborated by facts of pathology of another kind which prove that pain of neuralgic character may be produced by an organic lesion in the gray matter which is here supposed to be deranged in idiopathic neuralgia. The lesion, for instance, involving part of the sensory nucleus of the fifth nerve, shown in Fig. 223 (B x), p. 505, caused severe neuralgic pain in the face.

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* The central nature of idiopathic neuralgia was insisted on by the late Dr. Anstie (article, "Neuralgia," in "Reynolds' System of Medicine," and in the separate monograph, "Neuralgia, and its Counterfeits"), with characteristic ability, energy, and fertility of argument. But his position was unnecessarily weakened by the theory that the disease originated in the roots of the nerves as a "primary atrophy." At the same time it is right to say that some passages suggest that he included the nerve cells in which the fibres end in his conception of roots. That the seat of the disease is frequently the sensory cells of the central nuclei has been urged by Vulpian and also by Vanlair ("Les Nevralgies," 1882).

If, therefore, we regard idiopathic neuralgia as the result of the over-action, the "discharge," of the nerve cells constituting the proximate centre of the nerve, the question still remains, To what is this discharge due? It is often ascribed to hyperæmia of the centre, to dilatation of its vessels. The possibility of this cause cannot be denied, but neither can its efficiency be proved. It is an hypothesis, moreover, which only solves one problem by the introduction of another. Vasomotor disturbance means the deranged action of the vasomotor centre for that territory, and it is as difficult to explain the disturbed action of the vasomotor cells as of the sensory cells. All cells possess the power of evolving force; the discharge of the sensory cells is all we have evidence of, and it seems unjustifiable to assume the intervention of other cells for its production. But it is highly probable that secondary vasomotor disturbance may result. We know that in all organs vascular dilatation attends functional activity. If the cerebral cortex is stimulated by electricity, local dilatation of vessels quickly follows. It is probable that such secondary central hyperæmia may result from the discharge of other centres, and when established may increase the disturbance.

The over-action of the cells in idiopathic neuralgia has been spoken of as "independent." It is independent so far as our means of observation go. But we cannot tell to what extent the unstable cells may be excited to discharge by stimuli coming from the periphery. We know that some stimuli of this kind (cold or pressure) are effective in exciting attacks. It is highly probable that the afferent impressions of which we are conscious bear but a small proportion to those of which we are unconscious, which, continuous or intermittent, are due to slight cutaneous impressions, to the movement of the blood and to the nutritional processes in the tissues. It is quite possible that such afferent impulses, too trifling to affect our consciousness even when aided by attention, may excite the discharge of the unstable cells. That a strong sensory impression (*e. g.* painful pressure on the nerve) may sometimes relieve the pain is quite in harmony with familiar facts as to the effect of sensory stimulation; a strong stimulus may inhibit reflex action which may be excited by a slighter stimulus in the same nerve region. These two facts deprive of much of its force an objection which has been often urged against the central theory of neuralgia, that the disease may sometimes be permanently cured by division of the nerve. Division of a nerve does two things, it stops all impressions from the periphery, and it effects a very powerful stimulation of the centre, and to the conjoint effect of these two influences its success (not frequent) in idiopathic neuralgia may reasonably be ascribed.*

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* It is possible, as Jaccoud has assumed, that the pressure acts partly by arresting the peripheral impressions. But that this is its chief action is doubtful from the fact that pressure on another adjacent nerve is sometimes effectual, and so also is pressure on the peripheral side

We are accustomed to think of the sensory nerves as distributed chiefly to the skin, but the pain in neuralgia is rarely confined to the skin. It appears to the sufferer to be more deeply seated, and often corresponds in position to the nerve trunk and branches. The fibrous sheaths of both are abundantly supplied with nerves—the *nervi nervorum*,* which ramify and end in the sheath without penetrating the inter-fascicular septa. If a nerve is compressed (as the ulnar at the elbow) the first sensation experienced is a pain at the spot, from the stimulation of the sheath nerves; if the pressure is continued there is also a sensation (tingling) referred to the peripheral distribution of the nerve in the hand, due to the stimulation of the fibres of the nerve itself. In neuralgia, the central cells of the sheath fibres often seem to be disturbed in function more than those of the fibres of the nerve itself; the pain is referred to the nerve trunk rather than to the skin.

The fact that the pain seems to dart along the nerve must be due to the spread of the discharge in the centre in a certain order from cell to cell. We cannot at present say precisely on what this depends, or why the pain seems sometimes to dart toward, and sometimes from, the periphery.†

The irradiation of the pain, in severe attacks, to neighboring nerve areas is clearly, as already mentioned, a central phenomenon. An intense discharge always tends to spread to other connected cells, in proportion to its intensity, as the phenomena of epilepsy abundantly illustrate.

The origin of the tender points is obscure, and has been the subject of

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of the painful point. Cartaz has suggested that in the latter two cases the recurrent nerves may convey the pain and be compressed, but such an explanation does not apply to the effect of cutaneous irritation in arresting the pain, which is a phenomenon of the same class.

* Sappey (*Journal de l'Anat. et de la Physiologie*, vol. i, 1868, p. 47) has demonstrated their existence in the sheath of the optic nerve, and the fact mentioned in the text admits of no other explanation than the existence of similar nerves in all nerve sheaths, which has, indeed, been demonstrated by Horsley (*Proc. Roy. Med. and Chir. Soc., 1884*, and Appendix to Marshall's "Bradshaw Lecture," 1887).

† An ingenious hypothesis has been formulated by Vanlair, which may be mentioned. According to Pierret there is a relation between the length of a nerve fibre and the size of its cell. The sheath nerves will vary in length according to the distance from the centre at which they terminate. If the cells discharge in the order of their size, beginning with the smallest, the sensation will seem to dart centrifugally. For the apparently centripetal direction, a still more complex hypothesis has been suggested. Most nerves contain recurrent fibres which proceed from adjacent nerves (Magendie, Arloing and Tripier). These recurrent fibres ascend the branches and trunk, but all cease before the foramina of exit are reached. If these end in the nerve sheath, the same hypothesis applied to the recurrent fibres will serve to explain the centripetal darting; the longest fibres, which end nearest the centre, will have the largest cells, and discharge last. Unfortunately, we do not know that any of the recurrent fibres end in the nerve sheath, and if they do, it is probable that their central connection is the same as that of the direct fibres. It seems more probable that the arrangement of the cells in the centre depends on the distribution of the fibres in the sheath, and that this arrangement, and not the size of the cells, determines the order of the discharge, which may traverse the centre in opposite directions in different cases, just as in one epileptic an aura may pass down, and in another up, the arm.

much speculation.* Their localization to the places at which nerves emerge from deeper structures, or divide, suggests their dependence on mechanical causes. Accidental pressure, and traction in movement, will have most influence on the nerves at such places, and cause there a greater degree of stimulation of the nervi nervorum (Vanlair). Many phenomena of neuralgia suggest, moreover, that a neuralgia which is at first purely central may not remain so. We have seen that the pain often causes secondary vascular disturbance in the territory of the nerve. It is most unlikely that such disturbance, conspicuous in the skin, is confined to the surface. It probably involves, also, the deeper structures, and especially the nerve sheaths, in which the pain is especially localized. Such secondary vascular disturbance, and the tissue changes to which it ultimately leads, must constitute a source of irritation of the nervi nervorum, and, in a truly "vicious circle," must intensify the malady, which, at first central, may be thus, at last, peripheral also. It is probable that this mechanism takes an important share in the production of the tender points, and is also the cause of the intractability of some neuralgias.

Not only is it probable that peripheral disturbance takes part in the pathogenesis of central neuralgia, but it is certain that central disturbance is concerned in some neuralgias of peripheral origin. A traumatic cause, an injury to a nerve branch, may induce pain far wider in area than the distribution of the injured branch, or even of the nerve from which it comes. In some cases, again, the pain is felt not in the area of the nerve injured, but in that of some other nerve (reflex neuralgia). An interval usually elapses after the injury before the pain is felt. Lastly, in some traumatic cases, division of the nerve does not cure the neuralgia. These facts can only be explained by assuming that the chief cause of the pain is a morbid state of the central cells, excited by, but to some extent independent of, the peripheral lesion. Doubtless, in all neuralgias of "symptomatic" character, the symptoms depend, in varying degree, on an induced central disturbance.

VARIOUS FORMS.—Cases of neuralgia differ much according to their situation, character and cause, and hence it is necessary to describe in some detail the varieties of the disease. According to situation, we have to consider separately those which occupy the head, neck, arm, trunk and leg. According to character, we have "epileptiform neuralgia," and "reflex" or "sympathetic neuralgia," while of those which depend on special causes the most important are the traumatic, herpetic, anæmic, malarial, syphilitic and diabetic forms.

It is important to remember that neuralgia is often not confined to a single nerve. Those who are liable to the affection in a high degree sometimes suffer from neuralgia in many situations, simultaneously or in succession.

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* The hypothesis (of Cartaz and others) which connects these tender points with the distribution of the recurrent nerves, rests on too many unprovable assumptions to deserve detailed description.

VARIETIES DEPENDING ON SITUATION.

Neuralgia of the Fifth Nerve. Trifacial or Trigeminal Neuralgia; Tic Douloureux; Prosopalgia.—Under these various designations the most common form of neuralgia has been described. Neuralgia of the fifth is probably more frequent than all the other varieties together, and it presents, in most typical form, the characteristic symptoms of the disease. Nor is this surprising when we consider that the fifth is incomparably the most important nerve of common sensibility in the body.

The causes of this form are all those that have been described in the section on etiology; indeed, the general history of neuralgia is, to a large extent, based on the symptoms of this variety. It is equally common on the two sides. The seat of the pain may be in any of the three divisions of the nerve; and it more commonly occupies one or two of the divisions than all three. The tender points are often well marked, and in them the pain has its chief intensity.

Neuralgia of the *first division* is felt chiefly in the supra-orbital branch, and hence is often called *supra-orbital neuralgia*. The frequency with which it was formerly due to malaria has left for it the popular name of "brow ague," although this cause is now rarely operative in this country. The pain radiates from the supra-orbital notch over the anterior half of the head, and is often felt in the eyelids and even in the eye, and in the side of the nose. The most important tender points are the *supra-orbital* just above the notch or foramen of that name, a *palpebral*, in the outer part of the upper eyelid, a *nasal* at the emergence of the nasal branch at the lower edge of this bone, and sometimes an *ocular*, within the eyeball. Pain felt just above the eyebrows is sometimes due to a morbid state of the frontal sinuses, but pain from this cause is generally double, and is often secondary to coryza. The lining membrane of the sinus is supplied by the fifth nerve, and it has been conjectured that the pain occurs when the small opening of the sinus into the nasal cavity becomes closed. Seeligmüller thinks that this is the cause of the pain even in malarial cases. But the nerves of the sinus (or their centres) seem to be particularly obnoxious to certain influences, as is shown by the peculiar pain, evidently referred to these sinuses, which many persons experience after eating ices. We cannot, therefore, conclude that because the pain occupies this situation, it is necessarily due to a local cause. The supra-orbital region is a not uncommon seat of pain that is apparently neuralgic. This does not follow the course of the nerves, but it may be felt sometimes over one eye, sometimes over both.

Ocular Neuralgia.—The eyeball is an occasional seat of neuralgic pain, often of considerable severity. This is sometimes associated with some error of refraction, especially hypermetropia, but occurs also independently of any abnormality of the eye itself. Either one or both eyes may be affected. The pain may occur spontaneously, or may be brought on by use of the eyes; it is not often accompanied by photophobia. When severe, there is occasionally dimness of sight, apparently of inhibitory origin, and the amblyopia may be accompanied by a peripheral restriction of the field of vision. Ocular neuralgia may exist alone, or be associated with pain in adjacent parts, and sometimes with pain that extends far beyond the limits of the fifth nerve. Bilateral pain sometimes passes from the eyes over or through the head to the occipital region, and even down the neck. Anæmic girls often complain of a peculiar dragging pain at the back of the eyes, increased by their use. Ocular pain is often associated with rheumatism; the subjects of rheumatic iritis are often liable to pain in the eyeballs, which seems to be neuralgic in character.

In neuralgia of the *second division, infra-orbital neuralgia,* the pain occupies the area between the orbit and the mouth and extends over a great part of the cheek, and to the ala nasi. The chief foci of pain and tender points are an *infra-orbital,* at the emergence of the nerve beneath the orbit; a *nasal,* at the side of the nose; a *malar* over the most prominent part of that bone, and a *gingival line* below that bone, along the line of the gums of the upper jaw; very rarely there is a point in the palate, or in the upper lip. The most acute pain is often felt only in one portion of the nerve, as, for instance, the side of the nose; but it usually radiates, in a slighter degree, over a wider extent.

When the *third division* is affected, the pain often extends over a large area, occupying the parietal eminence and the temple, the ear, the lower jaw and the tongue. The chief tender points are the *inferior dental,* at the foramen of that name; the *temporal,* in the posterior part of the temple on the auriculo-temporal branch; it may be a little lower down, just above the zygoma in front of the ear, and is a very common focus. The *parietal,* over the parietal eminence, is common to this and to occipital neuralgias. Sometimes there is a focus of pain in the tongue. Separate portions of this branch are sometimes affected alone, especially the inferior dental and the auriculo-temporal. A boring pain limited to the temporal point is especially common. Most intense neuralgia is sometimes confined to the lingual branch. Occasionally, a tender point exists in the cervical spines, at the first two or at the fifth (Armaingaud); its exact cause is obscure.

The pain in trigeminal neuralgia is often peculiarly intense and presents every variety. It may radiate from one part of the fifth nerve to the next, and even to other nerve regions. Thus, in one case of violent neuralgia of the second division of the fifth, the pain often radiated to the occipital region and sometimes to the shoulder on that side. The effect of cold and contact in exciting the pain are well marked, and it is often increased by movement of the face or jaw, so that, in severe cases, mastication may be impossible, and it may be difficult to give sufficient food. If the ear is the seat of pain, either alone or with other parts, the attack may be accompanied or followed by auditory hyperæsthesia. When the pain is very acute and sudden, reflex muscular spasm may occur in the face (the "tic convulsif" of the French). Paralytic phenomena are rare, but transient paralysis of the third nerve has been observed to follow each paroxysm of pain in the supra-orbital branch. Sometimes paroxysms of severe pain in the fifth nerve are accompanied by subjective flashes of light, especially when the eyeballs are the seat of pain. The vasomotor disturbance already described is frequently seen, flushing, sweating, permanent dilatation of the vessels (often conspicuous in the eye), salivation, increased secretion of mucus in the nose, lachrymation. Trophic disturbances occasionally occur; acute, as erythema; or chronic, as thickening of the periosteum, loss of hair, or local grayness. Even unilateral atrophy of the face has been met with (Boisson). It is probable that in many of these cases the pain was the result of actual changes in the nerves. The course of the fifth in the base of the skull, through the membranes and bony foramina, exposes it to damage from many morbid processes, and causes its fibres to suffer when there is any inflammatory swelling of the sheath.

Occasionally, migratory pains are felt in various parts of the scalp, sometimes on one side, sometimes on the other, without any distinct relation to the nerve trunks. There may be tenderness of the skin during and after the paroxysms of pain. This form is sometimes more closely allied to rheumatism than ordinary neuralgia is. Rarely there is neuralgic pain over the whole scalp at the same time, so that, as one patient expressed it, there is "a cap of pain on the head." Pain at the vertex is a common form of headache, sometimes closely allied to neuralgia, and it may alternate with characteristic neuralgic pains in other situations.

Cervico-Occipital Neuralgia.—The pain is felt in the region of the neck supplied by the first four cervical nerves and in the posterior portion of the head, chiefly along the course of the great occipital nerve. Thus, the pain may extend over the greater part of the neck as well as over the head, as far forward as the parietal eminence and the ear. It is occasionally confined to the posterior branches, extending over the back of the neck and occiput. The most important tender points are (1) about midway between the mastoid process and the spine, at the point at which the great occipital nerve becomes superficial, (2) over the branches of the cervical plexus between the sterno-mastoid and trapezius, and (3) just above the parietal eminence, the focus common to occipital and trigeminal neuralgia. Fusion of these two forms of neuralgia occurs, so to speak, not only above but below, where the distribution of the cervical nerves blends with that of the third division of the fifth over the lower jaw. A primary cervical neuralgia may extend into this region of the fifth; doubtless the centres blend as does the distribution. It is probable that cervico-occipital neuralgia is more often bilateral than any other form, especially when confined to the occipital region. I have known most severe bilateral neuralgia to be limited to the anterior cervical region, from the jaw to the upper part of the thorax on each side. The pain in cervico-occipital neuralgia is rarely intermitting; there is more or less dull, constant pain with occasional exacerbations, less violent than in the trigeminal form. The scalp may become extremely tender, so that during the pain the patient cannot bear the hairs to be touched. This form of neuralgia is not common, and Anstie believes that it occurs especially in those who have suffered from other forms.

Cervico-brachial and brachial neuralgia includes those forms in which the pain is referred to the region supplied by the four lower cervical and the first dorsal nerves. The pain may be felt in the lower and posterior part of the neck or any part of the arm and hand, but is commonly most intense in the axilla, at the brachial plexus, and along the course of the ulnar nerve. The region of the last is a very frequent seat, but sometimes the pain is referred to other nerves. It is commonly increased by movement and may render the arm almost useless. It is often excited by writing, and this may give rise to an erroneous impression that it is connected with the act of writing. Some severe forms of brachial neuralgia, indeed, have their origin in a sensory occupation neurosis, but this variety has been separately described (p. 1067). The most common tender points in brachial neuralgia are the *axillary;* the *circumflex*, at the posterior border of the deltoid; a *superior ulnar*, behind the elbow, and an *inferior ulnar*, in front of the wrist. The latter is the most frequent of all the brachial foci. Others occasionally met with are the *vertebral*, by the side of the lower cervical spines; a *scapular*, at the inferior angle of that bone; an *external humeral*, on the outer side of the arm three inches above the condyle, over the musculo-spiral nerve; and a *radial*, in the lower and outer part of the forearm. There is usually some constant pain in addition to the acute paroxysms. Occasionally it may radiate to the side of the chest, and then, if on the left side, may simulate angina pectoris. The pain is almost always intensified by movement. Trophic disturbance in the arm is very rare in cases of true neuralgia, and, indeed, probably always indicates neuritis. When the pain starts from the fingers, it may begin with some sensation other than pain, such as tingling, which changes to pain as it passes up the arm. In some patients with brachial neuralgia the arm is peculiarly liable to be the seat of tingling at night. Brachial neuralgia is not often due to diathetic causes, with the exception of rheumatism, with which it is often associated even when there is no suspicion of neuritis. On the other hand, it is more frequently than any other the result of injury. Probably many cases of supposed neuralgia are really cases of neuritis of the brachial plexus, which may arise by migratory inflammation. But true neuralgia of the arm, widely spread, may be set up by a slight injury, as a blow, which does not apparently cause neuritis. Brachial neuralgia is occasionally associated with neuralgia of the fifth, and this when there is no connecting pain in the neck.

Trunk Neuralgia.—Of the neuralgias of the trunk, we have, first, the *dorso-intercostal* forms, which occupy the intercostal nerves, from the third to the ninth, characterized by pain coursing along the intercostal spaces or parts of them. It is sometimes bilateral and symmetrical. There is usually a constant, dull pain, with acute stabbing exacerbations, but sometimes the continuous pain exists alone, or the sharper pains are excited only by movement, respiratory or other. There are foci of intensity and tender points at the emergence of the three branches of the intercostal nerve—besides the vertebræ, near the middle line in front, and midway between these two points in the mid-axillary line. Intercostal neuralgia is most frequently due to cold, or to injury, such as a contusion. It is sometimes extremely obstinate and of long duration.

The thoracic wall is also the seat of more trifling neuralgic pains; one of these is pleurodynia, which differs from true intercostal neuralgia in being usually localized at one spot not corresponding to the course or exit of the intercostal nerves. It appears to be a true neuralgia, distinguished from myalgia by the fact that it is local, very acute in character, and is excited by expansion of the thorax rather than by lateral movements of the trunk. The theory that it is a neuralgia of the pleural nerves has much probability. Another common neuralgic pain is the infra-mammary pain of anæmic women. Very limited in position, it is more or less constant, and is rarely increased by respiration to such an extent as to interfere with the free expansion of the thorax. The relation between intercostal neuralgia and pulmonary trouble is a difficult subject, on which satisfactory facts are difficult to obtain, and few observers have ventured to corroborate the statement of Woillez, that acute intercostal neuralgia is always accompanied by a secondary congestion of the lung. It is more probable that when this association exists the true relation is the reverse. The intercostal nerves are frequently the seat of herpetic neuralgia.

The neuralgias that occupy the lower half of the trunk have been grouped as *lumbo-abdominal.* The pain has a course similar to that of the intercostal form. Foci of pain and tender points are found at the back, beside the vertebræ, over the posterior branches; at the middle of the iliac crest (*iliac point*); at the lower part of the rectus (*hypogastric point*), while sometimes there is, in the male a *scrotal,* and in the female a *labial point.* These pains are often bilateral, and may change their position from time to time. They are generally acute pains, but now and then have a constricting character, like the girdle pain of organic disease, but distinguished from them by their irritability. Lumbo-abdominal neuralgias seem to be sometimes secondary to disease of the pelvic organs, especially in the female. Neuralgia in the penis sometimes results from masturbation. It may also be due to lithæmia, and from this cause I have known it to be most severe and long continued.

The *spinal column* is a very common seat of neuralgic pain, especially in weakly women and after concussion of the spine. It constitutes one of the most troublesome of the many pains of hysteria, and one of the most enduring consequences of railway accidents. The pain is in most cases felt through a considerable vertical extent of the spine, and is specially intense in certain spots, commonly in more than one. The dorsal region is the most common seat, next the lower cervical, and least frequently the lumbar region. Sometimes the pain is localized on one side of the spine, close to it. The pain felt in the spine in cases of gastric ulcer seems to be a sort of reflex neuralgia. Often the pain seems to pass up to the back of the head. Spinal neuralgia may be associated with a similar pain in some other part of the trunk, shoulder, arm, or leg. It is usually accompanied by considerable tenderness, and is increased by fatigue, by use of the legs, by long sitting or standing, and also by vibration, such as the movement of a carriage. The latter point is often of considerable diagnostic importance, for it is far more marked in neuralgia than in spinal disease causing pain. On the other hand, the pain is not increased by slight movement, as is the pain of growths and caries. The pain is seldom distinctly paroxysmal, but as already mentioned in one case, paroxysms of pain in the cervical spine and vertex were most intense, and each was accompanied by opisthotonos. It is uncertain in what structure this spinal neuralgia is produced. It is often associated with neuralgia elsewhere, and also with rheumatism of the fibrous tissues, so that some cases seem to be of the nature of rheumatic neuralgia. It has been thought that the membranes are the seat of the pain, but there is no real evidence for or against the theory.

Another very common seat of neuralgia is the *sacral region*, no doubt in consequence of the plexus of nerves that lies between the bone and the skin. Pains of pelvic origin are often referred to this region, as those of parturition show. Occasionally the pain is felt chiefly about the coccyx—coccydynia it has been termed. It must be remembered that the fibrous tissues over the sacrum are sometimes the seat of acute rheumatism, such as higher up gives rise to lumbago.

Neuralgia of the Leg.—In the lower limb, neuralgia is rare in the region supplied from the lumbar plexus, although a *crural form*, in which the pain occupies chiefly the front of the thigh, is occasionally met with. In most instances pain in this region is of secondary origin, due to a lesion of the lumbar plexus, as, for instance, from the pressure of an abdominal tumor, or is due to the extension of neuritis from the sciatic up the lumbo-sacral cord.

The majority of neuralgic pains in the leg are in the region of the sciatic nerve, and are grouped under the designation of sciatica. The pain occupies especially the course of the nerve, but tender foci are met with in certain spots; *lumbar*, near the spine, just above the sacrum; *sacro-iliac*, at the articulation of the same name; a *gluteal* opposite the middle of the lower border of the gluteus; a series of spots varying in exact position, along the course of the nerve in the posterior aspect of the thigh; a *peroneal* behind the head of the fibula; and a *malleolar* behind the lower extremity of the fibula, and an *external plantar* at the outer border of the foot. It is, however, certain that sciatica is seldom a true neuralgia. Almost all severe cases are due to inflammation of the nerve trunk; the evidence of this has been stated in the account of the disease on page 103 *et. seq.*

Lastly, it should be mentioned that in rare cases neuralgic pains are felt almost everywhere, in the limbs, trunk, and head, and apparently constitute a sort of universal neuralgia. The few cases I have seen have been in adult men, and associated with hypochondriasis.

VARIETIES DEPENDING ON CHARACTER.

Epileptiform Neuralgia.—The term is applied (according to the example of Trousseau) to an intractable form, in which each attack of pain comes on very suddenly, with intense severity, and lasts usually less than a minute, sometimes only a few seconds, rarely for two or three minutes. The frequency with which the attacks recur vary in different cases; the daily number may be one or hundreds. The pain is most intense, so that the patient stamps about the room in agony, or tries to get relief by violent rubbing or by pressure. Trousseau relates the case of a lady whose malar bone was atrophied in consequence of the pressure, repeated every few minutes for years. The sufferers from this terrible form are usually in the second half of life. The pain is invariably in the region of the fifth nerve, sometimes in the whole, sometimes in part only, but rarely confined to a single branch. Convulsive spasm in the face may accompany the pain (convulsive epileptiform neuralgia).

Reflex or sympathetic neuralgias are those in which the pain is felt in another nerve region than that in which its cause exists. The radiation of neuralgic pain must, as already stated, be ascribed to the extension of the central discharge to adjacent centres, but the term "reflex neuralgia" is not applied to these cases, but to those either in which the pain exists only at a distance from its cause, or in which the distant neuralgia has no sensory continuity with the primary pain. The pain of a carious tooth may be associated with a distant and apparently disconnected pain in some other part of the fifth nerve, or such disease may cause only distant neuralgia, and no local pain. The proneness of the fifth nerve to be affected in neuralgia renders it a common seat of the reflex form. For instance, neuralgia limited to the fifth has been produced by injury to the ulnar nerve and to the occipital nerve (Anstie). Nevertheless, the converse relation is sometimes observed; carious teeth are said occasionally to cause neuralgia in other parts, as, for instance, in the cervico-brachial region (Salter). When no local pain is felt, we must assume that the centre to which the afferent impressions directly come is not thus excitable to painful activity, or even so as to influence consciousness, but that it is in connection with another centre which, by natural or acquired susceptibility, is disposed to excessive action. Curious cases are on record in which the act of micturition invariably caused a pain in the region of the arm supplied by the ulnar nerve.*

The cause and seat of reflex neuralgia may be most varied. When due to causes acting on the cerebro-spinal nerves, the pain is usually felt on the same side, and rarely far distant. A most important class of reflex pains are those which are produced by disease of the internal viscera, such as the spinal pain in ulcer of the stomach, the sacral pain in uterine disease, the scapular pain in affections of the liver, the pain in the testicle in renal colic, and pain in the front of the thigh and spine in gonorrhœal orchitis (Mauriac). Many of these are rather reflex pains than neuralgias properly so called, but they occasionally persist when their cause is removed, or assume disproportionate intensity. They are of very great practical importance on account of the readiness with which their cause may escape attention.

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* Several such cases are given by Vanlair ("Les Nevralgies" p. 330); one of them comes from a curious source—the autobiography of Clarendon, Chancellor to Charles II. See note on p. 1136.

Traumatic neuralgias are those that are excited by injury to nerves—by contusion, and by punctured, lacerated, or incised wounds. In some instances the pain is the result of a neuritis, set up by the injury, which may ascend the nerves and pass, at a junction, to other trunks. But neuralgia may also be set up when there is no more than the local and transient inflammation at the spot injured. Such neuralgia may follow nerve lesions in any part of the body, but are especially frequent in the arm, the nerves of which are much exposed to injury. In the days of venesection, a common form of neuralgia resulted from injury to a cutaneous nerve in the operation. It may follow wounds of both large and small nerves, and by some observers has been thought to be more frequently due to the latter, but it must be remembered that small nerves are more often wounded than larger trunks. Anstie believes that neuralgia is more common from partial injuries than when the nerves are completely divided. It is a frequent consequence of gunshot injuries of nerves. In some cases the disease is apparently due to the implication of nerves in a cicatrix, or to the morbid enlargement of the nerve ends after amputation, which goes by the name of "bulbous nerves."

The pain may commence a few hours or days after the injury, but more frequently not until after an interval of some weeks or months. It generally begins in the point injured, but is rarely limited to this, or even to the part supplied by the wounded branch. It usually radiates to adjacent regions, and sometimes to parts supplied by other nerve trunks. For instance, a woman received a blow on the thorax, probably contusing an intercostal nerve. The early pain of the injury ceased, but two months later neuralgic pains commenced at the spot and gradually spread over the whole region supplied by the cervical and brachial plexus (Ollivier). The pain may be less intense at the seat of the injury than it is elsewhere, and it may even be absent at the seat of injury, and then is of the purely reflex variety, instances of which have been already mentioned. These facts, and also the circumstance that the pain is not always influenced by excision of the injured nerve, show clearly that it is to a large extent of central origin, the expression of a disturbance which, though excited by the nerve injury, is to a considerable extent independent of it, and doubtless due, not only to the excitant, but also to a predisposition, such as is concerned in the production of other neuralgias. The same fact is seen even more clearly in cases in which some injury leaves a slight local weakness or disability, and years afterward the part may become the seat of neuralgic pain, under the influence of a constitutional tendency.

The pain is usually intermitting in character, but often most intense in degree and causes profound depression of the general system. It may excite muscular spasm, and, especially at the onset soon after the injury, other reflex symptoms, such as vomiting. Vasomotor disturbance occasionally ensues; trophic changes in the skin and joints have been described, but it is very doubtful whether nutrition suffers except in consequence of considerable secondary neuritis. These lesions are considered more fully in the chapter on neuritis, on page 72 *et seq.* The course of traumatic neuralgia is often tedious, sometimes most prolonged, and it has been known to last to the end of life, in spite of every medical and surgical measure that could be devised for its relief. When it ceases, it is prone to recur under the influence of general depressing influences.

Occupation Neuralgias.—These are pains, more or less neuralgic in character, brought on by some habitual act. They are the sensory forms of occupation neuroses, in the account of which they have been fully described.

Herpetic Neuralgia.—Herpes zoster, whatever its seat, is usually accompanied by pain of a neuralgic character. The explanation of this is found in the conclusive evidence that the eruption is the effect of nerve irritation, probably always inflammatory in character. It always corresponds in area to the distribution of certain nerves, and, post-mortem, inflammation has been found in the nerve (Haight) and in the ganglia of the posterior roots (Bärensprung, Charcot, etc.). The neuralgic pain is thus "symptomatic," the result of organic changes in the nerves. Sometimes herpes occurs in the area of a nerve which has long been the seat of neuralgic pain, and hence is regarded as an occasional trophic effect of neuralgia, but it is an extremely rare consequence, and probably only occurs in cases in which the pain is the expression of organic changes in the nerve, and is not a consequence of simple idiopathic neuralgia.

In cases of ordinary zoster, pain may occur before or after the eruption. The initial pain precedes the appearance of the eruption for a few hours or days, is usually moderate in severity, and commonly (but not always) subsides as the cutaneous lesion is developed. Sometimes this pre-herpetic pain is absent, especially in the young, although even then there is usually an initial sensation of tingling in the part. The pain that succeeds herpes is more constant. It comes on usually during the decline of the eruption, and its occurrence bears no relation to the seat of the herpes. The most common situations for neuralgia are those in which herpes is most common: the side of the trunk, the forehead, neck, and leg. The pain has the distribution of the eruption, corresponding to the nerve or nerves affected. It varies in degree, but is usually acute, lancinating, and for a time severe, and is accompanied by great tenderness of the skin. The same tender points are met with as in ordinary neuralgia in the same area. There is an important relation between the age of the patient and the severity and duration of the pain. In old persons it constitutes one of the most severe and persistent forms of neuralgia, often continuing for months and even years before, at last, it slowly lessens. Occasionally, it persists in unmitigated intensity to the end of life. Sir William Jenner, in his lectures, was accustomed to illustrate the obstinate persistence of this pain in the old, by the instance of a man who, before the days of anæsthetics, endured the excision of the skin to which the pain was referred, in the hope of relief, but found none, and then, unable to bear the continuous agony, he shot himself.

Hysterical Neuralgias.—Neuralgic pains are common in hysteria, but a distinction must be drawn between those which are merely associated with, and those which are due to, the general neurosis. Of associated forms, every variety of true neuralgia may be met with, due to the neuropathic disposition, which is also the cause of the hysteria, and they present the characteristic distribution, tender points, etc., of the ordinary form. Anæmic neuralgias are also common in hysterical patients, in whom the nerve disturbance is often due to poorness of blood. Of the neuralgic pains that are due to hysteria, some are in the cerebro-spinal system, some in that of the sympathetic. Certain local pains in the head are very common, but these differ from ordinary neuralgia in being confined to one small spot, instead of following the course of nerves, and the local pain has a sharp stabbing or boring charactar, as if a nail were being driven in, and hence has received the name of "clavus hystericus." It is important to remember that a similar pain may be met with apart from hysteria, especially in anæmic persons. Pains in the spine are also extremely common, sometimes very local, and of various characters. Other pains referred to the trunk are usually associated with local tenderness, in the so-called "hysterogenic points." The most important neuralgias of hysteria are those of the viscera, ovarialgia (not necessarily in the ovary itself), and gastralgia, being the most frequent. Pains in the joints and muscles are also common. The visceral neuralgias, and those associated with parietal tenderness, are usually persistent, but the local boring pains, and those in the joints and muscles, are often fugacious and migratory, and this constitutes a diagnostic point of considerable importance.

Rheumatic Neuralgia.—In a loose way, all neuralgias produced by cold are sometimes styled "rheumatic," but the mere causal relation scarcely warrants the epithet. The peculiar affection termed "muscular rheumatism" is also sometimes called a rheumatic neuralgia, but this is to extend the use of the term in a manner that is scarcely justified or needed. Pain that occurs only on movement should never be called neuralgia. Acute articular rheumatism is rarely associated with true neuralgia, but some forms of spontaneous pain are frequently produced by cold in those who present what is termed the "rheumatic diathesis," who perspire easily, are liable to catarrh, and whose urine readily becomes loaded with lithates after a chill. Such pains may correspond to a certain nerve, or may occupy some part of a limb, without any definite relation to nerves, and are often migratory. Their exact pathology is uncertain. Some other facts regarding this variety have been mentioned on p. 1133.

Gouty Neuralgia.—The subjects of gout not unfrequently suffer from nerve pains, apparently idiopathic in character, coming and going, and sometimes very severe. The pain may disappear when an attack of acute gout is developed. The fifth nerve, the intercostals, and the sciatic are those most frequently affected. Severe sciatica sometimes occurs in the gouty but is certainly due to neuritis. One visceral neuralgia is also sometimes due to gout, gastralgia.

Diabetic Neuralgia.—Patients with diabetes may suffer from neuralgic pains that have no special characters, but Worms has called attention to the symmetry of the pains, which occupy the same nerve on both sides, as a characteristic of diabetic neuralgia. The pain has hitherto been observed chiefly in the third division of the fifth nerves and the sciatics. It must be remembered, however, that ordinary neuralgia is occasionally symmetrical. The nerve pains met with in diabetes are often severe and obstinate until the cause is removed by dietetic treatment. They have been observed to increase and decrease with the amount of sugar in the urine. Ziemssen has suggested that they may be sometimes the result of a peripheral neuritis analogous to that which is met with in alcoholism. While there is some evidence that such neuritis may occur in diabetes, it seems improbable that it is the cause of the pains commonly met with.

Anæmic Neuralgia.—Anæmia is one of the most powerful causes of neuralgia in all its forms, but certain varieties are more frequent than others in this condition, especially in young women. One of these is situated in the fifth nerve, either in the first division or in the auriculo-temporal branch. It is generally intermittent and is increased by movement, while it is lessened by the recumbent posture. Another, still more frequent and more continuous, is that which is felt in the thorax, in the fifth and sixth interspaces on the left side, the well-known infra-mammary pain. Gastralgia is also common apart from ulcer. Headaches that have no true neuralgic character are also very common.

Neuralgia of the cerebro-spinal nerves sometimes occurs in *lead poisoning*, but it is uncertain whether it is the result of the toxic influence or of the anæmia which this causes. The knowledge of their cause, however, is very important. According to Briquet, lead colic is in part a neuralgia of the abdominal wall, but the evidence of this is scarcely satisfactory.

Malarial neuralgias are not very common even where ague is frequent. They present nothing characteristic in seat, although the supra-orbital and intercostal forms are the most frequent; nor is there anything special in the character of the pain. Their chief feature is regular periodicity, the intervals between the attacks being from one to four days. The periodicity is less characteristic when the attacks are diurnal than when three or four days intervene. Occasionally the paroxysms are attended with slight symptoms of an ague fit, a trifling cold and hot stage (Anstie). They must not be confounded with the vasomotor phenomena met with in cases of the ordinary form. It is very doubtful whether the neuralgia is, in most cases, a direct effect of the malarial poison, in the sense in which ague is. It is probably an indirect effect, the result of the anæmic and depressed state of the nervous system induced by malarial influences, even in those who do not suffer from intermittent fever. Neuralgia, apparently due to malarial causes, does not always yield to quinine, even when given in the most approved manner. It is probable that the exact periodicity of many malarial neuralgias is the result of the state of the nervous system produced by the poison, and it does not prove the neuralgia to be truly malarial. In some cases of supra-orbital neuralgia with exact periodicity coming from malarial districts (recorded by Seeligmüller), quinine failed entirely while other treatment was quickly successful.

Syphilitic Neuralgia.—The pains of syphilis constitute a prominent symptom of that disease, but, for the most part, have no correspondence with nerve distribution, and can therefore be scarcely regarded as neuralgic. Symptomatic neuralgic pain occurs in many syphilitic affections of the nervous system which cause irritation of the nerves or their roots, in chronic meningitis, neuritis, and pressure from growths. One of the most severe and obstinate cases of pain in the region of the fifth nerve I have seen, was due to chronic spyhilitic meningitis at the origin of the nerve. In such cases the nature of the lesion is usually clear from the evidence of structural damage to the nerve fibres. In the case just mentioned, for instance, there was anæsthesia and paralysis of the masseter. Whether idiopathic neuralgia results from the influence of the syphilitic poison is uncertain. Fournier believes that such neuralgia is common during the secondary stage, but very few conclusive cases have been recorded. It must be remembered that in the early stage of neuritis, etc., pain may be the only symptom, and, on the other hand, the anæmia which results from syphilis may be the real cause of the neuralgia. Anstie believed that syphilis does not cause true neuralgia, although it may recall a neuralgia which had long ceased. A peculiarly distressing post-sternal pain, apparently neuralgic in character, has been occasionally observed in constitutional syphilis (Eccheverria, Buzzard).

Degenerative Neuralgia.—In advanced life, and sometimes before the senile period is reached, neuralgia is occasionally met with, of extreme obstinacy, and associated with other signs of degeneration of the central nervous system, such as failure of memory or persistent mental depression. The neuralgia is apparently one effect of a degenerative tendency. The affection has all the characters of a central neuralgia. The fifth nerve is by far the most common seat, but the pain sometimes occurs in other situations.

Diagnosis.—The diagnosis of neuralgia rests on the unilateral situation of the pain, on its correspondence to the distribution of certain nerves, its intermitting or remitting character (*i. e.*, the occurrence of paroxysmal exacerbations), on the fact that the patient has suffered from similar pains elsewhere, on the variations in the seat of the pain, and on the absence of any indications of actual damage to the nerve fibres. The variability is a symptom of great importance. If the pain shifts about, now in one spot and now in another, it is not likely to be due to an organic cause. For instance, a man with fronto-occipital neuralgia had foci of pain in the forehead, temple and occiput, but he never had pain at the same time at more than one of these places. The last of the above indications, however, is the most important element in the distinction of neuralgia from the similar pains which result from organic disease of the nerves due to external pressure or neuritis. It is in the case of the fixed neuralgias (as distinguished from the migratory forms) that the distinction is of chief importance. The diagnosis is more difficult in the case of neuritis than of external pressure (tumor, etc.), because, in the latter case, the cause of the pressure usually produces other symptoms, and the effect of the pressure is progressive, so that gradually increasing signs of a structural lesion are added to the pain. But from neuritis the distinction may be much less easy. The difficulty is the greater, the slighter the degree of neuritis. Severe inflammation causes severe constant pain, at first more intense at its seat than in the distribution of the nerve, because the inflammation is most intense in the nerve sheath, and the sheath nerves suffer first. In severe forms, however, the proper fibres of the nerve are soon implicated to a degree that interferes with their conducting functions, so as to cause at first persistent hyperæsthesia, and then areas of anæsthesia, while if the nerve is "mixed," the muscles supplied become weak, flabby, and rapidly waste, with changes in electrical irritability. In slight cases, on the other hand, the sheath chiefly suffers; there may be no interference with conduction, and the pain resembles neuralgia more closely, although, as a rule, it is more continuous than in true neuralgia. The diagnosis is also difficult when the seat of the neuritis is such as to render the nerve inaccessible to direct examination. If it can be reached, there will be found from the first local tenderness of the nerve. In idiopathic neuralgia tenderness of the trunk in the intervals is only developed after the neuralgia has existed for some weeks. Moreover, in neuritis distinct swelling of the nerve can sometimes be felt. Local tenderness is thus chiefly of significance in the early stage of the affection, or when it occupies a considerable area of the nerve trunk, and is not confined to certain points.

If, therefore, the pain is migratory, if it is completely intermittent, especially if the intermissions are of long duration, if the attacks are induced by psychical influences, the suspicion of an organic cause will scarcely arise. If, on the other hand, there is continuous pain, rapidly developing to a considerable degree, organic disease should be suspected, and the suspicion will be confirmed if there is persistent alteration of sensibility, muscular wasting or altered irritability, or trophic changes in the skin. Great care is necessary, however, in testing the muscles, because a strong electrical stimulus may greatly increase the pain. The isolated faradaic shocks should be employed rather than the current, because they cause much less pain, and because they detect most readily the earliest change produced by neuritis, a slight *increase* of irritability; an altered reaction to voltaism will also often be found. Early tenderness of the nerve, not merely during but between the paroxysms, extending some distance, and distinct swelling of the nerve, indicate neuritis. The absence of these does not exclude neuritis, because it may occupy an inaccessible portion of the nerve. If the symptoms of structural damage gradually increase and progress, and especially if they involve the whole region of the distribution of the nerve, compression may be suspected, and is confirmed by the discovery of any other symptoms indicating organic disease in the vicinity of the nerve trunk, such as, in the case of the fifth, damage to other nerves which arise or pass near it. It must be remembered that in some cases the differential diagnosis of slight inaccessible neuritis from neuralgia may be impossible, because, on the one hand, the irritation of neuritis may cause neuralgic changes in the nerve centre, and, on the other, a primary functional neuralgia may cause, by reflex vasomotor disturbance, secondary changes in the sheath, so that in each case a mixed affection, partly functional, partly organic, is the result; or, in current terms, a neuralgia which is at first either symptomatic or idiopathic, may ultimately be both.

In all forms of neuralgia, the circumstance that the patient has previously had attacks of pain in other situations is a very important help in diagnosis. It does not, of course, prove that a given pain is of functional origin, but it is proof of a tendency to such pain, which may justly be allowed considerable weight in the absence of signs of an organic lesion. This character is frequently of very great practical value, especially in cases of neuralgic pain of unusual seat. Another similar indication is the fact that the neuralgia replaces some other functional disease, as, for instance, migraine.

Special Diagnosis.—There are certain affections with which the several varieties of neuralgia are liable to be confounded. Almost any form of neuralgia may be simulated by the pains of tabes, and the possibility of this cause should always be thought of, especially in the case of migratory pain. In some situations the risk of error is greater than in others, and these will be especially mentioned.

In neuralgias in the branches which are distributed over the skull (fifth and great occipital) it is often doubtful whether the affection should be called headache or neuralgia. The distinction in some cases is one of name rather than of real difference. Either term is employed, according as the pain seems superficial or deeply seated. But the fifth nerve gives fibres to the dura mater, and it is probable that some of the deeply-seated unilateral headaches are really allied to neuralgia, although it is customary, on account of their special association, to describe them separately (see Headache). The fifth nerve is often damaged by organic intracranial disease, and nerve pain, thus produced, is sometimes mistaken for simple neuralgia. Besides the indications already described, organic disease often causes deeply-seated headache and other symptoms, especially in the functions of other nerves, optic neuritis, and paralysis or convulsion in the limbs. A history of recent syphilis increases the probability of organic disease, but does not render it certain (see p. 1154). The occurrence of herpes in the course of a supposed neuralgia is also probable evidence, here as elsewhere, of organic changes in the nerves. It must not be forgotten that neuralgic pains are sometimes the first symptom of morbid growths in the upper jaw and parotid region.

The diagnosis of the cervico-occipital form rarely presents any difficulty. The neuralgic pain usually courses along the nerve trunk, but it must be remembered that occipital neuralgia is occasionally bilateral. In caries of the cervical vertebræ pain may be an early symptom, but it scarcely ever spreads to the occiput, and the early interference with movement is usually characteristic.

The neuralgias of the arm have to be distinguished chiefly from neuritis, by the indications already mentioned. The diagnosis is seldom difficult, because neuritis usually causes trophic changes in the muscles and skin, and the nerves are accessible in a large part of their course.

A more difficult problem is presented by the trunk neuralgias, which have to be distinguished from disease of the internal viscera, and from organic spinal disease. Unilateral pain is a frequent accompaniment of disease of the organs in the thorax and abdomen, and it is hardly necessary to point out that, in every case, a careful examination should be made of the organs adjacent to the seat of pain. The greatest difficulty arises in the case of deeply-seated tumors which cause pain by nerve compression, especially when, as is sometimes the case, the pains are "reflected" and do not precisely correspond in position to their cause. Aneurism of the aorta, for example, often gives rise to such pain, especially when seated in the descending part; pain in the course of the nerves may be the only symptom until sudden death occurs. The pain is usually very severe, and often burning in character, and has not the same foci of intensity and tender points as in ordinary neuralgia. In severe unilateral (and even bilateral) pain, persistent in occurrence, whether uniform in seat or not, this cause should always be suspected. One of the most severe cases of neuralgic pain I have ever seen—darting, stabbing, burning, migratory pains in legs, abdomen, thorax, and left arm—was due to an undiscovered abdominal aneurism. Aneurism of the ascending part of the arch is now and then accompanied by pain passing to the arm, apparently of reflex character. Intercosto-humeral neuralgia may simulate angina pectoris, of which, indeed, such neuralgia may be said to form part. The severity of the paroxysms of angina, and the other distressing sensations which accompany the pain, usually renders the nature of the attacks sufficiently clear.

The parietal pains which accompany disease of the spinal cord rarely have the acute lancinating character of neuralgia; they are sensations of tightness or constriction, and the obtrusive symptoms in the legs indicate the nature of the disease. To this, however, morbid growths of the cord offer an exception, especially in the early stage. The pains may be very severe and of various character, but they are constant in seat, are increased by any movement, and, before long, indications of compression of the cord are added to them. The "lightning pains" of tabes are sometimes felt in the trunk, and have often been mistaken for neuralgia. They are distinguished by their changing seat and momentary duration, by the similar pains in the legs, and especially by diminished sensibility over extensive areas. In most cases there are some pains in the legs and the knee jerk is lost, but I have seen one case in which the changes were confined to the dorsal region of the cord and the knee jerk was normal, but in this, as in other cases of the kind, the light reflex of the iris was lost, a symptom of great indirect diagnostic importance. More constant in seat and neuralgic in character are the pains of pachymeningitis, which are caused by the compression of the nerve roots by the thickened membranes. They are distinguished from neuralgia by the wide extent of the pains, their bilateral situation, by the presence of areas of anæsthesia due to still greater damage to the nerves, by muscular wasting in the limbs, and by the symptoms of compression of the cord.

The terrible nerve pain which is sometimes produced by organic disease of the bone of the spinal column closely resembles neuralgia in its fixity of site and unilateral situation, but is distinguished by its peculiar dependence on movement of the trunk (see p. 187). Leg symptoms are usually soon associated with it. The lumbo-abdominal neuralgia may be confounded with renal colic. The distinction rests chiefly on the urinary symptoms that accompany the latter.

The sharp pains of tabes are more frequently felt in the legs than in the trunk, and these also are often mistaken for neuralgia, but the indications already mentioned suffice to distinguish them. The distinction of sciatic neuralgia from neuritis must be made by the indication already given (see also Sciatica, p. 103). Of still greater practical importance is the distinction of crural and sciatic neuralgia from the pains due to pressure on the lumbar and sacral plexus by tumors in the pelvis and abdomen. Such pains are felt along the course of the nerves, and are almost invariably thought at first to be neuralgic. Pains in the front of the thigh are rare except as the result of extension of neuritis from the sciatic nerve to the lumbar plexus, or as the result of pressure. In each case there is generally muscular wasting, which shows organic damage. The diagnosis between neuralgia in the front of the thigh and pain due to a lesion of the nerves, is also aided by the state of the knee jerk, which is generally early lost in organic disease, but remains intact in neuralgia. In every case of neuralgic pain in this situation, the abdomen should be carefully examined, and whenever pain in the sciatic is of a progressive character, and apparently due to mischief above the accessible part of the nerve, a rectal examination should be made, by which the source of pressure, if there be any, will readily be felt. It must also be remembered that pain felt in the knee may be a reflex effect of the irritation of the branches of the obturator nerve in hip-joint disease, and that obscure pains in the groin and thighs are sometimes the result of disease of the femur.*

The diagnosis of the spinal forms of neuralgia need not detain us long. In the reflex or sympathetic variety the cause of the pain will usually be discovered, if it is remembered that it may be outside the area in which pain is felt. It is rarely far distant; often in another branch of the same nerve. Pain in any part of the fifth nerve, for instance, may be due to the irritation of a carious tooth.

It is possible to confound epileptiform neuralgia with the form of true epilepsy in which the aura is a sudden pain. I have known, for example, slight attacks of minor epilepsy to be preceded by a most severe momentary pain in one fifth nerve. The occurrence of distinct loss of consciousness, and still more of convulsion, sufficiently indicate the epileptic nature of the case. A painful epileptic aura in a limb, followed by local convulsion, could only be mistaken for neuralgia with reflex spasm, in a patient who had never had a severe fit. But the course of such an aura is usually centripetal and deliberate, and the spasm has also a deliberate march.

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* In one case of intense stabbing pains in the groin, closely resembling neuralgia, the cause was ultimately found to be necrosis of the great trochanter.

PROGNOSIS.—The prognosis in neuralgia is influenced by the age of the patient, by the duration of the affection, its situation, severity, and cause. It is far better when the disease is due to any constitutional condition than when no general cause can be discovered, since, as a rule, the constitutional states that cause neuralgia are amenable to treatment, at any rate in such a degree as to influence the pain. In hysteria, however, some neuralgias are readily removed; others, especially when there is no anæmia, are most obstinate. The prognosis is better when there is no hereditary tendency than when this is marked. We do not yet know whether the prognosis is influenced by the fact that the heredity is general or special. The more severe the pain, and the longer the disease has lasted, the more difficult is its treatment. It is generally believed that neuralgias of the fifth nerve are more intractable than others. During the decline of life, neuralgias of all kinds, especially those of the degenerative form, are peculiarly obstinate, and in old age they sometimes cannot be relieved by any treatment whatever. However severe the pain may be, neuralgia involves little danger to life; as Buzzard has well said, "the disease does not seem, of itself, to shorten the duration of the life which it fills with suffering." Epileptiform neuralgia is, of all forms, the most obstinate; Trousseau, in his large experience, never knew a case to be cured. When neuralgia has once ceased it is extremely prone to recur, and this fact, which is true of all varieties, must be remembered in giving an opinion regarding the future.

TREATMENT.—The treatment of neuralgia consists, first, in the relief of the symptom, pain, and, secondly, in the removal of its cause, *i. e.* in the restoration of normal conditions of function in the sensory apparatus. The means by which these ends are to be secured are threefold—by hygiene, by drugs, given internally and applied externally, and by certain surgical operations on the nerves. It is evident that the treatment to be adopted must be influenced, to some extent, by the nature of the case. When there is distinct evidence of neuritis, the treatment for this, already described, must be adopted. The treatment now to be considered is that of the "idiopathic" form, but the means for the alleviation of all forms is nearly the same.

Although the relief of pain has usually to be the first actual step in treatment, the first in importance is the removal of the causes of the disease. Any discoverable condition on which the neuralgia may depend must be treated. The detailed measures that are necessary need not here be indicated, since they are sufficiently suggested by the enumeration of the causes and causal varieties already given. Especially should any source of nerve irritation be removed, whether in the region of the painful nerve or outside it. It must be remembered, however, that it is not certain that the neuralgia will disappear on the removal of such a source of irritation. This is especially true of neuralgias of the fifth nerve and decayed teeth. The almost universal association of neuralgia with conditions of debility so strongly insisted on by the late Dr. Anstie, indicates the importance of hygienic measures calculated to improve the general health; fresh air, adequate rest, and nutritious food in full quantity, and a small quantity of alcohol at meals is usually directly beneficial. The importance of a good supply of animal food should be urged, for all but gouty subjects. I have known severe neuralgia to occur first on the patient commencing a purely vegetable diet, to disappear when meat was taken, and recur with severity at each of four successive attempts to return to vegetarianism. Cod-liver oil, or other easily digested fat, may often be added with advantage. Iron, when there is anæmia, will sometimes alone cure the disease, and even when there is no anæmia it seems occasionally to be beneficial. Of nervine tonics, quinine is of most value in true malarial forms, given in a full dose shortly before a periodical attack is due; but, as we have seen, it is not always successful. In smaller doses it is occasionally useful in other forms, although not so frequently as might be expected from its marked influence on the nervous system. In stupefying doses it may lessen the pain for a time, but does not often produce a lasting effect. It has been thought to be most useful in neuralgias of the first division of the fifth nerve. Nux vomica or strychnine often does more good than quinine. In general, it may be said that whenever the nervous system is feeble, as it so frequently is in neuralgia, nux vomica may with advantage be added to the other remedies that are given. Zinc (including the phosphide) is of little value. Arsenic is occasionally useful, especially in the neuralgias that have been set up by malarial poisoning, and in the degenerative variety. Free phosphorus ($\frac{1}{50}$ or $\frac{1}{30}$ grain) has been strongly recommended, but my experience coincides with that of most recent writers in assigning it a very low position in the list of remedies —low so far as numerical success is concerned, although in rare cases its influence is very striking. For instance, a woman aged forty-three, with neuralgia of the fifth nerve of thirteen years' duration, at one time associated with brachial, and afterward with crural neuralgia, lost the pain entirely during three months' treatment with phosphorus, although when the treatment was commenced about twelve severe paroxysms occurred every day. Ammonio-sulphate of copper has been recommended in cases of neuralgia of the fifth nerve. It may be given in doses of $\frac{1}{16}$ or $\frac{1}{12}$ of a grain after food. In syphilitic forms, dependent on actual nerve lesions, iodide of potassium and mercury should, of course, be given. Iodide of potassium is now and then useful in cases of neuralgia that are not due to syphilis.

Of remedies that have a sedative action on the nervous system, although they are not anodynes, the most important is bromide of potassium. It is occasionally of service in cases of idiopathic neuralgia, paroxysmal in occurrence, although it appears to have less influence on sensory than on motor nerve cells. Its value is greatest in irritable, anxious subjects. Chloride of ammonium has been recommended in intercostal neuralgia. In the rheumatic forms, salicylate of soda has been occasionally found useful, especially in facial neuralgia and sciatica, but in the cases of sciatica which depend on neuritis it has little influence.

Nervine stimulants given at the beginning of an attack occasionally cut it short, especially in neuralgias of the fifth nerve. The most effective are sulphuric ether, valerian and alcohol. The use of alcohol to relieve pain is, however, fraught with great danger to the habits of the patient, and should be avoided as far as possible. Another nervine stimulant, turpentine, is an old remedy for sciatica, and has been occasionally employed with cases of ordinary neuralgia. Nitro-glycerine is sometimes useful for the same purpose, and, as a rule, it succeeds whenever alcohol is effective. It is also extremely valuable in many cases, given regularly three times a day in doses from $\frac{1}{200}$ to $\frac{1}{50}$ of a grain. It is most useful, as in migraine, when the face gets pale at the onset of an attack, but sometimes succeeds when this indication is wanting, although very rarely when flushing is an early symptom.

Anodynes are necessarily very important elements in the treatment of neuralgia. It is the pain for which the patient seeks help; the cause of the pain can only be slowly influenced, and in the meantime the pain itself has to be relieved. In some cases relief is all that can be afforded; treatment fails to prevent the recurrence of pain, and all that can be done is to lessen its intensity. But in a large number of cases of neuralgia anodynes do more than merely palliate; the repeated removal of the pain tends to prevent its recurrence. This makes it probable that the relief afforded is not merely by an action of the drug on the general sensorium, but that it has an influence on the specific disturbance which gives rise to the sensation of pain. The same conclusion is suggested by the fact that most acute pain may be entirely relieved by an anodyne which has no apparent influence on the general sensory functions of the brain.

No drug gives relief so quickly and so surely as opium or morphia. The hypodermic injection of the latter has to a large extent replaced its administration by the mouth, on account of the rapidity of its action and the slighter degree of gastric and intestinal disturbance which it causes. In epileptiform neuralgia Trousseau found no remedy comparable to opium, which he gave in doses rapidly increasing up to 300 grains of opium or 60 grains of sulphate of morphia a day. Morphia beneath the skin may be given in all forms of neuralgia. It is probable that in most cases it is better to inject it into the seat of pain, because it can then exert some influence on the terminal nerve endings, and, moreover, the injection has a slight counter-irritant influence. But its chief action is on the centre, and this is exerted equally wherever the injection is made; therefore, if, for any reason, local injection is undesirable, a distant convenient region, as the back of the forearm, will answer almost as well.

The dose of morphia should not at first be large, one-tenth, one-eighth or one-sixth of a grain, according to the severity of the pain. It is unsafe to commence with a large quantity; death has in more than one instance resulted from an injection of one-fourth of a grain, and, still more frequently, alarming symptoms have been produced. It is remarkable, however, in some cases of neuralgia, how little effect beyond the relief of the pain morphia produces. The addition of atropine (in amount about one-twentieth of the morphia used) will often prevent nausea and giddiness and increase the influence of the pain. Caution should be used to prevent the dangerous "morphinism," most cases of which have been set up by the use of this drug for the relief of neuralgia. The hypodermic syringe should never be placed in the hands of the patient. In cocain, however, we have an agent that can often replace morphia. A local injection of from half a grain to a grain often gives great relief to the pain, apparently by arresting all impressions from the periphery, which, as we have seen (p. 1140), may have a great influence in keeping up the morbid state of the centre, even in cases in which there is no peripheral irritation, and still more when there is. It has little or no central influence, and has, therefore, less permanent effect than morphia. Repeated injection may, however, lead to recovery, if there is peripheral irritation, or a spontaneous tendency to the subsidence of the morbid process in the centre.

Belladonna may be given by the mouth (one-sixth to one-third of a grain of the extract) or atropine by the skin ($\frac{1}{120}$ to $\frac{1}{60}$ of a grain). Occasionally these give marked relief to the pain, and when this is the case they are said to produce a more permanent effect than morphia (Hunter, Anstie, Vanlair). The unpleasant dryness of the throat which is produced by full doses sometimes constitutes a difficulty in the use of these drugs. It is often convenient to alternate injections of atropine with those of morphia in cases in which there is danger of the establishment of the morphia habit.

Aconite and gelsemium are said to have a special action on the fifth nerve, and are frequently useful in trigeminal neuralgias. Aconite is the more powerful of the two, but often causes nausea and unpleasant symptoms. In slight cases, especially those which depend on dental caries, gelsemium is often useful; fifteen minims of the tincture may be frequently repeated. Aconitia may also be given in doses of $\frac{1}{250}$ to $\frac{1}{100}$ of a grain, but it is a somewhat dangerous remedy to employ hypodermically. The alkaloid of gelsemium (gelsemia), will also be found useful for subcutaneous use; the dose is $\frac{1}{60}$ to $\frac{1}{30}$ of a grain. Cimicifuga is often very useful for neuralgia associated with rheumatism, either alone or combined with Indian hemp. In gouty subjects, lithia may also be given with advantage combined with cimicifuga.

Indian hemp is another remedy of great value in certain forms of neuralgia, especially those in which the pains are sudden, brief and sharp, without the violence which marks true epileptiform neuralgia. It is often very useful continuously administered between the intervals; a quarter to half or even one grain three times a day. Piscidia erythrina has been recommended when pain is not severe; in moderate doses (ʒss of the liquid extract) it leaves no unpleasant after-effects. Chloral has little influence over pain, but croton-chloral (butyl-chloral) is sometimes useful, especially in neuralgias of the fifth. It is usually given in doses of five grains, but, as Ringer and others have shown, the dose may often be increased (up to twenty grains) with advantage. Combinations of nerve tonics and sedatives are generally necessary, but as the sedative has to be continuously given, its dose must, of course, be moderate. I have found, for instance, in neuralgia of the fifth, the combination of arsenic, quinine and Indian hemp of great service.

Neuralgic pain is often relieved by local treatment, which is, for the most part, of two kinds, irritant and sedative; and some remedies combine the two. Counter-irritation sometimes gives great relief to neuralgia, but the mode in which it acts is uncertain. Hypothetically, it acts through the vessels, but this influence is more intelligible in neuritis than in neuralgia. It may also exert an inhibitory influence, or may change the mode of action of the nerve centre; just as a blister around the limb will stop the aura of epilepsy. Either blisters, or sinapisms, or the actual cautery may be employed. Flying blisters may be applied over the tender spots, or by the side of the spine. Anstie believed that the latter yield the best results. Chloroform has been injected beneath the skin, fifteen or thirty minims, but, although it often gives some relief, it may cause a troublesome slough. In trigeminal neuralgia the blister may be applied beneath the occiput or behind the ear. The actual cautery is of most service in spinal neuralgias. The gas cautery, or galvanic wire, or thermic hammer is the form generally used. The local injection of carbolic acid or of osmic acid has lately been recommended; they have been employed chiefly in trigeminal neuralgia and in sciatica.* One or two drops of a one per cent. solution of osmic acid (in water and glycerine) have been injected at a time. It is said to give immediate relief to the pain; some cases are reported as being cured after about a dozen injections. It does not appear to be entirely free from danger; Jacoby observed palsy of the radial nerve to follow an injection into the arm, as it occasionally follows an injection of ether.

Acupuncture, the introduction of needles into the painful part, is a method introduced from China and Japan, the value of which is not great. If, as in the East, the needles are left in for some hours or a day, they cause much pain. Aquapuncture has also been employed; it consists in the injection of pure water into or beneath the skin. Originally, it was introduced into the skin, or between the true skin and the epidermis, under considerable pressure, so as to force the water to separate the tissues and make spaces for itself. This method gives much pain and does little good. The injection of water beneath the skin is innocuous and occasionally relieves a slight pain.

Of external applications that are at once irritant and sedative chloroform is the most important, pure, or as the Linimentum Chloroformi (B.P.), or a dilution of one part of chloroform to six of Lin. Saponis. Next in value are the ointments of veratria and aconitia. They should be rubbed in until tingling is produced, followed by numbness. The milder tincture of aconite may also be painted on the part two or three times a day, avoiding any sores. Camphor-chloral (camphor and chloral rubbed up together in equal parts, so as to liquefy) may also be applied on lint covered with oil silk. Both this and chloral will blister if left on too long. Menthol is also a useful application to the skin in the slighter forms of neuralgia. It causes a singular sensation of tingling and coldness, and for the time lessens the pain. It may be employed pure in the solid form, or rubbed up with chloral, or with spirit or glycerine. Preparations of opium (usually oleate of morphia) are of very little value as external applications. Belladonna is of greater service if employed sufficiently strong; the extract, diluted with one, two or three parts of glycerine or vaseline, may be smeared over the skin two or three times a day. The oleate of atropia (five per cent. solution in oil) may be used in the same way. Lanoline has been lately introduced as a better vehicle for external sedatives. These local applications apparently act by lessening the ordinary stimuli which pass along the nerves to the centre, and some of these agents substitute for the ordinary stimulation a different form, which apparently lessens instead of increasing the morbid action of the centre.

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* Merces, *Lancet, 1885*, No. 2; Jacoby, *Trans. Am. Neurolog. Ass., 1885*, p. 11; Schapiro, *Petersburg Med. Wochenschrift, 1885*.

Among external applications that of simple cold or warmth should be mentioned. Each occasionally gives temporary relief if applied continuously to the seat of the pain. In most cases heat is the safer and more effectual. Considerable heat is often little felt at the focus of most intense pain. If there is the least suspicion of active neuritis heat must be applied to the distribution of the nerve with extreme caution, or disastrous consequences may ensue (see p. 72), A warm douche through the nose has been strongly recommended by Seeligmüller in cases of supra-orbital pain, supposed to be produced in the frontal sinuses, even when of malarial origin. Ether spray to the spine has also been recommended in various forms of neuralgia. Cold to the painful part is rarely useful, except in the case of neuralgia of the testes, where refrigeration can be more completely effected than elsewhere.

Electricity, properly employed, is an agent of considerable value in the treatment of neuralgia. It may be used in two ways: (1) A strong current, causing, for the time, considerable pain, will occasionally remove the neuralgia at once, no doubt by altering the form of central action, just as does a counter-irritant. The effect is probably only produced in cases of idiopathic neuralgia of slight degree. In some cases of this class, and invariably in neuritis, a strong application renders the pain worse, and the risk of this effect must be borne in mind. The forms in which it is most likely to be successful are recent cases of hysterical neuralgia, especially when the pain is seated in the joints. To produce this energeting effect either faradaism or voltaism may be used. (2) A weak current may be used for its sedative action on the nerves. Either form of electricity may be employed, but the action of the two is essentially different. The voltaic current is the most frequently useful. Authorities are divided on the best method of applying it, but the majority are of opinion (and with this my own experience agrees), that it is best to neglect the direction of the current, and to place the positive pole near the seat of the pain, and the negative in some indifferent situation. If there is reason to believe that the neuralgia is central, the positive pole may be placed, during part of the application, as near as may be to the central termination of the nerve, and, for another part of the time, on the seat of the pain. The strength should be from two to five milliampères, but if the battery is not provided with a galvanometer, the number of cells must be regulated according to its effect on the sensory nerves, so as to cause a slight tingling or burning sensation, not actual pain. The number of cells to be used will vary in different parts, chiefly in consequence of the varying sensibility and resistance of the skin. The patient's sensations are, indeed, often the best guide. For the face, three, and elsewhere, five cells may be employed to begin with, and the current gradually increases until it can just be felt. All sudden variations in strength should be avoided. A well-wetted sponge should be employed as the electrode, and this should be very gently and gradually applied, and very gently removed when the strength is altered. If the faradaic current is employed as a sedative, it must be extremely weak (so as to be just felt, but to occasion no pain), and rapidly interrupted. The rapidly recurring slight stimulation of the nerves produces after a time a sedative effect, at first slightly increasing, but after a few minutes distinctly relieving, the pain. Its action is analogous to, and quite as effectual as, the mechanical percussors recently introduced.

The surgical treatment of neuralgia comprehends the division, excision, and stretching of nerves, and the ligature of arteries. It is a very large subject, and for detailed particulars the reader is referred to works on surgery. Neurotomy has been frequently adopted in cases of severe, old-standing neuralgia, especially in the branches of the fifth nerve. Sometimes it is successful, more often it fails. Temporary relief may be given, and may continue for some months, but is too often succeeded by a return of the pain in all its old severity. This has been ascribed to the union of the divided ends, and to prevent this the excision of a certain length of the nerve, or the deviation of the extremities has been recommended. In cases of neuralgia of the second division of the fifth, the excision of Meckel's ganglion has sometimes given better results than simple division of the nerve.* It is probable, however, that the transient relief is often due to the influence of the surgical irritation on the centre, which soon passes off.† In central neuralgia, or organic lesions high up the nerve (as, for instance, in the post-herpetic form), the operation usually fails. It is only to be thought of in cases in which other means have been fairly tried without success, and in which all the branches to which the pain is referred can be divided; it is said that the operation is more likely to be successful if pressure on the nerve relieves the pain. The operation is inadmissible if the nerve is one paralysis of which involves grave consequences, as, for instance, in the case of the sciatic, division of which causes permanent palsy and atrophy of a large part of the leg. These consequences might be deliberately chosen if it were certain that the pain would be exchanged for them, but, unfortunately, relief is too uncertain to make it justifiable to run the risk of adding physical disability to undiminished suffering. The same consideration (and the fact that it can do no more than neurotomy) precludes amputation, except, perhaps, in rare cases in which the pain starts from the extremity of a finger or toe, and local tenderness, etc., make it probable that a cause of nerve irritation exists.*

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* See a paper by Dr. Chavasse, of Birmingham (Royal Medical and Chirurgical Society, meeting of Feb. 20th, 1884, and discussion thereon).

† Neuralgia will sometimes disappear for a time under strong mental influence. Le Fort, for instance, mentions that a most obstinate lingual neuralgia ceased during the siege of Paris, and returned when the anxious time was over.

Fortunately, in nerve stretching we have an operation which produces some of the effects of neurotomy, arresting for a time the conducting function, and effecting what may be called an alterative stimulation of the nerve. Its influence is less lasting, but so are also its inconvenient effects, and, as it is sometimes successful, it should generally precede division of the nerve. It has been recommended that strong traction should be made on the central end, but it is doubtful whether this has much influence, and, in the case of the fifth nerve, it is somewhat perilous.† Recent literature abounds with records of cases of apparent cure from nerve stretching, even when the neuralgia seemed to be central. For instance, a severe intercostal neuralgia for twenty years' duration is said to have been cured by stretching the terminal branches of two or three of the nerves.‡ Intensely severe neuralgia of the third division of the fifth—the pain being in the ear, temple, lower jaw, and tongue—of five years' duration, is said to have been cured by a single stretching of the lingual nerve in the tongue.§ In this and many other cases the pain did not immediately cease, but gradually subsided in the course of one or two weeks. In some rare cases, in which the sheath of an accessible nerve can be felt to be greatly thickened, the nerve has been exposed, the sheath opened carefully, and the nerve for some distance separated from it. This operation has been successful in arresting the pain, especially in traumatic cases.||

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* It has been proposed to tear the nerve in two, by making traction on the central portion. The alleged advantage is that the nerve separates nearer the centre than it can be divided. But it always tears within a centimetre of the place where it is seized; the advantage is probably imaginary, and the method is not without danger (see a discussion at the Parish Société de Chirurgie, Dec. 6th, 1882).

† There is evidence that too vigorous stretching of the second division of the fifth nerve has caused destructive inflammation of the eyeball, probably by the mechanism of inflammation of the Gasserian ganglion (Nicaise and Tillaux, Soc. Clin. de Paris, March 9th, 1882).

‡ Nussbaum, *Aerzt. Int.-bl.*, *1878*, No. 53.

§ Le Dentu, *L'Un. Méd.*, *1881*, vol. ii, p. 766.

|| An instance is recorded by Le Fort "Soc. de Chir.," July 26th, 1882, in which the

Ligature of arteries has been confined to that of the carotid, as a last resort in cases of neuralgia of the fifth nerve. All that can be said to justify so dangerous an operation is that it has sometimes, but very rarely, been successful. Compression of the carotid occasionally cuts short an attack of pain, and the repetition of this treatment, continued for a long time, has even produced permanent alleviation.

An attempt to estimate the true position of the surgical treatment of neuralgia is unfortunately beset with almost insurmountable difficulties, due to the fact that after many operations the pain has ceased, or been much lessened, for a time, but has afterward returned, and the cases have been published before sufficient time has elapsed to permit an opinion to be formed of the permanence of the effect. Moreover, the numerous cases in which there has not been even temporary relief are seldom published. In some instances, operation after operation has been submitted to by the patient under the urgent compulsion of continued suffering, and the records of many cases illustrate very strikingly the need for caution in drawing any inferences from transient relief.*

VISCERAL NEURALGIAS.

The internal viscera of the thorax and abdomen are sometimes the seat of neuralgic pain. Such neuralgias are described in full in the works that deal with the diseases of these organs, and this arrangement is convenient, since the principles of diagnosis involve a differential discussion of the symptoms of organic diseases of these organs which would be out of place in the

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median was surrounded by a dense sheath of connective tissue, due to inflammation produced by a gunshot wound.

* The following cases are illustrations of this. In one instance, a man, at the age of forty-five, had his first attack of neuralgia in the third division of the fifth nerve. Four teeth were extracted from the lower jaw and then one from the upper, and, as the pain ceased for three weeks, the case was published as cured. The pain returning, resection of the alveolar process was performed; the pain ceased for five months, and the case was again published as cured. After a relapse, the inferior alveolar nerve was excised, and freedom for some time was followed by a return, for which the carotid artery was tied, but the effect of even this was not permanent (J. C. Hutchinson, *Am. Med. News*, *1885*, p. 395). The same author relates a case of neuralgia of the fifth, in which the following operations were performed without success:—an incision through the skin above the ear, division of the supra-orbital and infra-orbital nerves, excision of half an inch of the supra-orbital, ligature of the carotid, destruction of the nerve in the infra-orbital canal. In a case, recorded by Schupper, of neuralgia of the fifth with reflex spasm in the face, the first procedure was scarification of the outer and inner surfaces of the upper jaw; this failing, the surgeon excised the infra-orbital nerve; this likewise failing, he excised the superior maxillary nerve in the spheno-maxillary fossa; there being no relief, he removed the peripheral segment of the same nerve from the base of the orbit. The pain then migrated to the third division of the fifth: the inferior maxillary nerve was therefore excised. An interval of freedom was followed by a return of the pain, and the common carotid was tied. The pain continuing, the facial nerve was divided, and on account of severe hemorrhage, the external carotid and temporal arteries were tied, and the patient is said, at last, to have been "cured."

present work. A brief outline of the general facts may, however, be given here.

Most of the organs in which these neuralgias are felt receive their chief nervous supply from the sympathetic system. Little sensation attends their normal function. Although it cannot be doubted that afferent impressions are constantly passing to the cerebro-spinal centres, these fail to affect consciousness under normal circumstances. But repeated attention may vastly increase the sensitiveness of the perceptive centres to such impressions, and from such increase arises a large amount of the discomfort of those patients who are termed "hypochondriacs"—correctly, in so far as the organs which lie below the rib-cartilages disturb their conscious life. But the sensation from the viscera may also amount to actual pain, in consequence of the afferent impressions being abnormal, owing to organic disease and varied functional disturbance. Pain may also be felt apart from either of these causes, and such pain is called "visceral neuralgia." Often we cannot tell to what extent it is the result of a local abnormal condition of the nerves of the organs, and how far it is central. It is probably local in causation to a larger extent than in the case of the cerebro-spinal forms of neuralgia, but the same general pathological laws doubtless underlie the two. The difficulty of investigation depends not only on the fact that the organs are concealed from direct examination, but also on the circumstance that they receive their innervation from two sources, from the sympathetic, and from the cerebro-spinal centre by the pneumogastric and spinal branches, and we do not know what share these sets of nerves respectively take in the production of visceral pain. There is, moreover, reason to believe that not only the nerves in the viscera but also the nervous plexuses outside them may be the seat of neuralgia. The difficulties of investigation are greatly increased by the fact that pain may be the only symptom of organic and of functional disease, and it is certain that many examples of such disease have been included among the visceral neuralgias by some writers on the subject.

Most forms of visceral neuralgia are more frequent in females than in males, and may be produced by the same inherited tendency that causes the cerebro-spinal forms. Their general causes are also, for the most part, similar. In women, anæmia and hysteria are especially prominent. They may also result from causes that have a local action, and this fact adds not a little to the obscurity of their diagnosis. The essential symptom is pain, which varies greatly in character, sometimes dull and diffuse, sometimes sharp, circumscribed, lancinating or burning. It usually presents paroxysmal exacerbations, and is sometimes actually intermitting. I have known paroxysms of gastric pain to alternate with headache. The exacerbations may be apparently spontaneous, or may be produced by various stimuli, especially by those concerned in the functional activity of the organ. We ought not, however, to include among the neuralgias (as some have done), cases in which pain is confined to periods of functional activity. It is doubtful whether, in such cases, the affection is ever a pure neuralgia.

The diagnosis of visceral neuralgias is as difficult as it is important. Pain is the common, and may be the only, expression of various and very different maladies. The first and chief element in diagnosis is the exclusion of organic disease, by every method of investigation that can be made available. If functional disturbance exists, the affection can only be regarded as neuralgic when the pain is not related to the disturbance of function either in time or in degree.

The treatment of visceral neuralgias must be based on the same general principles as that of the cerebro-spinal forms. It consists in the removal of causes, general tonic treatment, abundant rest, and the use of sedatives. The mode of employment of the latter differs in each case, and in each, also, special measures are necessary, related to the function of the organ concerned.

Special Forms.—Of the intra-thoracic organs, the lungs do not appear to be the seat of neuralgia, although the pain of "pleurodynia," already described, is probably due to an affection of the pleural nerves. The only important cardiac affection of this class is the disease known as "angina pectoris." Severe nerve pain is a prominent symptom of the disorder, but its peculiar and special characters and associations prevent its inclusion among the forms of pure neuralgia.

The most important visceral neuralgias are those of the abdominal organs. They are usually local and well defined, but occasionally abdominal neuralgic pain varies in its seat, and is felt now on one side, now on another. That of the stomach (*gastralgia*, *gastrodynia*) is one of the best-marked forms. It is frequent in anæmia and hysteria, and a special form constitutes the gastric crises of ataxy. The pain is felt at the epigastric region, and, like most gastric pains, passes through to the back. Pressure does not usually increase it, often, indeed, it relieves the more intense suffering. There is usually a constant dull pain, with more acute exacerbations. It may be most intense when the stomach is empty, and may be relieved by food; it is then possibly due to the morbid action of the nerves or centres from which, in health, the sensation of hunger arises. Or the pain may be increased by food, and in such cases food is often vomited as soon as it is taken. Appetite may be absent, lessened, increased or perverted. The relation of the condition of appetite to the effect of food in relieving or increasing the pain deserves further study.

The stomach is readily influenced by sedatives, and their administration by the mouth forms an important part of the treatment. Other agents are sometimes useful. In the form that is relieved by food, oxide of silver is often of great service. When there is anæmia, the combination of subcarbonate of bismuth and saccharated carbonate of iron, given before food, is frequently effective. No sedative, however, is on the whole so useful as cocain, of which a grain may be given when the pain comes on. By repeatedly relieving the pain in this manner, the disease is often cured, apparently by the rest thus given to the nerves of the mucous membrane.

The frequency of intestinal neuralgia (*enteralgia*) has probably been exaggerated. We are not justified in regarding as enteralgia either vague abdominal pains, which are not increased by peristaltic action, or pain that occurs only when the intestines are in energetic action, or in which there is conspicuous disturbance of the mucous membrane. If these are excluded, the cases of enteralgia become extremely rare, and the history of the affection has yet to be worked out. There is, however, one part of the intestine which is, undoubtedly, the seat of neuralgic pain—the lower part of the rectum. This form occurs in both sexes, as a deeply-seated pain above the anus and coccyx, more or less constant, but with severe exacerbations, apparently due to spasm. It is usually effectually relieved by suppositories. In some cases, indeed, it is probably a primary spasm, and I have known it produced in a child by the use of senna as an aperient.

Of neuralgia of the liver, *hepatalgia*, much the same may be said as of enteralgia. Deep-seated pain is occasionally felt in the position of the organ, sometimes diffuse and dull, sometimes sharp and lancinating, but the cases in which we can be sure that it is truly neuralgic are extremely few. In some cases such pain may be due to disturbed function of the organ. Disturbance of function has been regarded by some writers as a consequence of neuralgia. It cannot be denied that such nerve pain may cause such reflex derangement of function, just as it causes vasomotor disturbance in other situations. But the frequency with which pain is the first evidence of disease outside the nervous system, which afterward causes other symptoms, renders it necessary to exercise great caution in regarding such cases as primarily neuralgic.

The existence of neuralgia of the spleen is not well established, but occasionally, especially in cases of hysteria, there is deeply-seated pain and tenderness in the position of the organ, apparently situated in it or in the nerve plexuses in its vicinity.

The kidney, and apparently the ureter, may be in rare cases the seat of the neuralgic crises of tabes, analogous to the more frequent gastric crises, but the occurrence of primary *nephralgia* is a still more rare event, and can hardly be regarded as proved. Many cases of supposed renal neuralgia have certainly been due to the passage of a calculus. I have, however, met with one case in which paroxysms of pain in the renal region had occurred at times during forty years without any indication of a calculus, and it seemed on the whole probable that the pain was of nervous origin.

Neuralgic pain which cannot be referred to any organ is sometimes felt within the abdomen. Such pain is diffuse, varies in intensity, is not increased by pressure, and is not related in time to the functional activity of the organs, or in degree to their disturbance. It is generally central in position, but may be felt above or below the umbilicus, apparently seated in the sympathetic nerves, but has not such a relation to their plexuses as to permit definite localization.

The female generative organs are frequent seats of pain and tenderness. Tenderness in the region of the ovaries is extremely common in hysteria and conditions of nervous weakness; sometimes there is much spontaneous pain in this situation, deeply seated and aching or burning in character.

This pain may exist when no evidence of organic change in the ovary can be detected, and appears then to be a pure neuralgia; but in most cases the tenderness is extensive, and exists in the vicinity of the ovary as well as in the organ itself, so that it is probably due, at least in part, to the abundant nerve plexuses with which the ovary is surrounded. The uterus is also the seat of spontaneous pain and of tenderness, apart from organic disease or of displacement. These symptoms may occur in the young and also in later life, even when the involution of the organ is complete. They are often associated with neuralgic pains elsewhere—in the ovaries, and especially in the spine, including the sacrum. By some writers the pains of menstruation, when no organic cause for them can be discovered, are regarded as neuralgic.

Of the male organs of generation, that which is most frequently the seat of neuralgic pain is the testicle, but even here such pain is comparatively rare. It is important to remember, however, that inflammations of the urinary passages are not uncommon causes of neuralgia seated in the adjacent cerebro-spinal nerves.

MIGRAINE: PAROXYSMAL HEADACHE.

Migraine is an affection characterized by paroxysmal nervous disturbance, of which headache is the most constant element. The pain is seldom absent and may exist alone, but it is commonly accompanied by nausea and vomiting, and it is often preceded by some sensory disturbance, especially by some disorder of the sense of sight. The symptoms are frequently one-sided, and from this character of the headache the name is derived, the Greek "hemicrania" (still often employed) furnishing the French migraine, the German migrän, and the English megrim.[*] The French word is that most frequently employed, and is, on the whole, the most convenient. On account of the associations of the pain, it has received the popular names of "blind headache," "sick headache," and "bilious headache," the latter being derived partly from the fact that bile is often vomited, partly from the old humoral pathology which regarded the bile as one of the chief morbific fluids of the body. The disease is often associated with high intellectual ability, and many distinguished scientific men have suffered from it and have supplied more careful observations of the subjective symptoms than we posses of any other malady. Among the sufferers may be mentioned the celebrated Dr. Fothergill, Marmontel, Haller, Wollaston, Du Bois Reymond, Sir Charles Wheatstone, Sir John Herschell, Sir George Airy, and his son, Dr. Hubert Airy.[†]

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[*] The English word has been developed by the following steps, going backward: megrim, megrene, emigranea; Low Latin, hemigranea; Latin, hemicranea, hemicranium; Greek, ἡμικρανίον (Skeat).

[†] Dr. H. Airy has given a very interesting account of his ocular symptoms in the *Philosophical Transactions* for 1870. The best systematic account of the disease is that of Dr. Edward Liveing ("Megrim, Sick Headache and some allied Disorders," London, 1873).

Etiology.—Females suffer from migraine more frequently than males, but their preponderance is not great, and has been much exaggerated by some writers. The affection usually commences in the first half of life. One-third of the cases begin in later childhood, between five and ten; about two-fifths between ten and twenty, and most of the others between twenty and thirty. The maximum periods are late childhood, puberty and early adult life. Now and then the disease commences after thirty; I have met with one well-marked case which began at sixty. In the ratio between the sexes, and the large number of cases which commence at the time of puberty, the disease resembles another paroxysmal neurosis, epilepsy, and we shall see that there are other points of resemblance between the two diseases. Migraine is strongly hereditary; in more than half the cases, inheritance can be traced, and it is usually direct, *i. e.* other members of the family (very often a parent) suffer from paroxysmal headache. Now and then the inheritance is indirect; relatives suffer, not from migraine, but from some other neuroses, especially pure neuralgia and epilepsy. For instance, a woman aged forty-six had suffered for many years from migraine (paroxysmal headaches with dimness of sight and vomiting). One of her children was epileptic and her mother had been epileptic and insane. I once had under treatment a brother for migraine, and his sister for epilepsy. One patient's brother suffered from paroxysmal headache, her father from severe neuralgia, and her father's brother was insane. Occasionally migraine seems due to the inheritance of the gouty diathesis: a father may suffer from gout, and his son from migraine. A similar transformation may occur in the course of the disease; migraine, commencing in early life, may cease when distinct gout is developed. Trousseau has emphasized (but also exaggerated) this relation by saying that "migraine and gout are sisters."

When migraine begins early, no immediate cause can usually be traced, but when the disease begins later, its occurrence, or in cases that begin earlier, its exacerbation, is related to influences that depress and weaken the nervous system, either directly or through the general health. Such causes are excessive brain work (especially combined with anxiety and loss of rest), over-fatigue of all kinds, work in hot and crowded rooms, anæmia, over-lactation and the like. I have seen one case that appeared to date from attacks of ague, which the patient had had in early life.

Symptoms.—The essential feature of migraine is paroxysmal headache, but a large number of the patients present also other sensory symptoms in association with the headache, and, in rare cases, these sensory symptoms occur alone, without headache. These associated symptoms are so peculiar and striking, that undue importance has been given to them as the characteristics of the disease, but they are often inconstant. Even the same patient may have some headaches with, and others without, these accompaniments, or may have simple headaches at one period of his life and the more complex series of symptoms at another period. The simple headaches have the same characters, and occur under the same causal conditions of heredity, etc., as those in which there are in addition other sensory symptoms.

The characteristic feature of the symptoms is their paroxysmal character. During the intervals, most patients are free from any symptoms of nerve derangement, although a few suffer from slighter headaches of a different character, occasional or continuous. The precise elements that make up the paroxysmal seizure vary in different cases, and often even in the same individual. Headache, as already stated, is the most constant, and is very seldom absent; next in frequency are nausea and vomiting, then some disturbance of vision, affection of speech, disturbed sensation in the limbs, and vasomotor derangement, while the least common are motor symptoms in the limbs. The frequency of vasomotor derangement will be variously estimated, however, according to the phenomena included in the term, and if a simple change in frequency or tension of the pulse, or in the color of the face, is included, this disturbance becomes one of the most frequent features of the attacks.

But the above order of frequency is not that in which the symptoms occur during an attack. The various sensory accompaniments of the headache usually occur first, then comes the pain, and after the pain has lasted for a time nausea occurs, followed by vomiting, and this often ends the attack. Vasomotor disturbance may be present in some form throughout an attack or may come on only toward the close. When sensory disturbance is absent, the pain is the first symptom.

Various influences will induce a paroxysm. Fatigue and excitement are the most common. Digestive disturbance is a potent cause, and sometimes a particular article of diet will always induce an attack, but most of the sufferers who are thus susceptible learn by experience the dietetic errors that are efficient, and carefully avoid them; hence, as an actual fact, it is very common for attacks to be thus induced. Frequently, after the usual interval between the attacks has nearly elapsed, a slight error in diet is sufficient, although, soon after the patient has had an attack, even actual indigestion has no effect. The influence of stomach derangement is also exaggerated on account of a misconception of the significance of the vomiting that so often occurs. The bile that comes up is thought to be a proof of "biliousness," when its rejection is merely the result of the repeated vomiting. In some patients exposure to cold will bring on a paroxysm. Another occasional excitant is a visual impression, such as watching moving objects or seeing some peculiar kind of motion. Over-use of the eyes may also bring on an attack. In some patients a bright light, or a sudden change of light, has the same effect, and so has a loud noise or a peculiar odor. Indeed, it seems as if a peculiar habit may become established, so that a certain sensory impression will always induce a paroxysm.

Premonitory symptoms are present in some cases, but are less frequent when there are accessory symptoms than in the attacks that consist of pain only. The day before an attack the patient may complain of heaviness in the head, or of slight pain, or of somnolence. When attacks consist of simple headache, the patient often wakes up with it. When sensory symptoms occur first, these often begin quite suddenly. The patient, for instance, may feel perfectly well, when he is suddenly conscious of some disturbance of vision, of a bright spot, for instance, on one side of the field of vision, which slowly enlarges and spreads, becoming darker in the centre as it extends, and changing its round outline into an angular form. Or he may suddenly be conscious of dimness of sight toward one side, which increases in extent and intensity until one-half of each field is blind. Or, the first symptom may be tingling in one hand, which spreads up the arm. Such sensory disturbance lasts for ten, twenty, or thirty minutes, and then passes away, and as it subsides, headache comes on ; it is usually of great intensity, commences at one spot, and slowly spreads. After a few hours the patient feels sick, and the nausea slowly increases and at last ends in vomiting; then the sufferer goes to sleep for an hour or two or for the night, and wakes up well. These various symptoms are generally unilateral in distribution. The pain is sometimes bilateral, but it is then greater on one side than on the other. The symptoms may now be considered in detail.

Visual disturbance occurs in at least half the cases, and is the earliest symptom of an attack. It may consist in partial loss of sight, or spectral appearances, or both. The unilateral character of visual symptoms is always manifested as affection of the corresponding halves of both fields of vision.

Loss of sight is always imperfect. There may be sudden general dimness of vision, or there may be a lateral limitation of the field, extending from one side and not reaching the centre, or commencing first on one and afterward on the other side, during the same attack. The resulting hemianopia may be complete. In other cases, the first change in vision is a spot of dimness of sight, lateral or central in position, which gradually increases in size and extends toward the periphery ; when lateral in position it usually does not pass beyond the middle line, so that from this also hemianopia results. Very rarely the spot is situated in the upper or lower parts of the field, and may cause a form of transverse hemianopia. The degree of loss varies ; it is often described as a "cloud," but the darkness may be noticeable only when a bright light is looked at. As the dark spot increases in size it often clears in the centre. When spectral appearances occur, they may commence as a bright spot, gradually expanding, or they may develop out of this area of dimness. In the latter case, as the dark spot increases it becomes luminous at the periphery and expands, so as at first to form a circle, but, if lateral, it may break at the middle line into a crescent. In some cases it spreads over almost the whole field of vision. When a luminous spot is the first change and this expands, it may become dim in the centre. Very commonly the outer edge assumes a zigzag shape with prominent and reëntrant angles, like the ground plan of a fortification, and hence called "fortification spectrum." At one part it becomes fainter and ceases, so that there is a break in the outline. The outer boundary is the most brilliant and is often limited by color ; inside, the luminosity extends for a little distance, gradually becoming fainter. Very frequently within the bright outline, however it arises, there seem to be luminous particles in rapid irregular movement. The spectrum increases with the blind area, gradually becomes indistinct, and disappears at the periphery of the field. These visual phenomena always affect the field of vision of both eyes, although the patient often imagines that the phenomena observed on one side are seen with one eye only. They may present considerable variation in the same case. Thus one patient described sometimes hemianopia, sometimes colored lights, sometimes merely a sensation as of moving water before the eyes. Many patients experience only slight and vague ocular symptoms, such as sparks or mere flashes of light. Very rarely there is double vision.

Disturbance in the function of the other special senses is exceedingly rare, but a few cases are on record in which phenomena have been observed in hearing and taste similar to those in vision. Thus, there has been one-sided deafness followed by a noise in the ear, or loss of taste followed by a subjective sensation. Transient tinnitus is occasionally observed in the subjects of migraine, without connection with the attacks.

Other sensory symptoms are felt in the limbs, face, throat, tongue, and adjacent parts, but these are far less frequent than is the visual disturbance. In the limbs, the sensation is felt chiefly in the arm, very seldom in the leg. It may occur alone as the first stage of the attack, but is more often associated with the visual phenomena, succeeding the latter but commencing before the affection of sight has quite ceased. In character, the disturbance of cutaneous sensibility is very similar to that of vision, allowance being made for the difference in the character of the function. There is the same combination of sensory irritation and sensory loss. Tingling in the skin, or "pins and needles," is felt in some part of the hand, as the fingers, or in the wrist, and as it spreads it is succeeded by numbness and loss of sensibility, sometimes amounting to actual anæsthesia. In other cases, the numbness occurs first, and is succeeded by tingling. The sensory disturbance in its double form may pass up the limb from the extremity, and the leg may be affected after the arm, just as in the sensory aura of epilepsy. Sometimes, instead of a gradual extension, the tingling passes from one part to another at a distance. It is generally confined to one side, but sometimes is felt first in one arm and then in the other. A sensation in the lips and tongue is generally secondary in time to that in the limbs; it rarely exists alone. It may be felt in the cheek, lips, tongue, or fauces, on one side, or on both. The side affected is the same as in the limbs, but it may be bilateral in the lips and throat, and unilateral in the limbs. The duration of these sensations is generally about ten or fifteen minutes. Occasionally slight motor weakness accompanies the tingling, just as it may do in the sensory discharge of epilepsy. For instance, the attack in one patient commenced by dimness of sight, and this was followed by tingling in the fingers of the left hand; the sensation passed up the arm to the shoulder, and was followed by weakness of the limb for about ten minutes; then headache came on in the right occipital region, and lasted for twelve hours.

Motor symptoms in the limbs are usually confined to such transient weakness, with or after the sensory disturbance, as occurred in the case just mentioned. If any motor spasm is present, the case usually diverges very much from the type, and sometimes is of such a character as to render it doubtful whether it should be classed with migraine or not. In one patient each attack of headache was preceded by sudden tingling in the calf, followed by painful cramp in the calf-muscles, lasting a few minutes only. The same patient, however, had at other times attacks in which her face suddenly became crimson, sharp pains occurred in the head, and seemed to pass down the side to the leg, which was then "drawn up" in spasm for a few minutes.

Difficulty in speech, transient aphasia, is another occasional symptom of the commencing attack. If there are sensory symptoms, these are almost always right-sided, and are situated, in most cases, in the right arm. If there is visual disturbance, this also is in the right half of the field, but I have only once met with aphasia in association with an affection of sight alone. The rule of the right-sided association probably does not hold good of left-handed persons, and it is not quite absolute in other cases. I have met with one case, in a right-handed man, in whom the attack began with left-sided hemianopia, followed by tingling in the left foot, which passed up the leg and side to the mouth and tongue, and then the speech was deranged, the words of a sentence "coming out in wrong order." The common character of the defect is that there is a difficulty in finding the right word, or a use of wrong words in both speaking and writing, and very rarely a total inability to speak. The latter suggests motor aphasia (see p. 544), but the more common form has the character of the sensory variety. In one case the affection of speech was clearly of this character; the patient was, for a few minutes, completely "word-deaf;" when spoken to, she heard the sound perfectly, but could not tell what was said. A few similar cases are on record.*

Slight mental change occurs in some patients during the attack. Emotional depression, restlessness, or confusion of ideas are the most common; sometimes there is transient loss of memory. Brief stupor, without complete unconsciousness, sometimes occurs soon after the onset of an attack, without interrupting the progress of the sensory disturbance. Among other peculiar conditions that have been described is a sensation of "double consciousness" or a vivid recollection of events long past. The mental change may be the earliest symptom, or it may succeed the affection of sight, or occur when the sensation in the limbs ascends to the head.

Giddiness is not a frequent symptom of the attacks themselves. It is usually a vague sense of defective equilibrium, rarely amounting to definite vertigo. It varies in time, being occasionally early, but more often succeeding the sensory disturbance, and it often accompanies the headache. Any one of this series of symptoms may precede the headache, or all may be absent. Even when many of them occur, their duration is short, varying from fifteen to thirty minutes, before they give place to the pain in the head.

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* It is probable, and in harmony with the other symptoms, that the chief disturbance of speech is "sensory aphasia," and that when there is "motor aphasia" this is analogous to the weakness of the arm that accompanies the sensory discharge.

The headache is not only the most constant symptom, it is also the most distressing. It is not, however, so alarming to the patient as the sensory symptoms are, especially in those who are unaccustomed to them, and in whom they occur at long intervals, or for the first time in adult life. The pain has generally recurred during many years, and the sufferer knows what to expect and how best to endure it. It varies much in degree, but is very seldom trifling. Often it has a characteristic course, uniform in the same patient; it begins gradually, slowly increases to a considerable degree of intensity, and after a variable time it subsides, sometimes slowly, sometimes rapidly. It is usually an acute pain at the onset, and may remain so throughout, or may assume a duller character as it spreads. Movement, noise, and light usually increase its intensisy. Sudden stooping also makes it worse, but the patient is, nevertheless, most comfortable in the recumbent posture, and suffers more when he attempts to sit up or to stand. In most cases the headache begins on one side, and in many it is confined to one side; in others it becomes general, so that the name, while founded on a common characteristic, has no exact descriptive significance. When the pain begins at one spot it most frequently is in the temple, and is confined at first to a small area that can be covered by the tip of the finger. In other cases it begins in a small spot on the forehead, or in the forehead and eyeball, seldom in any limited area at other parts of the head. When thus limited at first, it seems generally superficial, but often has a boring character, as if some instrument were being forced into the skull. After a time the pain often spreads through a considerable part of one side of the head, and not unfrequently through both sides. Sometimes it begins at the back of the head, in the occipital region on one side, and may then extend forward to the temple. It occasionally commences in the middle of the head, and spreads down one side. From the back or side of the head, the pain may pass down the side of the neck and even into the arm. Occasionally it spreads from one side to the other, and may then cease on the side first affected. In one instance in which it had this course, it ultimately ceased on the second side and recommenced, in slight degree, on the side first affected before finally passing away. Even when the pain is limited in area and superficial in character, there is seldom any local tenderness, but now and then extensive pain over the head may be accompanied by some general tenderness of the hairy scalp, which continues for a time after the headache has ceased. When the pain is unilateral and felt over a considerable area, it is generally on the side opposite to the peripheral symptoms. It usually comes on as the sensory disturbance is declining, and always lasts for several hours, often for the rest of the day. It is not always constant either in character or seat, but when inconstant in seat, the patient has certain kinds of headache which maintain their characters, although sometimes one, sometimes another comes on. In other cases, at a certain period of life there is a change in the seat and character of the pain. Thus in one case the pain for many years was occipital, but afterward it was always limited to one frontal region.

Nausea accompanies the headache in a large number of cases, but often does not commence until the pain has reached its height. It is attended with a total inability to take food, and food that is taken is not digested, apparently from a derangement of the gastric secretions. The nausea often results in vomiting, but retching is still more frequent. Neither occurs, as a rule, until after the pain in the head has reached its climax, and often not until the pain is subsiding, and then may terminate the seizure, and this even when nothing is ejected. It is common for the headache to commence in the morning, for nausea to come on in the course of the day, and vomiting in the evening. Sometimes the vomiting begins earlier; in one case, in which the visual disturbance was unusually prolonged, lasting two hours, the vomiting commenced before it had ceased and before the headache. If it occurs while the headache is severe, the vomiting does not influence the pain. When there is no headache there is no vomiting. The condition is accompanied with great prostration, and the patient is very much like one suffering from sea sickness.

The most common vasomotor symptom is pallor of the face at the onset of an attack, and often throughout its course. The extremities, also, are usually cold. The face is not only pale, but has a "pinched" or "drawn" expression. The accessible arteries may be sometimes felt to be contracted. In some cases this aspect continues throughout the attack; in others the pallor gives place to flushing as the pain in the head develops, and there may even be general perspiration. Rarely the face is flushed from the first. Still more rarely, there is a conspicuous difference in the aspect of the two sides of the face; there may be pallor only on one side, and in addition the eye may be retracted, the conjunctiva injected, and the pupil small; as the paroxysm goes off this condition may be exchanged for one of hyperæmia, the face becoming warm, the ear red, and the pupil resuming its normal size (Du Bois Reymond, Morselli). The latter must be referred to diminished action and the former to increased action of the sympathetic fibres, although in over-action the retraction of the eye and redness of the conjunctiva are exceptional. Unilateral sweating has also been observed. At the end of an attack, in which the final dilatation of vessels has been marked, puffiness of the scalp has been observed in rare cases, and even ecchymoses at the seat of the most intense pain. It must, however, be remembered that such unilateral sympathetic symptoms are not only exceptional but extremely rare. In a woman, aged fifty, liable to right-sided migraine from youth, the right temporal artery was harder and more rigid than the left, and the right cornea presented an arcus senilis twice as broad as that on the other side (De Giovanni). Œdema of the optic disc has been said to occur during the paroxysm (Möllendorff), but, as a rule, both during the attack and in the intervals, the appearance of the discs is perfectly normal. Occasionally, retardation of the pulse occurs during the paroxysms. In one of my patients the pulse always fell to about fifty-six and a retardation to forty has been observed.

The termination of the paroxysm is sometimes attended, not only by vomiting, but also by some secretion, copious diuresis or perspiration. The pain passes away gradually, very seldom quickly. The most frequent termination is by sleep. During the height of the attack the patient may be drowsy and doze, but this brings no relief; as the pain is subsiding, however, he goes to sleep, sometimes for a short time only, and wakes up free from pain. The duration of the headache is always several hours; often it commences in the early morning and lasts the entire day; in severe cases it may last for several days.

Varieties.—The cases in which the collateral disturbance is absent and the attacks consist only of pain and sickness are very common. The pain has the same characters as in the cases with other sensory symptoms, and may be attended by the same vasomotor disturbance. In one patient, for instance, the pain began in one eye and the supra-orbital region and commenced alternately on each side; from the place of commencement it extended over the whole head and into the throat. It is very common for two kinds of headache to occur, and for one only to be accompanied by other sensory symptoms. Sometimes one is attended with vomiting, and the other is not. One patient, for instance, sometimes had attacks of pain in the forehead and temples, and at other times pain at the top and back of the head, and only the latter were attended by sickness.

Migraine in adults is not commonly attended by any alteration in the temperature of the body, but in children there may be considerable pyrexia, which impresses a special and sometimes misleading character on the attack. Thus, one child at the age of two became liable to attacks which lasted only a few hours, and recurred at intervals of two or three months. In each there was severe one-sided pain in the head; the temperature rose to 102° or 103°; sickness came on, the child went to sleep, and woke up well.

The sensory symptoms of migraine, as we have seen, sometimes occur without headache, or, more frequently, with headache that is so slight as to cause the patient no distress, and to lead him to place no weight upon it in his description of the symptoms. These cases are of great importance, because their nature is so often misunderstood. In one case, with characteristic visual disturbance (an expanding luminous spot and hemianopia), the only discomfort was that a cough or deep inspiration caused momentary pain over the eyebrows during two or three days after an attack. A more common history is that some attacks are complete, consisting of sensory disturbance and headache, while in others the former occurs alone. In rare cases the sensory disturbance or aphasia generally occurs by itself, headache being seldom or never associated. Lastly, some sufferers from migraine often have slighter and variable sensory disturbance, evidently of the same nature, although not of the same form, as that which precedes the headaches. One patient, for instance, with characteristic headaches preceded by hemianopia, complained of bright stars before the eyes whenever she had looked at a brilliant light, and sometimes one of these stars, brighter than the rest, would start from the right lower corner of the field of vision and pass across the field, generally quickly, in a second, sometimes more slowly, and when it reached the left side would break up and leave a blue light in which luminous points were moving. These sensations were not succeeded by headache, although the pain always followed the hemianopia. Aphasia does not often occur without headache; such attacks may be very puzzling unless their nature is suspected.

I have met with one curious case in which visual disturbance, exactly such as precedes attacks of migraine, occurred frequently during many years, as an isolated symptom; at no time was there any pain. The patient was a man of sixty, the subject of chronic bronchitis and some loss of memory, but with no other indications of nerve disease. The visual spectrum was generally a brightly colored zigzag; sometimes it had the shape of a broken oval, but more often a long comet-like form, commencing on one side of the field of vision and extending downward. Rarely there was a luminous disc, which would ascend, break into a four-leaved object, and then disappear. A noteworthy feature of the illusion was that the angular spectrum was sometimes related to the image of some seen object. Thus, on one occasion, a plate which was before the patient as he sat at table appeared surrounded by the colored angular spectrum.*

Sensory disturbance in the limbs does not often occur without headache, but occasionally the headache is slight. Sometimes an attack is apparently rendered abortive in consequence of some drug that is taken, such as bromide, as in the following case, which deserves description on account of the illustration it affords of the deliberate march of the sensory disturbance. A gentleman, the subject of migraine, was working with the microscope one afternoon, when his sight became dim, so that he could only just read large print, and continued so in spite of a drachm of bromide. After two hours, tingling suddenly commenced in the left thumb, and spread to the fingers and then was felt in the middle of both lips, in the tip of the nose, and beneath the tip of the tongue. Then it was felt in the left arm near the axilla, and in the left side of the fauces and of the face over the lower jaw. A few minutes later it involved the fauces on both sides and the palate, and caused an unpleasant sense of constriction. It then ceased and headache came on. Another attack began in the same manner, but after being felt in the fingers and lips and tongue, it became intense at the wrist and ceased in the lips; the sensation passed up the ulnar side of the forearm and then ceased, but afterward recurred in the cheek and side of the throat.

Course.—The interval between the attacks varies in different cases; it is usually between a fortnight and two months. It is generally about three or four weeks, but the periodicity is not often exact. In a case mentioned by Trousseau, however, the attacks occurred each fortnight, almost to an hour.

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* The patient was a mechanical draughtsman, and he had a small book full of drawings of the appearances that he saw.

In women they often occur about the menstrual period, generally after the catamenia have ceased. The intervals are, on an average, shorter in the cases in which there is only pain than in those in which there are associated sensory symptoms, and when these symptoms occur alone, without headache, the intervals generally amount to several months. The intervals are doubtless rendered more irregular than they otherwise would be by the influence of exciting causes, effective when the usual period has nearly elapsed. In the intervals there are often no symptoms, or there may be slight headache of a different character, or definite neuralgia, or some other functional disturbance.

It is not uncommon, as already mentioned, for some change in the character of the disease to occur at a certain period. Sensory disturbance may cease, or, if previously present, may come on. Thus one patient had hemianopia with the attacks of migraine until the age of fifty, when the visual disturbance ceased, and the headaches occurred alone. Occasionally, some morbid influence, chronic ill health, acute disease, anxiety, or injury, may induce an increase in the intensity of the affection or a change in its character. Thus one patient, whose mother was the subject of migraine, suffered from simple "sick headaches" since childhood. At twenty-five he had a slight concussion of the head, falling against a wall. Hemianopia came on in a few minutes, followed by headache more severe than the patient had had before, and from that time each attack was preceded by the same visual symptom.

Migraine does not itself involve any danger to life. We have seen, however, that the vascular disturbance may lead in time to local vascular degeneration, and this affords an explanation of the observed fact that sufferers from migraine occasionally suffer from vascular lesions of the brain comparatively early in the degenerative period of life; but this sequel is rare. In some cases, after many attacks attended by intellectual impairment, some failure of mental power has been observed in the intervals.

Complications and Associations.—Vertigo is occasionally met with in the subjects of migraine, not only as part of an attack, but also as an occasional symptom at other times. Some of those who present it are in the second half of life, and the associated symptoms show that the vertigo is of the labyrinthine variety. Thus, one patient, forty-seven years of age and gouty, who had been liable for many years to migraine, had an attack of vertigo with brief tinnitus and sickness; the watch was almost inaudible through the bone on each side, and no note of Galton's whistle could be heard through the air. In him tinnitus was inconstant, and heard occasionally on each side. It is probable that in such cases the central tendency to functional derangement renders the patient peculiarly susceptible to the influence of labyrinthine changes. I have several other examples of the same combination. In other cases, again, sudden attacks of vertigo occur without any indication of aural changes, and sometimes seem to be the result simply of the central instability. Thus, one patient was liable, in the intervals between the attacks of migraine, to sudden sensations of unsteadiness, vaguely referred to the legs, without any aural symptoms. Another patient had sudden attacks, in which there was a tendency to fall backward, accompanied by sickness. At other times she had paroxysmal headaches without sickness.

The relationship of migraine to other diseases is of great importance. That to gout has been already mentioned in the account of the causal relations of the disease. An alternation is often observed with some other forms of neurosal disorder, or at least a transition from one to the other. Migraine occasionally ceases, and is replaced by simple neuralgia. Many other instances of such transition have been collected by Liveing, as, for instance, to gastralgia, laryngeal spasm, anginal seizures and paroxysmal insanity. In one case acute mania came on.

The most important, and one of the most frequent, of these associations is the relation of migraine to epilepsy. The connection of the diseases is of special interest because the sensory disturbance of the two has so many common features. I have met with no less than twelve cases in which these maladies occurred in the same individual. In seven instances, migraine had existed for many years, and the patient afterward became epileptic. In five of these the migraine either ceased or became much slighter when the epilepsy developed; in one the opposite relation was observed; an epileptic patient began to suffer from migraine when the fits ceased. One patient, who was liable to migraine before the epilepsy commenced, suffered scarcely at all from the headaches while the fits occurred, and when these ceased the attacks of migraine again became frequent. In five cases both maladies coexisted in considerable intensity. In almost all the individuals who had suffered from the two diseases, the attacks of migraine were attended by well-marked sensory disturbance in addition to the headache and vomiting, and in one or two, abortive attacks of sensory disturbance occasionally occurred. In several cases, moreover, the epileptic attacks began by a local aura in the limbs, a rare feature in idiopathic epilepsy. In one instance of this, the first convulsive attack occurred after a fright. In some cases, in which epilepsy succeeded migraine, the epileptic fits seemed, as it were, to grow out of the attacks of migraine, being preceded by such sensory symptoms as had occurred before the attacks of headache. Thus, a young man, whose sister was epileptic, began to suffer from attacks in which he saw glimmering lights in the right side of the field of vision, lasting about twenty minutes, and followed by headache, sometimes for half an hour, sometimes for the rest of the day, accompanied by nausea but no vomiting. He had such an attack every few months, and one day a similar light appeared, brighter than usual, and after it had lasted for twenty minutes he lost consciousness in a convulsive attack, which, from the intensity of the subconjunctival ecchymoses, must have been severe. In rare cases of epilepsy, again, a visual aura may consist of fortification spectra with colors, and even, as I have seen, with hemianopia. In one such case the visual disturbance lasted ten minutes, occurring sometimes alone, sometimes with transient loss of consciousness, sometimes with a convulsive attack. Again, a woman had suffered from epileptic fits for two years, and also, since youth, from attacks of severe headache, lasting all day; in the course of the headache she would have attacks in which there was first a sensation commencing at the epigastrium and passing up to the head; it seemed to spread over the head, and then the sight became dim, and there was complete word-deafness and aphasia; after about ten minutes these symptoms suddenly ceased. Many of her epileptic fits were preceded by the same sensation starting from the epigastrium and going to the head.

PATHOLOGY.—No anatomical changes are known to underlie the phenomena of migraine, and from the character of the symptoms, and the analogies of the disease, it is unlikely that any will be discovered. Hence the nature of the malady is a matter of inference, and hypotheses are sufficiently abundant and precise.

Two chief theories have been held regarding the origin of the attacks. One is based upon the alteration in the state of the vessels that is so conspicuous in the aspect of the patient. The pallor of the surface must be due to contraction of the arteries, and the flushing of the skin to their dilatation, and it is assumed that a corresponding condition of the vessels of the brain is the cause of the derangement of function. The suggestion that spasm of the cerebral arteries is the cause of the symptoms was first made by Whytt, and the evidence in favor of this opinion, afforded by the state of the accessible vessels, was pointed out by Du Bois Reymond. Mollendörf urged that vascular dilatation, rather than spasm, caused the symptoms, and the fact that the condition varies in different cases has led to the theory, extensively held in Germany (by Eulenburg and most other writers), that there are the two varieties of the disease, already mentioned, the "sympathetico-tonic," and the "sympathetico-paralytic" form, as they are sometimes termed. Dr. Latham, of Cambridge, who arrived independently at the same conclusion, that the attacks depend on vasomotor derangement, has suggested that the early symptoms of the paroxysm are due to spasm and the headache to dilatation of the vessels. According to these theories the malady is essentially one of the sympathetic nerves.

According to the other and alternative explanation of the disease, the primary derangement is of the nerve cells of the brain. Their function from time to time is disturbed in a peculiar manner, and the visible vasomotor disturbance is of secondary origin. The periodical derangement of function has been called, by a somewhat inapt metaphor, a "nerve-storm." This theory has been put forward and ably advocated by Liveing.

The sensory symptoms must depend on deranged action of the sensory centres in some part of the brain. They indicate a combination of arrest of action and of over-action in the nerve cells concerned. In the language of modern physiology, there is a combination of inhibition and discharge; the loss of sight, for instance, must be due to inhibitory arrest of action, the visual spectrum to discharge. We have already seen (p. 1085) that the same combination occurs in some attacks of epilepsy. The peculiarity in the disturbance of migraine is its deliberate character and its limitation to sensory structures. The uniformity of these symptoms in the same case is another feature that must be taken account of in any theory as to their origin. To explain them on the vasomotor hypothesis we must assume, first, an initial spasm of the arteries in a small region of the brain; secondly, that the contraction always begins at the same place; and, thirdly, that it can give rise to a definite, uniform, and very peculiar disturbance of function. There is no evidence of the truth of any one of these assumptions. As was pointed out in the discussion of the pathology of epilepsy, we are not justified in assuming that the state of the surface vessels and accessible arteries is any indication of the condition of those of internal organs. If it were, inasmuch as the recognizable vasomotor spasm is bilateral in almost all cases, even when the sensory disturbance is unilateral, we must assume a general contraction of the vessels of the brain. A general contraction could only cause a local disturbance of function by virtue of a local change in the functional tendency of the nerve cells. But if such local change is admitted, the need for the vasomotor mechanism disappears. Lastly, that vasomotor spasm can cause the "discharge" of nerve cells is also an unproved hypothesis, resting only on the fact that cerebral anæmia will cause convulsions. That it should cause a deliberate, uniform, and peculiar discharge is not only unproved, but in the highest degree improbable. In short, the difficulties in accepting the vasomotor explanation of the sensory symptoms are so great that it could only be admitted as a tenable hypothesis if there were no other explanation of the coincidence of the two phenomena. But we know that the vascular system is in a special way under the influence of the cerebral centres. An emotional blush and the pallor of fear are conspicuous examples of this fact. It is at least as easy to conceive that the vascular changes are the result of the disturbance in the sensory centres, or are the effect of associated derangement of vasomotor centres, as it is to consider that the vascular condition is the primary change. The vasomotor nerves are peculiarly sensitive to sensory impressions that are felt as pain.

It is as difficult to explain a primary derangement of the cells of the vasomotor and sympathetic centres as it is to explain that of the cells in the sensory centres in the brain. The functions of the pneumogastric are certainly affected secondarily in the later stage of the attack. Further, to assume, as is actually done, that similar nerve disturbance should be the result, in some cases of vascular spasm, in other cases of the opposite condition, vascular paralysis, is to go far beyond the bounds of legitimate assumption. No symptoms at all resembling those of migraine have been observed when the sympathetic is actually diseased. Headache is not a symptom even of a paralyzing lesion of the sympathetic. We have proof, in the symptoms, of the deranged action of sensory cells; we know that cells are susceptible of primary disturbance of function, and there is at present nothing to justify us in going beyond this derangement in our search for the primary morbid process. The peculiar character of the visual impressions affords strong reason for regarding it as the result of a tendency to functional derangement in the cells themselves. This opinion is supported by the fact that it may occur as an isolated symptom, and that it may be related to an actual visual impression. In the instance mentioned on p. 1180 the activity of the cerebral cells concerned in the image of the plate clearly determined the distribution of the abnormal action causing the colored spectrum. Such an occurrence can have been due only to a primary functional disturbance of the cells themselves, and that which we feel sure exists in one case probably exists in all.

What part of the brain is concerned in the production of the sensory symptoms? Dr. Liveing has suggested the optic thalamus as the part probably deranged, but this suggestion was based on theories of the function of the optic thalamus that we now know to be erroneous. It is indeed possible that hemianopia may depend on arrest of action in this structure (see p. 490), but it is equally possible that it is due to a similar condition in the half-vision centre in the occipital cortex. The thalamus is not in the path of those sensory impressions from the limbs that influence consciousness; this ascends by the internal capsule to the cortex, and the sensory limb symptoms therefore point also to cortical derangement. Of similar but clearer significance is the aphasia that sometimes occurs when the function of the left hemisphere is disturbed. Thus the sensory symptoms are probably due to a peculiar kind of functional disturbance in some of the nerve cells of the cerebral cortex. At the same time it is possible that the peculiar disturbance may spread to cells of lower centres, especially when sensations are felt in the nose, fauces, etc.

The cause of the headache is obscure. We know very little of the mechanism of this symptom in any condition (see p. 529). When the pain is opposite in side to the sensory symptoms we are obliged to assume that its seat is the cerebral hemisphere that is deranged, or the membranes covering the hemisphere. When it is local in situation—as at a point in one temple—and is on the same side as the sensory symptoms, it appears to be neuralgic in character, the result of some central disturbance referred to the periphery, and not necessarily corresponding in side to the morbid process causing it. It is more easy to conceive that the first kind of pain may be the result of vascular conditions than that the sensory symptoms are so produced. It should be noted, however, that the aspect of the face usually remains the same during both the initial sensory symptoms and the headache. At the same time it is probable that vascular derangement may intensify symptoms which it does not cause, and most nerve pains are rendered more intense by arterial pulsation.* Nor does the nausea and vomiting throw any distinct light on the mechanism of the pain. We have seen, in many affections, how widespread is the central representation of the pneumogastric, and that vomiting may result from organic disease in any part of the brain. In migraine it seems as though the nervous discharge of the act of vomiting afforded some relief to a morbid state of the nerve cells, but only when the disturbance has nearly run its course and is ready to subside.

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* Du Bois Reymond suggested that the pain is actually felt in the arteries, and is due to the spasmodic contraction of their walls; but until some other evidence is forthcoming of the occurrence of arterial pain, the theory can scarcely be regarded as admissible.

The hypothesis that the migraine is essentially a derangement of nerve cells of the brain enables us better to understand its relation to other neuroses. Epilepsy depends on a disturbance of function of a different kind, but it is intelligible that the two should occur in the same subject, and that intermediate forms of nerve disturbance should be met with.

Diagnosis.—From other forms of headache, that of migraine is distinguished by its sensory accompaniments, and, if these are absent, by its paroxysmal character, severity, and definite course. In malarial headaches the intermissions are shorter, and the cause is generally traceable. The pain of chronic brain disease is more or less constant; it may present remissions and even intermissions, but not the long intervals of migraine. The sensory disturbance in the limbs, and the affection of speech, may excite a suspicion of acute brain disease, but it is only in a first attack of migraine that the question will arise, and even then the brief duration of the symptoms, and their association with the characteristic visual disturbance, sufficiently indicate the nature of the attack.

A greater difficulty is presented by the diagnosis from minor epilepsy, in which the visual aura may closely resemble that of migraine. In most cases of epilepsy with a visual aura this is brief, lasting only a few seconds, while the visual disturbance in migraine lasts twenty minutes to half an hour. The fortification spectrum is suggestive of migraine rather than epilepsy, although not conclusive. The occurrence of a convulsion, of course, decides the question, but where the patient's account is all we have to guide us, we must make our diagnosis from the general features of the case, not forgetting that the one disease may unquestionably pass into the other, and that some attacks seem to be of intermediate nature.

A diagnostic difficulty of another kind arises in cases in which the subjects of migraine become affected with some other malady, and the symptoms of the former continue and complicate the latter. Thus one patient was attacked with Bright's disease; he had retinal changes and optic neuritis, and the intensity of the headache led to a diagnosis of intracranial tumor. But the pain was purely paroxysmal, and of the same character as that to which he had been long liable. The opinion that there was no organic cerebral disease was confirmed after death. In another case, a subject of migraine became affected with general paralysis of the insane, and the sensory disturbance that had preceded the attacks of headache occurred alone, and complicated the paroxysmal symptoms of the cerebral disease.

Prognosis.—The prospect of recovery from migraine, that is of the entire cessation of the attacks, is never considerable. It is best in the rare cases in which the malady is of short duration, and in which there is some removable condition in the general health or mode of life, which has distinctly contributed to the production of the malady. The longer the disease has lasted the less is the prospect of improvement, and hereditary tendency has also an unfavorable influence on the prognosis. In the second half of life the tendency to cessation is greater than in the first half. In no case, however, in which the disease is well established, is it likely that the attacks will altogether cease, but, on the other hand, in all cases there is a fair prospect that the attacks will be rendered less frequent and less severe by judicious and persevering treatment.

Treatment.—If any error in mode of life or defect in general health can be traced, the removal of this is the first and most essential step in treatment. The details vary in every case, but the most frequent conditions that need attention have been already indicated in the account of the causes of the disease. Of especial importance are increased rest, regularity in meals, attention to diet; whatever is known to induce a paroxysm should be carefully avoided. Hot, crowded rooms are especially injurious.

The special treatment consists first in the continuous administration of drugs, with the object of rendering the attacks less frequent and less severe, and, secondly, the treatment of the attacks themselves. The influence of drugs is singularly variable. That which does great good in one case will fail in another, apparently quite similar. The influence of bromide in epilepsy naturally leads us to turn first to this as likely to be of service in a malady that has so many features in common with that disease. In some cases it is certainly of service, but far less often than might be expected. It is most likely to be efficacious in the cases in which there is no change in the color of the face, or in which the face is flushed throughout an attack. In the majority of cases, and especially in those in which there is conspicuous pallor during the attack, the drug that has most influence is nitro-glycerine. Given regularly during the intervals, just as bromide is given for epilepsy, it has a striking effect in many patients, rendering the attacks far slighter and far less frequent, and occasionally stopping them altogether. It should be given twice or three times a day, after food. If taken when the stomach is empty it passes rapidly into the blood, and may cause brief cephalic discomfort, which, though not objectionable in itself, sometimes deters the patient from continuing the medicine. To avoid causing alarm, it is therefore desirable to begin with a small dose, $\frac{1}{150}$, $\frac{1}{180}$, or even $\frac{1}{200}$ of a grain. By far the most convenient mode of prescribing it is in the 1 per cent. solution in alcohol, which can be given in combination with tinctures or acids, but is decomposed by alkalies. A very useful combination is with tincture of nux vomica, tincture of gelsemium, and dilute phosphoric acid. If there is much dyspepsia it may be given with hydrochloric acid, and some liquid preparation of pepsine. The trisnitrate of bismuth may also be given with it. I have found such combinations of the liquid preparation of nitro-glycerine with other drugs far more useful than the administration of nitro-glycerine in tablets. It is not well to continue it during an attack; at the very onset a dose may be taken, but if this is not effective the medicine should be omitted till the attack is over; it seldom gives relief to the symptoms, and occasionally makes them worse. It is possible that the drug acts chiefly by periodically flushing the nerve centres with arterial blood, and so improving the nutrition and the function of the nerve cells. A purgative at the onset will occasionally cut an attack short in some patients, but more often it fails.

During the attack, absolute rest is essential, and is indeed impressed on the patient by the distress occasioned by activity. The recumbent posture is generally that in which the sufferer is most comfortable. All strong sensory impressions should be avoided. Although a dose of alcohol or of nitroglycerine will occasionally cut short a commencing attack, when the headache is developed, it is usually increased by drugs that dilate the vessels or excite the heart. Most relief is afforded to the pain by a good dose (thirty or forty grains) of bromide, and its effect is increased by the addition of five or ten minims of tincture of Indian hemp; this may be repeated every two or three hours. Twenty grains of chloral, or fifteen of butyl-hydrate (croton-chloral) usually makes the patient drowsy and easier for a time, but after an hour or two the pain returns in its former severity, and the duration of the attack does not seem to be lessened. The same is true of most other sedatives. A hypodermic injection of morphia often acts no better than other sedatives, but occasionally it does give great relief, and may end an attack that has already nearly run its course. When there is much mental depression during the attack, valerian and asafœtida have been found useful by Latham. Drugs that cause contraction of the arteries are almost powerless; all that a full dose of ergotin does is to lessen the throbbing intensification of the pain complained of by some patients. Strong tea and coffee are popular remedies, and occasionally give some distinct relief, which may also be obtained by a few grains of caffeine. Guarana (the powdered seeds of *Paullinia sorbilis*) has been introduced for the treatment of migraine. It contains an active principle, guaranine, which is identical with caffeine.* Three or four doses, of twenty or thirty grains each, may be taken at intervals of half an hour, or a single dose of sixty grains, or guaranine may be given in doses of one to five grains. It has, for the most part, disappointed the expectations that were raised by the praises at first bestowed upon it. Some relief is often given to the pain, but the relief is transient only, and it is doubtful whether guarana is really more effective than tea and coffee. Antipyrin, in ten- or fifteen-grain doses, has also been recently recommended.

Local applications sometimes afford slight relief—sedative liniments of belladonna, aconite, etc., or simple counter-irritation by a mustard plaster to the nape of the neck. Solid menthol is occasionally useful, rubbed on the skin for a few minutes where the pain is greatest, but it is too feeble an agent to have much influence on any severe pain. Sometimes relief is afforded by a hot bath of mustard and water to the feet, and in all cases it is well to keep the extremities warm.

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* Guarana contains twice as much of the alkaloid as tea, and five times as much as coffee.

Electricity is not often of service. Faradaism usually does harm. The voltaic current passed through the head occasionally gives transient, but rarely permanent, relief. Repeated galvanization of the sympathetic has been recommended in Germany as a remedial measure, one pole pressed deeply in front of the sterno-mastoid, the other held in the hand, and it is advised that when the symptoms of vasomotor spasm predominate, the positive pole should be placed over the sympathetic, and the negative when there is evidence of vasomotor paralysis (Berger, Horst). The value of the treatment is very doubtful.

HEADACHE.

Headache is a symptom of almost all kinds of morbid states of the system, and can scarcely ever be regarded as, in itself, a definite disease. Nevertheless, it is a symptom for which patients seek treatment more often than for any other of equally varied causation. Hence, it may be useful to indicate the chief varieties that are met with, and briefly to describe the symptom in its purely functional form. As an indication of organic disease it has been already described.

We know almost nothing of the structures in which the pain of headache is felt, or the mechanism of its production (see p. 529). The subject is one on which it is easy to theorize, but there are no facts that give to any hypothesis a considerable degree of probability. One conclusion is, however, suggested by the symptoms, and that is, that the seat of the pain varies considerably in different cases. Outside the skull are nerves that frequently give rise to pain, but all pain that corresponds to the course or distribution of nerve trunks must be regarded as a neuralgia and not as headache. Some diffuse headaches seem superficial in character, but if this is a trustworthy indication of their origin, they may still be due to processes not in the nerves, or the gray matter in which the nerves directly end (which is supposed to be deranged in neuralgia), but in higher centres of the cerebral hemispheres through which the superficial pain is perceived. More frequently, headache seems to be deeply seated, and the pain is referred to structures within the cranium. Of such structures the membranes, when inflamed, are the seat of intense pain, which is probably really produced in them, because other organic disease, such as a tumor, which is seated at the surface of the brain and involves the membranes, may also cause intense pain corresponding in seat to the disease. Whether pain is produced directly by morbid states of the cerebral tissue we cannot say. We are certainly not justified, however, in denying the possibility of this seat of pain (see p. 529.)

When pain is felt, in whatever part the nerve processes occur that cause it, the consciousness of pain arises during the activity of certain nerve cells of the cortex of the brain. In pain that is produced in the limbs, for instance, the cortical cells concerned are those that are related to, and receive impressions from, the fibres by which the sensory impulses pass from the periphery. The same relation, of course, obtains in the case of sensory impressions from the head itself—from the structures covering the head, from the bone, probably from the membranes. The pain of meningitis, for instance, if due to the irritation of the meningeal nerves, must be perceived by means of the activity of those cells in the cortex that receive the impressions from the membranes. Whether there is such a representation in the cortical cells, of the brain itself we do not know. Sympathetic nerve fibres accompany the arteries of the cerebral substance, and there is reason to believe that the functional state of the cortex influences the state of its arteries (as is the case in all other organs), and this must mean a relation of the cells of the cortex to the vasomotor centre; hence, it is quite possible that there may be a sensory representation of the cortex in the cortex itself, but of this we cannot feel so confident as of a sensory representation of the membranes. The phenomena of epilepsy seem to show that the central representation of the head, and perhaps of the brain itself, is connected in a special manner with the structures related to the mental state of consciousness. It is very common for an aura, felt first in the epigastrium or limbs, to seem to ascend to the head, and when it reaches the head consciousness is almost invariably lost. The aura is the result of the spread of the discharge in the brain, and the phenomena indicate that the central sensory representation of the head has a special importance, and it may be that it has a special susceptibility. But a functional derangement of the cortical cells in which the membranes are represented would, by itself, explain many forms of headache.

It may be held, indeed, that the primary derangement in headache is not of the highest sensory cells, but of the cells of lower centres, to which, for instance, the nerves of the membranes directly pass, but this view is less easy to reconcile with the difference between headaches and the true cranium neuralgias (in which the lower cells are probably concerned), and also with the resemblance often observed between the headache of organic and of functional origin.

Another mysterious factor in the mechanism of headache is the influence of the vessels. It is probable that mere vascular dilatation, passive or active, will cause pain;[*] it is certain that pain already existing is increased by even trifling mechanical congestion. We do not know how the vascular dilatation causes pain. There are no facts to show that the nerves of the vessels ever become sensitive. It is certain, however, that the intracranial pressure must be increased by the dilatation, since, however quickly the movement of the cerebro-spinal fluid may compensate for vascular repletion, this movement is due to the mechanical pressure from the vessels, and this pressure must act on the whole brain. We know how sensitive nerve fibres are to pressure; it is highly probable that their sensitiveness is vastly exceeded by that of nerve cells. If the sensory cells of the cortex, in which the cranial and intracranial sensitive structures are represented, are the most readily influenced of all the sensory cells, we can understand that headache should result from vascular repletion.

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[*] It is certain that pain results from mechanical congestion, but we cannot separate the effects of the state of the vessels and the increased amount of venous blood in the brain.

The frequency with which pain due to general causes is referred to the frontal region is very peculiar, and does not seem to be susceptible of any satisfactory explanation. It is occasionally a feature of the pain of organic disease, such as a tumor, situated away from the frontal region.

Varieties.—Toxæmic headache may be the effect of either acute or chronic blood states. Of the acute form, the typical example is that of fever, in which the pain is generally frontal, rarely occipital or general, and scarcely ever vertical or one-sided. In slight cases it may have a neuralgic character for a short time, and may be felt in one part of the head, as the temple, but this form is usually transient. The pain is commonly dull in character, but often very severe, and is rendered worse by stooping or by whatever causes mechanical congestion of the head. The deep-seated character of the pain suggests that it is referred to some intracranial structure. There is no evidence that it depends on any vascular state; the increase by mechanical congestion is a common feature of almost all nerve pains.

Many poisonous substances cause headache as one of their toxic effects, especially when the dose is small, or as an after-effect of large doses. Some of these poisons, such as nitrite of amyl and nitro-glycerine, dilate the vessels, and the pain may be in part or altogether the effect of the active congestion produced, but it is also possible that the pain is the direct effect of the agent on the nerve elements, and the congestion merely increases it. The inhalation of a small quantity of ether, as, for instance, when the air of a room is charged with it in moderate degree, generally causes headache, which seems to be the direct effect of the ether, since it is not attended by any sign of vascular distention.

Chronic toxæmic states often cause very severe headache, which may be quite intractable unless its cause is discovered. In uræmia, for instance, there is often intense headache, usually frontal and continuous, but with occasional exacerbations. It is sometimes increased by alcohol in a special manner, but this is a feature also of some other forms. The severity of the pain has many times given rise to an erroneous diagnosis of cerebral tumor in cases in which there is albuminuric optic neuritis. Headache also results from diabetes, and from chronic alcoholism, and from lead poisoning.

Congestive headache is often produced by mechanical hindrance to the return of blood from the head. It is generally frontal, but sometimes general. A tight collar, for instance, or rest with the neck so bent as to compress the veins, frequently causes slight headache of this character. The repeated mechanical congestion produced by cough is another frequent cause, and may even occasion the patient more distress than the cough itself. Mechanical congestion not only distends the veins but causes over-filling of the capillaries, and increases the amount of venous blood in the brain. Thus there are several factors at work to which the pain may be due. Active congestion of every kind also gives rise to pain very similar to that produced by passive hyperæmia, but more distinctly throbbing in character. The action of poisons that dilate the vessels has been already mentioned.

Gastric disturbance is another exceedingly common cause. The pain is most frequently occipital or vertical; now and then it is frontal. Sometimes there is slight pain of a neuralgic character; the severe neuralgic headaches associated with vomiting cannot, as we have seen, be ascribed to gastric influences. We do not know by what mechanism stomach disorder causes headache—whether it is by some influence on the cerebral vessels, or whether by absorption of some toxic material from the stomach or bowel—some morbid secretion, or product of the imperfect digestion of food, or altered bile. A purgative often relieves the pain, and old theories regarded the bile as an influential agent in the production of this and other symptoms, but when there is much bile circulating in the blood, as in jaundice, headache is usually trifling or absent.

Neuralgic headache constitutes one of the most common varieties. The subjects of neuralgia often suffer from continuous or occasional pain in the head, which may be felt in any part, sometimes general but more often local, and not distinctly paroxysmal. Such headache is especially common in hysteria. Many forms, such as the sensation of a nail being driven into the vertex, are intermediate between neuralgia and headache. A common cause of neuralgic frontal headache is the use of the eyes when there is hypermetropia. The strain in the ciliary muscle seems to be the immediate excitant. The pain goes off when the use of the eyes for near objects is discontinued.

Anæmic headache is met with in both common anæmia, especially in chlorosis, and also in cases in which much blood has been lost. In chlorosis it is especially intense. The pain is frontal or general; sometimes it is peculiarly intense at the back of the eyes, and is accompanied by a sensation as if the eyes were being dragged back into the head.* Neuralgic headache and true neuralgias of all kinds are also common under the same conditions.

Brain work and brain exhaustion are other common causes; sometimes there has been an amount of brain work that is absolutely excessive; in other cases the amount has not been large, but is excessive on account of the deficient nerve strength of the individual. Pain from this cause is often general, sometimes local, and then generally felt in the forehead. Occasionally a very intense form of headache results, especially where there has been an acute break-down from overwork. It may even be so severe as to cause a suspicion of meningitis.

In various conditions of nervous weakness, "neurasthenia," as it is now termed, headache is also common, and may have almost any seat or character. Many cases in young girls are really of this character, and are often associated with symptoms of hysteria; the headache is then called hysterical, but it is doubtful whether there is any closer association with that disease than the state of defective nerve strength which underlies both conditions. Many of the slighter continuous headaches associated with nervous weakness are largely maintained by attention, as is the case in even greater degree with the cephalic sensations presently to be described.

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* The fact that optic neuritis may result from anæmia is of great interest in connection with the occurence of headache, since it shows that the disturbance of the nerve elements may be more than functional, and suggests that their nutrition may be impaired in cases of apparently functional disturbance. It is noteworthy that in Bright's disease, when optic neuritis preponderates over retinal changes, headache is often severe, a circumstance that increases the readiness with which an erroneous diagnosis of intracranial disease may be made.

DIAGNOSIS.—The diagnosis of the form and nature of headache consists first in the search for any general condition on which it may depend. It is important to note the time at which the pain comes on, its relation to food, to mental work, and to other influences that may possibly be concerned in its causation. The general state of the patient must also be carefully investigated, and the effect of various influences in augmenting or reducing the pain. Anæmic headaches, for instance, are often relieved by the recumbent posture; those of congestive or toxæmic origin are generally increased by stooping or effort or mechanical congestion, but not always by simple recumbency, which, perhaps through the rest it involves, often lessens the pain, and does so, also, in cases of neuralgic nature. The seat of the pain is often suggestive, but too much weight must not be placed on this indication, since many variations are met with. The pain produced by acute blood states, such as fever, is generally frontal and deeply seated, and is increased by cough and movement. The forehead is also a frequent seat of neuralgic headaches; these are, however, generally superficial in character, and are often felt just over the eyebrows. Pain of gastric origin is also sometimes frontal. Pain in one or both temples is generally neuralgic, and so are all one-sided headaches. Occipital pain, deeply seated, is more often due to disturbance of the stomach than to any other cause. Vertical headache is seldom due to blood states; occasionally it is of gastric origin, but when very limited, it is usually neuralgic. General headache may be of almost any nature; it is often due to toxæmic conditions, anæmia, and conditions of nervous weakness. It must be remembered that blood states which, on account of their slight degree or other causes, do not produce actual headache, sometimes cause slight neuralgic pains.

TREATMENT.—The most important element in the treatment of headache is the discovery and removal, as far as possible, of the general condition on which it depends. The measures necessary for this object must vary with each variety of headache, and cannot here be even enumerated. Attempts to relieve the headache by direct treatment usually fail unless the cause is discovered and removed. It is important that in all cases except those in which there is evidence of anæmia, the patient should sleep with the head well raised, and with the shoulders raised as well as the head, so that there is no compression of the cervical veins by flexion of the neck. Most forms of headache, except those of neuralgic nature, are relieved by aperients, which probably act, partly by lessening the amount of blood in the cephalic vessels, and partly by improving the state of the blood. Diuretics are useful in many toxæmic headaches. The treatment of the neuralgic forms is similar to that of pure neuralgia.

Headache of every kind is often relieved, although seldom removed, by external applications that sting the skin. Chloroform, or any stimulant liniment, will answer the purpose, provided evaporation is prevented by an impermeable tissue. Bisulphide of carbon has been recommended for the purpose, but it does not seem to have any special value. Mustard plasters to the back of the head are useful when the pain is occipital. Slight neuralgic pain about the forehead is often removed by the application of menthol, which is especially useful when such pain is just enough to prevent sleep; after the menthol has been rubbed on, the patient will often sleep, and wake up free from pain.

Sedatives are very uncertain in their influence. Opium and morphia are seldom useful, and often do more harm than good, in consequence of the indirect effect of the constipation that is produced. Gelsemium and Indian hemp frequently lessen the pain, the former chiefly in neuralgic forms about the front of the head, the latter not only in neuralgic, but in anæmic, and also other ill-defined forms of headache. Bromide of potassium may be given in addition to the Indian hemp, if there is restlessness, irritability or insomnia, but has not alone much influence on the pain. Butyl-chloral is occasionally serviceable. It must be remembered, however, that these palliative measures should only be regarded as adjuncts to the causal treatment.

HEAD PRESSURE AND OTHER CEPHALIC SENSATIONS.

It is common for patients to suffer from various more or less distressing sensations in the head, which have not the character of actual pain. These sensations are much more frequent in men than in women. They occur chiefly during the first half of adult life, between twenty and forty, but are sometimes complained of by lads of sixteen or eighteen, and occasionally by persons in the second half of life, at fifty or sixty. In late life they are relatively more common in females, coming on sometimes after the cessation of the menses, at the "climacteric period." Brain-workers are chiefly liable to these sensations; they are seldom met with among those who earn their living by manual labor, and hence are rarely complained of by hospital patients. Many of the sufferers are the subjects of pronounced hypochondriasis, who attend to their sensations, and often complain of other kinds of nerve disturbance. Excessive brain work is often the distinct exciting cause of the symptoms; in other cases they succeed a definite headache, of short or long duration, severe or slight. I have known the sensation to follow a blow on the top of the head. It is sometimes associated with a gouty state of the system.

The sensations vary in different cases, but by far the most frequent is a sense of pressure, sometimes trifling, sometimes as if a heavy weight were on the head, and occasionally as if the top of the skull were being driven in. Many patients describe the sensations in language that is clearly exaggerated, so that it may not be easy to ascertain the actual intensity of the sensation, but it is certainly often very distressing. It may be called "headache" or "pain," but however intense it is, inquiry shows that the sensation has not the character of actual pain, either acute or dull. Its common seat is the vertex and top of the head, but it is sometimes felt at the back of the head, less commonly in the forehead. One patient described the sensation as a feeling as if there were an iron band round the back of the head. Sometimes it is lateral, in the parietal region or the temples, "as if the sides of the head were being driven in." Much less common is the opposite sensation, a feeling of expansion, as if the bone of the skull were being pushed out or up. A sensation of heat or burning, not amounting to pain, is sometimes described. A vague sense of fullness is not uncommon, and it is sometimes accompanied by a sense of throbbing; in other cases, again, the feeling is said to be as if the skull were empty. Various other sensations are occasionally described, which the patient, in default of adequate terms, describes by some simile beyond the range of ordinary experience, such as "a feeling as if the brains were being stirred up with a stick," or "as if red-hot coals had been placed on the brain," or "as if the head were being alternately opened and shut." Occasionally, some superficial sensation of tingling, creeping, etc., is complained of in some part of the head, sometimes symmetrical. The sensation is often uniform in the same case, and may continue unchanged for many years.

Less commonly the patient has first one sensation and then another. The sense of pressure may seem, when most intense, to give rise to dull, actual pain. The discomfort is commonly increased by brain work, and often by any unpleasant emotion, while it may be unnoticed when the mind is occupied by some agreeable subject. It is often only observed when the patient is indoors and is unfelt when he is out in the open air.

The chief agency in the production of these sensations is certainly the mental state of the patient. There is probably at the outset some actual sensory impression, often some headache, and the attention is constantly directed to the part, with the result that the patient perceives sensations which, under normal circumstances, would be unperceived. Nerve impulses, in health unnoticed, must be continually passing from all parts to the centres, and they may be readily perceived if attention is directed to them. A person imagines that he is unconscious of his body and limbs, but let him direct his attention to any part, and in a few moments he will be conscious of a distinct sensation in it, and with especial readiness of sensations in the head. If the reader will direct his attention to the vertex, he will probably soon be able to detect a distinct sense of pressure there, especially if he is fatigued or has been engaged in mental work. It is easy to understand that such a sensation may be increased, by constant attention, to an unpleasant and even distressing degree, especially if some real sensation originally "drew" the attention to the part, and if the individual is in feeble health, or of a nervous temperament. The physical side of "attention" is a lowering of resistance in nerve cells, so that they are more readily aroused to activity, and their activity is greater than normal. Thus the ultimate result may be the same as if the afferent nerve impulses were increased in energy, and a sensation is produced by nerve processes that should be unperceived. The condition may be described as a "receptive dysæsthesia."

Rarely, when the sensation is at the worst, there is slight tenderness of the scalp. Although the sensations are usually constant, in exceptional cases they are paroxysmal, and then may be accompanied by some general nervous excitement and insomnia. It is common for the patient to say that sleep does not refresh him. There is occasionally, also, a little mental confusion. An inability to concentrate the mind on a particular subject, and a little consequent failure of memory, is often described, but is merely the result of the engrossing sensation which obtrudes itself on the mind and prevents attention. The sensations are sometimes distinctly increased by stimulants. Very rarely there is slight flushing in the face during paroxysmal sensations, but this is not common.

Some of these sufferers complain at times of other slight functional nerve disturbance. An actual headache may alternate with the sense of pressure, etc. In other cases there are occasional sensations of giddiness, almost always vague and slight, and perhaps sometimes scarcely real, since the patient looks out for them. Often it is merely a sensation of the "legs giving way."

The treatment of this condition is a matter of very great difficulty. Sedative drugs have little influence upon it, unless they are given in large doses, and then their effect is transient. This fact is in harmony with what has been said of the probable pathology of the affection. As long as the influence of attention is at work, the patient's distress continues, and the mental cause is beyond the influence of drugs. The sufferer cannot, by a deliberate effort of the will, ignore the sensations that distress him. The only method of treatment that is effective is to make the patient realize the unimportant nature of the sensations, and try to neglect them by directing his attention to other subjects. While it is wise for him to avoid whatever greatly increases the sensations, he should persistently endeavor to "snub" them rather than to cultivate them. When this effort is made and maintained, the discomfort slowly ceases to trouble the patient, and at least is no longer noticed. Unfortunately, the mental constitution of some patients makes it impossible for them thus to ignore their sufferings; they continue to attend to the sensations, doubt the assurances of physicians who assert the unimportant nature of the sufferings, and seek the advice of others, placing most trust in those who pander to their apprehensions by the assurance that the sensations indicate some grave disease, such as congestion of the brain. Many a patient of this class may be truly said to be suffering from a *morbus medicorum*, and his best chance of recovery is to keep away from doctors altogether. Of course any conspicuous defect in general health should be put right; a gouty diathesis should be treated; distinct nerve weakness may require tonics; and if there is vertigo or insomnia, bromide with small doses of Indian hemp gives some relief.

FACIAL HEMIATROPHY.

Facial hemiatrophy, as its name implies, is a wasting of one side of the face, in which, however, the bones and subcutaneous tissue suffer more than the muscles. The precise relations of the disease are uncertain. It is commonly classed with exophthalmic goitre and migraine, but it is very doubtful whether this is its true position. Many facts suggest that its closest alliance is with organic disease of the fifth nerve. An instance of the affection was first recorded by Parry in 1825, but the disease only received attention after it had been described by Romberg thirty years later. It is a very rare disease, and is of small practical importance; it involves no danger to life or health, and cannot be influenced by treatment.

Causes.—The disease generally begins in early life, sometimes in childhood, sometimes in the second decade. It has been known to commence as early as two or three years of age. In a few instances it has commenced in adults. A considerable disparity in size between the two sides of the face is sometimes congenital, but such cases are commonly distinguished from those that commence after birth. Females seem more liable to the affection than males. Most of the cases have been isolated, but in one recorded case, an aunt of the patient had suffered from the same disease. In early life it has generally come on without any exciting cause, but in a few cases in adults the affection has succeeded some morbid process, which may or may not have had to do with the subsequent wasting. In one case, for instance, it followed a blow on the outer angle of the orbit; in other cases it has succeeded acute rheumatism or some other acute disease. In one very remarkable case the affection commenced on the right side of the face after an attack of scarlatina with diphtheria at six, and an attack of typhoid many years afterward was followed by distinct atrophy of the left side.* In many instances early pain in the corresponding side of the head suggested some local morbid process, possibly in the fifth nerve.

Symptoms.—A gradual diminution in the bulk of one side of the face has been the first indication of the disease in many instances. In some, however, the atrophy has begun at one spot, commonly on the cheek, at which the skin has been observed to become thin and pale from loss of pigment, and depressed from the loss of the subcutaneous tissue. The hairs drop out in the affected parts and the sebaceous follicles waste. The condition has gradually spread, and has involved the bones. In the cases that begin in early life the ultimate difference between the two sides of the face has been greater than in those that developed later, a hindrance to growth increasing the effect of the wasting, so that it is chiefly in these cases that the disparity in the size of the bones is considerable. In these cases, also, the usual growth of hair does not take place on the face, although there may not be much difference in the anterior part of the scalp. The ultimate condition in well-marked cases is very striking. The appearance of the patient is as if the face were made up of two halves from different individuals. One side may have the fullness of youth, and the other the furrows of age, while the contrast in character is intensified by the difference in size. On the affected side the bone of the forehead and cheek is considerably smaller than on the other side; the skin covers the bone closely and so presents depressions without counterparts on the opposite half, while a vertical furrow usually marks the transition from the affected to the normal side. The side of the lower jaw may be only two-thirds the length of its fellow, and is diminished in depth, so that the face appears much shorter. The cartilage of the nose shares the wasting, and in the cases that begin early, it may be so small on the affected side as to render the nose altogether unsymmetrical, the tip deviating toward the smaller side; the teeth are sometimes small, and may drop out. In spite of the wasting of other tissues, the muscles seem to undergo little or no change, either in size, in voluntary power, or electrical excitability. They may be more conspicuous than normal in consequence of the wasting of the other tissues. Rarely, however, there has been some wasting of the muscles of mastication, on the affected side. In a few instances the corresponding half of the tongue and of the palate has also been wasted. Sensibility is unaffected. It is doubtful whether there is any alteration in the size of the arteries and veins. The ultimate wasting of the skin may be very great; it is said that a fold of the skin on the affected side may be only one-tenth the thickness of a similar fold on the other side. The temperature is usually unchanged, but in the early stage it has been found to be slightly lessened. The organs of special sense on that side are not changed, but the eye often appears retracted in the orbit in consequence of some wasting of the orbital tissue. The secretion of saliva and of tears is not affected. As a rule, there is no wasting elsewhere, but a case of bilateral atrophy has been already mentioned, and Virchow has described a typical hemiatrophy of the face which was associated with an atrophy of the skin and subcutaneous tissue of part of the back and arm on the same side. In the back, it extended from the fourth to the seventh dorsal spine, in the arm affected chiefly the radial side of the forearm. A curious case of local wasting of all the subcutaneous tissues in small areas on the back and arm has been mentioned on p. 359.

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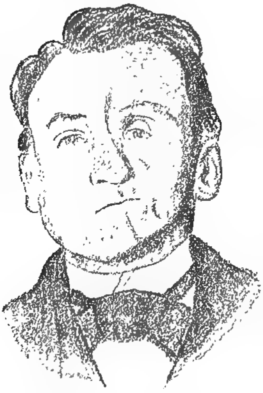
* Wolff, *Virchow's Archiv*, Bd. 94, p. 3.

FIG. 326.



Facial hemiatrophy (*Schwann*).

The affection, beginning commonly in early life, progresses steadily for some years and then becomes stationary and remains so for the rest of life. The illustration is the case of a man named Schwann, who exhibited himself in this and other countries a few years ago; he was then aged forty; the atrophy began in childhood, and his case was described by Romberg in 1851.

PATHOLOGY.—Two theories have been held regarding the nature of facial hemiatrophy. According to one it is due to derangement of the trophic and vasomotor nervous influence; according to the other, it is a primary local wasting of the subcutaneous cellular tissue, in consequence of which the elastic skin compresses the vessels and so causes wasting of the bone on the one hand and of the elements of the skin on the other. The latter hypothesis involves a mechanism the efficiency of which is not only unproved but improbable. Wasting of the corresponding side of the face has been described by Seeligmüller in a case of injury to the cervical sympathetic, but has been absent in most cases of the kind.* The most significant fact of this condition is the limitation of the atrophy to the parts supplied by the fifth nerve. The significance of this limitation is increased by the fact that other symptoms of deranged function of the nerve have been observed at the onset. Pain has been noted in several instances, and in one remarkable case of slight but distinct atrophy, there was spasm in the muscles of mastication on that side, greatly increased by attempts to open the mouth.† In another case, the affection was limited to the parts supplied by the two lower divisions of the nerve, the first being free.‡ Thus it seems, on the whole, probable that the disease is due to some alteration in the trophic influence of the fifth nerve.

The only condition with which the affection can be confounded is that in which there is a congenital difference between the two sides of the face. In this, however, the difference is slight, and on the smaller side there is not the difference in the growth of the hair or the color of the skin seen in most cases of hemiatrophy.

There is no prospect of improvement except, perhaps, in some of the rare cases that come on in adult life after injury or are accompanied by distinct symptoms of disease of the fifth nerve. The treatment of these is that of the disease of the nerve. In the cases of the ordinary type, treatment is useless.

EXOPHTHALMIC GOITRE.

Exophthalmic goitre is a mysterious malady manifested by three sets of symptoms: enlargement of the thyroid, prominence of the eyes, and disturbance of the vascular system. These vary in relative degree in different cases, and any one of them may be inconspicuous. The disease was first fully described by Graves in 1835,§ who, while describing all the symptoms, duly recognized the predominance of the cardiac disturbance. In Germany the first account of the affection was given in 1840, by Basedow. The name has been the subject of one of those unbecoming contests inseparable from a system of cognominal nomenclature; the affection is often called in this country "Graves' disease," and in Germany "Basedow's disease." Many other designations have been proposed—struma exophthalmica, cardio-thyroid exophthalmos, etc., no one of which has obtained currency or can claim special exactness.

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* Much weight has been placed on a case described by Brunner, in which the condition was associated with prominence of the eyeball and pallor of the skin, supposed to be dependent on an affection of the sympathetic. But the case stands almost alone.

† Penzolat, *Münch. Med. Wochenschr.*, *1886*, No. 14.

‡ Repmann; the case was published in a Russian (Moscow) paper; it is abstracted in *Virchow's Jahresb.*, *1885*, ii, 508.

§ Cases had been published earlier; in 1816 in the *Medico-Chirurgical Journal* anonymously; in 1818 by Demours (thyroid and eyes only); by Parry in 1825 (posthumous publication of a case observed in 1785); and by Adelmann in 1828. See Wickham Legg, "Note on the History of Exophthalmic Goitre," *St. Barth. Hosp. Rep.*, vol. xviii, p. 7.

Causes.—Women are far more prone to suffer than men, the ratio being about five to one. The disease may occur at any age, from two to sixty, but is met with chiefly in adult life, between fifteen and fifty, and more cases begin between twenty and thirty than in any other decade. The rare cases that have been met with during childhood have not, as a rule, been well marked. Although not usually inherited, instances are occasionally met with in which a family tendency to the disease is very clear and sometimes very strong. In several instances two sisters, or mother and daughter, have suffered; in one a mother and a son aged eight, in another an aunt and three nieces (Cheadle). Eight out of ten children suffered in various degrees and at various ages in a family observed by Oesterreicher. In these and other analogous instances some of the cases were so slight that they would probably have escaped recognition had not attention been directed to them by the other cases. In a few instances a more general neuropathic inheritance has been indicated by insanity, epilepsy and hysteria, in near or distant relations. Thus, I had at one time under my care two sisters, one for epilepsy, the other for severe exophthalmic goitre. I have seen lymphadenoma in one sister and exophthalmic goitre in another, an association that derives some significance from the fact that enlarged lymphatic glands are not very uncommon in this disease. The causes of ordinary goitre seem to have little influence in producing the exophthalmic form,* and recovery has actually followed removal to a place where goitre was endemic (Trousseau). General debility and anæmia are unquestionably powerful predisponents. In many cases the first symptoms have shown themselves after a period of constitutional weakness due to some distinct and adequate cause, such as exhausting discharges, especially hemorrhage, weakness after childbirth or abortion, etc. But previous weakness is not essential; the disease may come on in a person who otherwise appears to be in perfect health. It seldom develops during pregnancy. There is not usually any initial disturbance of menstruation, but sometimes amenorrhœa precedes the onset. In some instances organic heart disease has preceded the malady, but the association of the two is not frequent, and its significance is uncertain.

No immediate cause is so frequent as depressing emotion—sudden terror, or prolonged distress. The symptoms have been known to follow fright with great rapidity, and to become distinct after a few days. Many cases occurred in Alsace and Lorraine after the Franco-German war. Cold, perhaps, may excite an acute development of the symptoms in a predisposed person.* Among other rare causes that have seemed influential may be mentioned stimulating climatic influences acting on some peculiar constitutional state, injuries to the head, alcoholic excess, syphilis, various acute diseases, scarlet fever, pneumonia, rheumatism, diarrhœa, and severe exertion, such as mountain climbing.

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* In India it is said that, although endemic goitre is common, the exophthalmic form is practically never seen among the natives (Brockman, *Trans. Oph. Soc., 1886*, p. 117).

SYMPTOMS.—The onset of the disease is usually slow and gradual. In rare cases it is acute, and these may run a rapid course, or the acute onset may subside into a chronic stage. Usually the disease, when once developed, is permanent. Occasionally one or two slight attacks, perhaps recognized only afterward as of similar nature, may occur and pass away, months or years before the onset of graver symptoms.

The three sets of symptoms, in eyes, thyroid and heart, do not usually commence together. Their order varies, but it is common for the cardiac disturbance to lead the way. Palpitation, more or less frequent, troubles the patient for weeks, or months, or even years, before the other symptoms are added. The enlargement of the thyroid and prominence of the eyeballs often come on together.

Heart.—The characteristic condition of the heart is excited action, shown by increased frequency and increased force of its contractions. The degree of excitement varies from time to time, and is readily increased by emotion or exertion. At first, between the exacerbations, the action may be normal, but gradually the subsidence becomes less complete, and the over-action correspondingly persistent. The increased rate of action is sometimes trifling, and the pulse does not exceed 90–100 per minute. More often the rate is higher, 120 or 140, even when tranquil; sometimes 150 or 160, and under excitement it may rise to 180 or 200. It is usually regular, but often small; there is not time, apparently, for the ventricle to receive the due quantity of blood, and its systole is probably incomplete. Thus the circulation is not accelerated in the same degree as is the action of the heart. The increased force of the heart's action is shown by the impulse, which is unduly sharp, and felt over a larger region than in health. The area of cardiac dullness is sometimes normal when the area of impulse is increased. The action of the heart resembles that of a person under considerable nervous excitement or after exertion. Usually, after a time, there are signs of enlargement of the heart. The impulse extends outward and becomes distinctly diffused, and the cardiac dullness is increased to the left. These signs of dilatation usually preponderate over any indications of hypertrophy. The dilatation is secondary in time, and doubtless, also, in origin, to the over-action of the heart. The sounds are usually increased in loudness; Graves mentioned a case in which they were audible four feet from the patient. Occasionally they are accompanied only by that murmurish roughness that is common in excited hearts; more often, however, there is a distinct systolic murmur, which may be loud. It is heard over the whole heart, loudest at the base and great vessels, but loud, also, at the fourth interspace, and audible at the apex. The basic murmur is apparently produced at the arterial orifices; that heard over the ventricle and up to the apex is probably often a ventricular murmur, produced in the blood within the dilated heart. In many cases, however, the murmur extends outside the apex, and has the character of that of actual mitral regurgitation, which is probably the result of the dilatation which the mitral orifice suffers with the ventricle. Slight mitral endocarditis has been occasionally found after death, and is perhaps not rare; it is more likely to be due to the mechanical strain on the valves than to a state of the blood. When there is no murmur at the apex, the first sound is often loud and sharp. The impulse of the heart may be attended by a thrill, which has not, however, any special significance. As the disease advances, the paroxysmal attacks of palpitation increase in severity, and are often accompanied by considerable dyspnœa.

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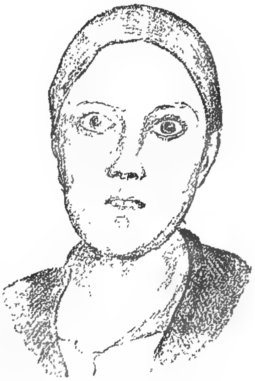
* See, for instance, a case recorded by Dr. Bristowe, *Trans. Oph. Soc.*, *1886*, p. 46.

The *arteries* present increased pulsation and are dilated. The condition is due in part to the force with which the blood is driven into them by the heart, but in part, also, to a lessened tone of the vessels. The pulsation is especially conspicuous in the carotids and thyroid arteries, and often in the abdominal aorta. The medium-sized arteries frequently present relatively less pulsation than those of larger size, but that the exalted state of the circulation extends through the whole arterial system is shown by the fact that pulsation may sometimes be seen in the arteries of the retina. A systolic murmur may almost always be heard in the arteries of the neck, and sometimes in other arteries, even in the femorals. It is often very loud, and is absent only in rare cases. A venous pulse has occasionally been observed in the neck, and even in the arm, probably due to the influence of the movement of an artery on the flow through an adjacent venous trunk.

The *thyroid* usually enlarges after the cardiac symptoms have existed for some time. It is a slow, painless, soft enlargement, commonly uniform, sometimes greater in one lobe than in the other, rarely confined to the isthmus. When one lobe is the larger this is commonly the right. The superficial veins over it may be dilated; the arteries of the thyroid always pulsate strongly; sometimes pulsation may be detected in the gland itself, and in it there is often a distinct arterial thrill and a loud, blowing, systolic murmur, rarely musical, still more rarely double. The degree of enlargement is not often extreme; seldom equal to that which is common in ordinary bronchocele. It varies somewhat in size from time to time, being greater, as Graves noted, during the attacks of palpitation. Diminution in size is often accompanied by an increase in firmness. In many cases the enlargement is only trifling in degree, and it is occasionally absent altogether.

Eyes.—The prominence of the eyes, like the enlargement of the thyroid (with which it often coincides), commonly succeeds the cardiac overaction. In rare cases it has been the first symptom. It is usually equal in the two eyes, but occurs first and remains greatest in one eye, more frequently in the right. It is very rarely confined to one side, and then the thyroid is usually most enlarged on that side.* In degree it may be so slight as to easily escape notice, or it may be so considerable that when the patient looks straight forward the white sclerotic is visible above and below the cornea. It may be extreme in degree, so that the insertions of the recti may be exposed, the movements of the eyes limited, and so that the lids cannot meet over it even in sleep. It is said that the eyeball may even be dislocated from the socket and may have to be replaced with the finger. The degree of prominence varies from time to time, with the over-action of the heart. When it is greatest pain may be felt in the eyeball. It seems probable that there is, in some cases, in addition to the prominence, an actual increase in the size of the globe. Its diameter has been estimated to be one-tenth greater than normal (Neumann). Vision, as a rule, is unaffected. In rare instances myopia seems to have developed, probably from an increase in the size of the globe. The pupils are normal, as a rule, but inequality in size and slight irregularity in form have been met with. Conjunctivitis, opacity of the cornea, and even sloughing have been seen in rare cases, apparently in consequence of the imperfect protection of the prominent eye. Œdema of the lids is occasionally present; I have seen it considerable in degree and associated with œdema of the conjunctiva, in a case in which the prominence was slight. The ophthalmoscopic appearances are usually quite normal, beyond the arterial pulsation already mentioned; œdema of the disc is occasionally met with; I have seen one instance of this. Its occurrence is not related to œdema outside the eyeball. Atrophy of the optic nerve has been observed by Emmert.

FIG. 327.



Exophthalmic goitre; early stage. Slight enlargement of the thyroid; considerable prominence of the eyes.

Another remarkable symptom connected with the eye, first noted by v. Graefe, is that when the eyeball is directed downward, the upper lid does not follow it as it does in health, but remains in a state of spasmodic elevation (Figs. 328 and 330). The descent of the lid on closure of the eyes is not interfered with. This symptom may be present when the prominence of the eyeball is slight. It is not a constant symptom, and is often absent even when the exophthalmos is considerable, and, it may be, to the end of fatal cases. It is commonly equal on the two sides, but may be unequal. In some cases, in which there is no absolute defect in the descent of the lid, its downward movement is hesitating and lags a little behind the eyeball.

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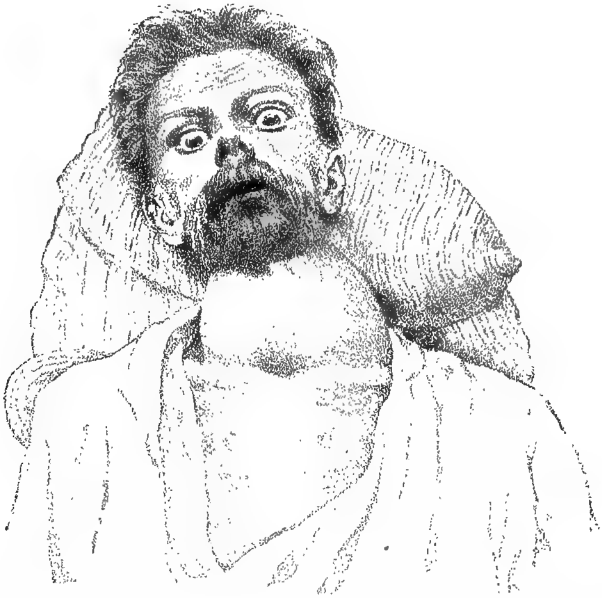
* To this rule a remarkable exception has been recorded by Burney Yeo, in which great enlargement of the right half of the thyroid coincided with prominence of the left eye, and when right exophthalmos came on, the left lobe of the thyroid enlarged (*Brit. Med. Journ.*, *1887*, i, 320).

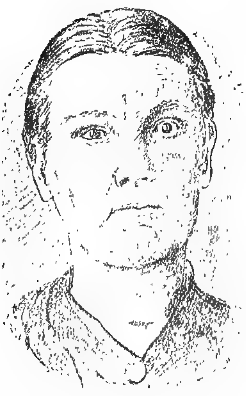
With or without a defective descent there may be (as Stellwag first pointed ut) a slight retraction of the lid so that it is habitually higher than normal; sometimes this is observed on one side only, as in Fig. 329. Retraction of the lower lid has been very rarely observed.* Occasionally there is some weakness of the ocular muscles, especially of the internal recti, and more extensive ophthalmoplegia has been observed (see p. 1206).

FIG. 328.



Exophthalmic goitre. Defective descent of the upper lid on looking down. (*After Wilks.*)

FIG. 329.



Exophthalmic goitre, early stage. Retraction of the left upper lid.

Of the three chief symptoms of the disease, the cardiac disturbance is the most constant. It is, perhaps, absent in rare cases, but, on the other hand, is often alone obtrusive. The patients may suffer from persistent frequency of pulse, occasional severe paroxysms of palpitation and dyspnœa, and may complain of nothing else. There may be, indeed, nothing to be found except the over-action of the heart and pulsation of the arteries, but in most cases careful examination detects a slight enlargement of the thyroid and a slight prominence of the eyes. The latter may not exceed the degree that is met with as a normal condition, but the friends of the patient may recognize it as unnatural.

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* By Dr. Hill Griffith, *Trans. Oph. Soc.*, *1886*, p. 61.

If the cardiac disturbance makes rapid progress, the patient may die before the other symptoms attain such a degree as to attract attention. When the enlargement of the thyroid and exophthalmos precede the derangement of the heart, they increase very rapidly after the latter has developed.

The general health of these patients usually suffers. Anæmia is frequent, although not invariable. Menstruation is irregular and sometimes ceases. Loss of flesh occurs, but is less marked in the face than elsewhere. Pyrexia is frequent in the later stages of the disease, usually moderate, from 1° to 3° F., but it is inconstant; in many cases the temperature is perfectly normal throughout. There is sometimes a subjective sense of heat without actual fever. Profuse sweating is an occasional symptom, and is especially common in acute cases. The appetite is sometimes deficient, sometimes capricious.

Other symptoms on the part of the nervous system are often met with. A sense of throbbing in the head, and a pulsating noise in the ears, occasionally complained of, are doubtless due to the vascular condition. Headache is sometimes troublesome, and may be one-sided. There is often insomnia, and sometimes transient vertigo. Mental depression or irritability is very frequent.

Another very common symptom, which is sometimes described as a complication, but deserves, on account of its frequency, to rank as a symptom of the disease, is muscular tremor. It varies in character, but is usually a rather coarse, somewhat jerky tremor, occurring on movement only. In some cases it is so regular as to resemble that of paralysis agitans; in others it is so irregular as to remind the observer of chorea. When regular, it is a little more frequent in time than that of paralysis agitans.* It may be general or partial, and has been known to be unilateral when the goitre and exophthalmos were also unilateral.

Complications.—Many cases have been recorded in which there was also glycosuria. The change in the urine has followed in some cases, and preceded in others, the special symptoms. In one instance of this complication all the symptoms followed a blow on the head (Brunton). The sugar has been known to lessen when the other symptoms improved. Intermittent albuminuria has been recorded by Begbie, and observed in one case by myself. Increased secretion of pale urine is common, and has been observed to precede paroxysms of palpitation and dyspnœa.

Enlargement of the lymphatic glands sometimes occurs in the course of the affection, resembling that of lymphadenoma, but moderate in degree, and not progressive. It may develop rapidly, and slowly subside. I have seen three very marked instances of this complication; one was a severe and fatal case of exophthalmic goitre in a girl whose sister suffered from lymphadenoma. Enlargement of the tracheal and bronchial glands has been observed by G. de Mussy. Among recorded cutaneous complications are vitiligo (Raynaud) and bronzing of the skin (Begbie). It is said by harcot that there is commonly a diminution in the electrical resistance of e skin.*

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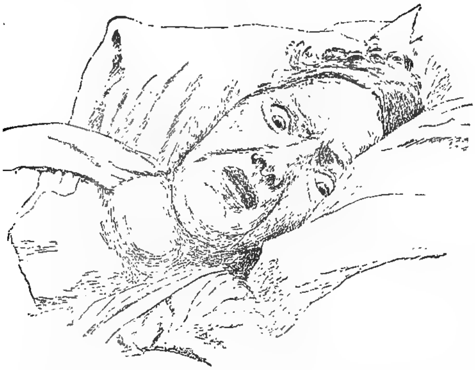
* According to Marie, 8–9½ per second; paralysis agitans 5–6 per second; but, as we have seen, that of paralysis agitans varies much.

FIG. 330.



Exophthalmic goitre. Acute mania (fatal). (*From a photograph.*)

An important occasional complication is mental derangement. We have seen that slight mental change is common; the derangement now referred to amounts to actual insanity. Its form varies: it may be melancholia, or simple mania or recurrent mania, or even general paralysis of the insane (Savage). Mania is the most common condition, and may be the cause of death. Fig. 330 represents the aspect of woman suffering from acute mania that came on in the course of exoph-ıalmic goitre and was fatal in a few weeks. Hysterical symptoms are also ommon in this disease. The hemiplegic weakness and altered sensibility bserved in some cases were probably of this character.

The tremor already mentioned has amounted in some cases to a degree of lonic spasm or choreoid movement that must be regarded as a complication. n a few cases in childhood or youth, true chorea has been supposed to xist. In one instance this was probable, since the movements increased or a time, and then subsided. In the other cases, however, the spasmodic isturbance persisted, and its nature is therefore doubtful.

Many other kinds of functional derangement of the nervous system, esides those already mentioned, have been observed to coexist with xophthalmic goitre. Epilepsy, neuralgia, and migraine in typical form, ave been met with. The paralysis of the ocular muscles sometimes goes n to complete ophthalmoplegia, and is then more than a functional affecion; it seems to be due to degeneration of the muscular fibres. A very emarkable example of these nervous complications has been recorded by Bristowe.† The symptoms of exophthalmic goitre were followed by almost omplete external ophthalmoplegia, and this by complete right hemianæshesia (including the special senses), epileptic fits, palsy with rigidity of the ight side, hemorrhages from the ears, and persistent pyrexia. The mystery of the origin of the symptoms remained unsolved at the post-mortem examnation, which revealed no lesion of the nervous system.

Many symptoms occasionally met with are apparently due to vasomotor listurbance. Sweating has been already mentioned; in a case recorded by Money a copious acid sweat broke out on the central parts of the face during attacks of palpitation and extreme frequency of the pulse.* Diarrhœa is sometimes troublesome, and is thought to be a kind of intestinal sweating. Occasionally there is an abnormal flow of tears. Local œdema in the limbs, local congestion of surface vessels, atrophy of the mammæ and other parts, some skin diseases, and even gangrene, have all been regarded as manifestations of vasomotor disturbance related in some obscure manner to the primary disorder.† Indications of chronic organic diseases of the central nervous system are very rare.‡ Bronzing of the skin existed in one recorded case.§

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* *Gaz. des Hôp.*, *1885*, No. 13.

† *Brain*, No. 31.

Course and Varieties.—The onset is usually gradual, and the course of the disease is prolonged through several years. But the differences in course are extreme. Occasionally the onset is acute; marked symptoms may develop in the course of a few days, commonly after some psychical disturbance. The symptoms may even pass away in a week or two. Such cases occur especially in families in which there is a hereditary tendency to the disease. Thus a boy, aged eight, the son of a woman who suffered from the disease, after a disappointment at school, was seized with palpitation and profuse sweating; the next day the thyroid was large, the eyes were prominent, and the pulse was 180. After two days more, the symptoms gradually disappeared, and in ten days he was well (Sobrig). Such an acute case may, however, run a rapidly progressive course and has been known to end in death at the end of six weeks (Bamberger). Cases so rapid are extremely rare. Death at the end of six months is uncommon, and, on the other hand, in many cases, the disease lasts for five or even ten years. The acute onset may subside into a chronic stage, similar to that of the cases that commence gradually.

The course of the symptoms is also variable. Considerable and prolonged remissions are frequent, and sometimes actual intermissions occur, of such duration and completeness that the patient, in effect, suffers from several distinct attacks. I have known a patient to have three such attacks at intervals of several years. Permanent recovery may also occur. It is most frequent in the cases that develop rapidly, and in which the cardiac symptoms preponderate over those in the neck and eyes. What proportion of cases recover is uncertain. It has been estimated at a quarter, and this is probably not an over-statement if the cases are included in which the cardiac symptoms are chiefly marked. When there is much enlargement of the thyroid and much prominence of the eyes, complete recovery is very rare.

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* *Trans. Oph. Soc.*, *1886*, p. 113. It is noteworthy that this patient had an epileptic aura consisting of "an awful smell that seemed to ascend from the left side of the body and reach to the mouth," but it was not followed by any actual fit or loss of consciousness.

† In the curious case recorded by Burney Yeo (p. 1203, note), the occurrence of exophthalmos, first on one side and then on the other, was accompanied by loss of the hairs of the eyebrow and eyelid.

‡ Progressive muscular atrophy occurred in a case described by Cazal (*Gaz. Hebd.*, *1885*, No. 21).

§ Carrington, *Trans. Oph. Soc.*, *1886*, p. 97.

I have seen one patient in whom well-marked symptoms, great prominence of eyeballs and enlargement of thyroid in early womanhood, passed away entirely, and twenty years later there had been no recurrence.* Even when there is not complete recovery, however, great improvement sometimes occurs, and the disease may subside from a most distressing degree to comparative insignificance. Just as the cardiac symptoms commence before the others, so they persist, in cases that recover, long after the enlargement of the thyroid and prominence of the eyes have disappeared. The malady is said to run, on the whole, a more rapid course in men than in women.

In most fatal cases, death seems to be the result of the cardiac affection, and of increasing general weakness. Attacks of palpitation and dyspnœa become more frequent and troublesome; œdema of the legs comes on; the loss of flesh increases to extreme emaciation, and the patient gradually sinks. Other occasional causes of death have been tuberculosis, cerebral hemorrhage, rarely hemorrhage elsewhere, cerebral softening, and pachymeningitis. Sometimes sudden death occurs, the exact cause of which cannot be ascertained.†

Pathological Anatomy.—In the heart, signs of dilatation and hypertrophy are commonly found after death, but are far less than might be expected from the symptoms present during life. Slight endocarditis is occasionally met with, and graver lesions of the valves are more frequently found than might be anticipated; no doubt because such cases more frequently die. The arteries are found dilated, and may appear to be distinctly hypertrophied (Vogt). The veins also may be enlarged. In the old it is doubtful what significance is to be attached to the state of the arteries.‡ Aneurismal dilatations have been seen on the arteries of the thyroid.

In the thyroid body, all the vessels are conspicuously enlarged, and the proper tissue of the gland is increased in quantity, without being materially altered either in naked-eye or microscopical appearance. Occasionally however, there is a greater change, either cystic or "colloid" degeneration.

In the orbit the most frequent change is an increase in the quantity of fat, but this is never extreme, and in many cases the amount of fat has been perfectly normal. Dilatation of the orbital veins, atheroma of the ophthalmic artery, and fatty degeneration of the ocular muscles, have been met with in rare instances. In one case in which the fatty degeneration of the straight and oblique muscles was very marked, the levatores palpebrarum presented a marked contrast in their normal condition. It is also noteworthy that in this case there was no increase in the orbital fat, and no trace of the tissue of Müller's muscle could be discovered.§

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* The disease was, however, succeeded by other curious symptoms of the derangement of the sympathetic—pigmentation of the skin, and persistent œdema of the eyelids, allied to myxœdema.

† Two instances of this are recorded by Hale White, *Brit. Med. Journal*, July 24th, 1886.

‡ As, for instance, a case recorded by Wähner, in which thickening of the arteries of the spinal cord was found in a woman of sixty-two.

§ Silcock, *Trans. Oph. Soc.*, *1886*, p. 104.

The cervical sympathetic has been carefully examined in many cases by competent observers. It has often been free from any indication of disease. In other cases slight changes have been found; the most frequent has been an increase of connective tissue in and about the lower cervical ganglion, and with this change the ganglion has been described as enlarged above, or reduced below, the average size, as normal in consistence, or as distinctly indurated. An atrophy of the nerve cells has been described in a few cases. The cardiac ganglia have been found normal when they have been examined.

No significant changes have been discovered in the central nervous system. Cheadle has described signs of congestion in the medulla, but these have not been seen by most observers. The other organs of the body are also, as a rule, normal. Disease of the kidneys has been found in some cases, but in most of these there had been organic heart disease. The thymus and other closed glands undergo no change.*

PATHOLOGY.—Two questions are involved in the pathology of exophthalmic goitre, as in that of most other diseases—the mechanism of the symptoms, and the nature of the malady—questions that are in part the same, in part distinct.

The dilatation of the vessels and over-action of the heart have directed attention to the sympathetic system of nerves as capable, by its derangement, of giving rise to the symptoms. The cervical sympathetic contains fibres which, when they are irritated, increase the frequency of the heart's action. But the vascular dilatation suggests paralysis of those sympathetic fibres that influence the vessels. If we assume that an active dilatation exists, we must assume the excitation of certain fibres of the sympathetic, and not of this nerve as a whole. Thus we must assume either a partial affection of the sympathetic, or else an opposite disturbance of its different elements, and either hypothesis seems incompatible with the assumption of any general morbid process in the nerve, such, for instance, as an irritation or inflammation beginning outside the nerve elements. This consideration has led many pathologists to look beyond the sympathetic, to the central nervous system, for the seat of the primary morbid process that deranges the vessels and the heart. This view is supported by the fact that the association of arterial dilatation and frequency of the heart's action is in harmony with the physiological law, discovered by Marey, that there is an inverse ratio between frequency of pulse and general blood pressure, so that lowered arterial tone, and a frequently acting heart, coincide. These considerations certainly suggest that these two symptoms depend essentially on the derangement of a central mechanism, rather than on a random process in any system of nerves. On the whole, the results of pathological anatomy favor this opinion. The frequency with which no morbid appearance has been found in the sympathetic, and the equivocal character of the changes that have been described in a few cases, suggest that these, when present, are of secondary origin, or else are accidentally associated. Leaving, for the present, this general question, we may consider the origin of the other symptoms.

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* See Hale White, loc. cit.

The enlargement of the thyroid was regarded by Graves as a kind of erection, and modern pathology has been unable to suggest any better explanation. The great dilatation of its vessels, and its variation in size with the action of the heart, seem to confirm the opinion. It is, moreover, certain that some enlargement of the thyroid will result from vascular distention, such, for instance, as follows ligature of the thyroid veins (Boddaert). According to this view the overgrowth of the tissue of the organ is the result of the increased blood supply. And yet two considerations suggest that the relation of this symptom to the primary derangement, whatever this is, may involve more than mere vascular dilatation. (1) The thyroid may be scarcely enlarged when the cardiac and vascular symptoms are very marked; conversely, although rarely, the thyroid may enlarge considerably when the heart is but little excited; and lastly, its enlargement has been known to precede the disturbance of the heart. (2) The mental weakness, etc., that sometimes result from extirpation of the thyroid, seems to show that the organ has a mysterious influence on the central nervous system, and an opposite influence is at least conceivable, if equally mysterious. We must beware of the assumption that the mechanism we can trace is the only one in operation.

The prominence of the eyeballs is very difficult to explain. (1) An increase in the orbital fat cannot have more than a trifling influence, even when it is present. (2) The distention of the vessels of the orbit is probably a more powerful factor. (3) The unstriated muscular fibres of Müller, which run from the eyelid to the membranous lining of the orbit, are generally believed to be capable by their contraction of causing prominence of the eyeball. The eye recedes a little when the sympathetic is divided. Although it has been questioned whether in man these fibres are sufficiently developed to exert an appreciable effect, it is possible that they may become hypertrophied under permanent excitation. It is certain that our present knowledge permits us to invoke no other than these three mechanisms, although it is not easy to conceive how even their combined influence can produce the extreme prominence sometimes seen. We must thus assume an irritation of the sympathetic to explain the exophthalmos as well as the cardiac over-action.

The loss of descent of the upper eyelid was ascribed by von Graefe to spasm of these fibres of Müller, and this remains, on the whole, the most probable explanation. It must be due to spasm either in these fibres or in the levator. The fact that it does not interfere with closure of the eyes—in which movement the orbicularis at once overcomes the spasm—is less easy to understand on the assumption of spasm of the levator than on that of an affection of the fibres of Müller.

The retraction of the upper lid is a phenomenon analogous to the defective descent, but the fact that the two are not related in degree or even in occurrence increases very much the obscurity of the subject. Moreover, if the condition of the lid is due to the contraction of the fibres of Müller, and this is the chief cause of the exophthalmos, the two symptoms should correspond in degree, but, as we have seen, this is far from being the case.

It is a curious fact that cocain applied to the conjunctiva causes a slight retraction of the lids and a defective descent similar to that seen in exophthalmic goitre.* It is assumed that the cocain acts upon the sympathetic nerve endings, since Jessop has found that the effect cannot be obtained a few days after division of the sympathetic in the neck. This does not, however, throw any light on the muscular mechanism by which the symptoms are produced, but it supports the view that they are due to over-action of the sympathetic nerves.

Thus the symptoms seem to be due partly to the direct effect of deranged action of the sympathetic, and partly to the indirect effect of this derangement on the vascular system. It is probable that the cause of the derangement is to be sought beyond the sympathetic in the central nervous system. All the sympathetic functions are under central control, and the character of the symptoms, as we have seen, is in harmony with the physiological relation between the degree of blood pressure and the frequency of the heart's action. The chief controlling centres, and especially those that determine this relation, are in the region of the medulla oblongata, and it is to this part that we are thus directed in our search for the seat of the disease. The negative results of pathological investigation, and the character of the disturbance itself, make it probable that the morbid state is one of function and of that finer nutrition that baffles, and will baffle, our means of investigation.

Attempts have been made to discover whether experiment can supply the evidence that pathological anatomy fails to afford. Filehne, in particular, has tried to demonstrate the dependence of the disease on the medulla oblongata. He divided the anterior fourth of the restiform bodies, and succeeded in producing exophthalmos. In a few experiments he caused, also, some enlargement of the thyroid, and in one case all three symptoms of exophthalmic goitre were produced. These results certainly confirm the view which refers the disease to the medulla oblongata. If this is correct it is possible that some of the cardiac disturbance is produced through the agency, not of the sympathetic, but of the vagus.

Thus the negative evidence of pathological anatomy, and the positive evidence afforded by the character of the various symptoms, alike suggest that the malady is a neurosis, a disease of the nerve elements themselves, having its character determined by their functional relations, and its origin in conditions at present beyond our range of scrutiny. We can thus understand the multiform derangements of the same class with which its symptoms may be associated.

Diagnosis.—When all the symptoms of the malady are present, and are considerable in degree, the disease is one that is easy to recognize. It is difficult to detect, and apt to be overlooked, only when it is in an early stage, and partially developed; yet it is in these cases that the diagnosis is of the greatest importance, since it is in these that treatment has most prospect of affording permanent relief. In such cases the chief symptoms are cardiac, and the malady may be mistaken for mere nervous excitement or for organic heart disease. The strong pulsation of the arteries, and the persistent excess and frequency of the heart's action, are the most important diagnostic indications. A pulse rate of 120, maintained even when the patient is tranquil, should always arouse a suspicion of the disease, and the thyroid and eyes should be carefully examined.

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* This fact was pointed out by Koller in his original account of the action of cocain, and the phenomena have been carefully studied by Jessop, *Trans. Oph. Soc.*, vol. v, p. 240, and vi, 123.

Ordinary goitre is distinguished by the absence of the cardiac and ocular symptoms, by the degree of enlargement of the thyroid, and by its relation to endemic influences. But there is a class of cases with cardiac symptoms which most authorities consider ought to be kept distinct from the true exophthalmic form. A large goitre of endemic origin becomes associated, after a time, with over-action of the heart and exophthalmos, in consequence of the pressure of the tumor on the sympathetic, and perhaps, also, on the vagus. The exophthalmos is usually one-sided, corresponding to the chief enlargement of the thyroid, and is accompanied with spasmodic mydriasis, from irritation of the sympathetic fibres to the iris.

PROGNOSIS.—In fully-developed cases the prognosis is grave. When the eyes are distinctly prominent, the thyroid enlarged, the heart dilated, and its habitual frequency much increased, it is rare to obtain more than a moderate degree of improvement as the result of the most careful treatment. In many cases even improvement cannot be obtained. Nevertheless the prospect, even of these cases, is not entirely gloomy. Occasionally a degree of improvement occurs that amounts almost to restoration of health, and, in rare cases, to perfect recovery. This result is, however, far more frequent in the slighter and earlier cases, in which the heart disturbance is recent, and the enlargement of the thyroid is very slight or is absent. The prognosis is rendered worse by conspicuous failure of general nutrition, and by the presence of organic heart disease. It is better in women than in men; nevertheless, cases of recovery in men are sometimes met with.* It is better if the disease shows a remittent tendency, and I am inclined to think that, if the disease is in an early stage, it is better when there is a family tendency to the affection than when there is none. . A distinct and removable exciting cause certainly also improves the prognosis. If no improvement is obtained, an opinion is often sought regarding the probable duration of life. This is, however, impossible to forecast. The sufferers may present little change, for month after month and year after year, and there is hardly any degree of severity that absolutely precludes all hope of amelioration, and even of considerable improvement. One patient, when I saw her, was in a condition in which it seemed scarcely possible that she could live three months, and yet three years afterward she was not only alive, but so much better that she had become a wife and mother. Several other instances of similar improvement have come under my notice. The average prognosis is certainly worse among hospital patients than in those of better position, in whom suitable conditions of life can be secured and maintained for a long time.

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* See *Trans. Oph. Soc., 1886*, p. 26.

TREATMENT.—The first and most important element in treatment is to secure tranquillity of mind and rest of body. Frequently as the heart, in this disease, beats during rest, it is quickened still more by emotion and exertion. In a severe case it is well to keep the patient in the recumbent posture for a time—in bed if the onset has been recent and acute, or if the symptoms are severe. Absolute rest may quickly bring the pulse down from 120 to 90, or even 80. In any case, several hours of each day should be spent on the sofa, and only the gentlest exercise should be permitted. If perfect rest is desirable, the lack of exercise may, in some degree, be compensated by gentle daily massage. Every kind of mental or physical excitement should be absolutely avoided. Complete change of air has often been followed by marked improvement, but as a rule mountain air is not desirable. Most patients do best at the seaside, if the air of the place is not too stimulating. A sea voyage has apparently largely contributed to the cure in a few cases, but is a hazardous remedy unless the "sailing" capacity of the sufferer is known. In each case the effect of change must be carefully watched, since it is not always possible to foretell its influence. There is no trustworthy evidence that any foreign "watering-place" has any special influence on the disease.

Medicinal treatment is singularly uncertain in its results. Drugs that have clearly done good in some instances have been quite ineffectual in most other cases in which they have been given. This statement applies to iron, quinine, arsenic, strychnia, and other tonics. In spite of anæmia, iron fails in most cases to improve the condition of the patient. It seems sometimes even to do harm. Von Graefe, whose experience of the disease was very large, came to the conclusion that iron is useless in the height of the malady, but that it does good when a certain amount of improvement has occurred, and the pulse has fallen below 100. Iodine and iodides have been thought by many to be positively harmful; nevertheless, cases are occasionally reported in which benefit has followed their internal administration or external use. Bromides have no influence on the disease itself, but are often useful, and even necessary, for the purpose of lessening irritability and promoting sleep.

Drugs that slow the pulse would seem to be specially indicated, but the experience of different observers is as much at variance on this point as on the action of tonics. It is certain that digitalis fails, in most cases, to produce any permanent effect, and yet cases are occasionally published in which digitalis and iron together have seemed to cure the patient. Trousseau thought digitalis useful when given in full doses, so as to reduce the pulse to sixty or seventy beats per minute. Veratrum viride does not seem to possess any special advantage. Aconite has been recommended in doses of five minims of the tincture, increased gradually to twenty minims.

Belladonna is the drug that has seemed most distinctly useful in the largest number of cases, and this is entirely confirmed by my own experience. It should be given in gradually increasing doses, until the maximum quantity is reached that the patient can bear. Either the tincture or the extract may be given, or atropine may be injected hypodermically. The latter method, however, does not possess any advantage. The weakness of the tincture is no drawback to its use, if the dose is increased until considerable dryness of the throat is produced. I have once known pronounced symptoms to disappear entirely under the use of belladonna, and in several other cases marked improvement resulted. Both belladonna and digitalis, it may be noted, have an influence on the small arteries, increasing their tone. Ergot has rarely failed to disappoint those who have given it in this disease. Hydropathic treatment has been strongly praised, chiefly by French physicians.

Galvanization of the cervical sympathetic has been recommended, and often employed. Perhaps it would be more correct to describe the treatment as the application of voltaism to the position of the sympathetic. It is not quite certain that the sympathetic is influenced, since we cannot by such application in a healthy individual obtain any dilatation of the pupil. It has been thought by some to do good by acting on the vagus rather than on the sympathetic. The application is followed in many cases by a distinct fall in the frequency of the pulse (amounting to ten or even twenty beats per minute), and sometimes by a slight diminution in the size of the thyroid. I have known, for instance, the pulse fall from 90 to 72, a lower rate than had been observed during the preceding two years. The effect, unfortunately, is usually transient, but repeated applications certainly sometimes cause a slight degree of permanent improvement. I have not, myself, seen more than this, but several cases have been recorded in which all the symptoms disappeared under this treatment. Both electrodes may be placed along the anterior edge of the sterno-mastoid, one above and one below, or one electrode may be placed in either of these positions, and the other on lower cervical spines or beneath the occiput. A weak current only should be employed, such as causes a moderate amount of tingling in the skin. Galvanization of the cardiac region has been advocated by Vigouroux and Charcot.*

Surgical Treatment.—Excision of the thyroid is impracticable when the disease is fully developed. The dilatation of the blood vessels renders the operation too formidable. In a few very early cases it is said to have been successful in arresting the disease, but the evidence of the nature of these cases is scarcely satisfactory. In this disease, as in ordinary goitre, excision of the isthmus causes atrophy of the whole gland, but the operation has not been followed by permanent results to the other symptoms of the exophthalmic form. In one patient on whom the operation was performed, the resulting atrophy of the gland did not materially influence the exophthalmos, and there was also considerable valvular disease of the heart, which caused death four months later.* The same may be said of setons, cauterization, and injections of ergotin. Handfield Jones has seen injections of iron into the thyroid give rise to alarming symptoms. On the whole, the results of surgical treatment are not at present sufficiently encouraging to justify their adoption.

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* *Gaz. des Hôp., 1885,* Nos. 13 and 15.

Among special symptoms that may call for treatment, the paroxysms of palpitation and dyspnœa are best relieved by "derivatives," as hip baths and mustard plasters to the feet, with cold to the thyroid (if its enlargement is sufficient to be a cause of dyspnœa) and by full doses of digitalis. Ether, chloroform, and morphia also sometimes give relief in this condition. Trousseau found a copious venesection effectual in relieving such an attack when other measures failed. Any inflammation of the eyeball must be promptly treated, to obviate, if possible, the danger to sight.

PARALYSIS AFTER ACUTE DISEASES.

Many acute diseases, especially the acute specific diseases, are occasionally accompanied or followed by symptoms of impairment of function of the nervous system. Of these symptoms, motor palsy is the most frequent. One of these diseases, diphtheria, is followed by palsy of a special type, such as is seldom or never met with after any other affection, and must therefore be separately described. In the other maladies the nervous symptoms vary much in their character, and evidently also in their mechanism. They are, on the whole, rare, but less so after some diseases than after others.

In the course of any acute disease, sudden symptoms may come on, such as indicate the occurrence of a vascular lesion in the brain. Usually there is sudden hemiplegia, which may be accompanied by loss of speech, and may persist or may pass away, according to the position of the lesion. In some cases, especially in children, convulsions attend the onset. The morbid process varies in different cases. In some there is embolism, consequent on endocarditis, which may complicate any one of these diseases, but is most frequent in scarlet fever. In other cases the lesion is probably thrombosis in an artery or in a surface vein. In other very rare cases, again, there is hemorrhage. These lesions are most common in children.

The other symptoms met with present no general correspondence in character; they may be cerebral or spinal, and may suggest a specific action of the blood state on the nerve centres or subacute organic lesions.

TYPHOID FEVER.—Apart from the sudden hemiplegia already mentioned, which is very rare in typhoid fever, the most common nerve symptoms indicate a special influence of the poison on the brain. The common deafness is apparently produced in this manner by an interference with the functions of the auditory nerve. Loss of speech is not uncommon. It occurs chiefly toward the close of the disease and during convalescence; it may come on as the deafness is passing off. There is generally complete speechlessness, and, although it is called aphasia, there is no disorder of speech, or partial loss, such as occurs in cases of organic disease of the brain. The patient can understand perfectly what is said, and may be able to express assent and dissent by signs, or even to write. In one case under my observation the condition developed gradually by a disuse of the lips, which were dry and cracked; the loss of the labial articulation rendered the words almost unintelligible, and they gradually ceased to be uttered. The condition may last for some weeks. In one recorded case it was associated with general choreoid movements.* Typhoid fever is sometimes followed by mental defect, which may amount to actual imbecility. Impairment of memory is common. Occasionally there is some more pronounced mental derangement, generally in the form of chronic mania. These cerebral symptoms always pass away in time, but in adults the memory may never be so good as before the fever.

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* Bristowe, *Trans. Oph. Soc.*, *1886*, p. 46.

Paraplegic weakness is not uncommon, and also occurs usually when the disease is on the decline, or during convalescence. In some cases the weakness of the fever seems to persist in the legs, and as in one case under my observation, when the patient is seen some months after, there is distinct defect of power, with excessive knee jerk and foot clonus.† Money has indeed found that there is generally an excess of myotatic irritability during the disease.‡ In other cases actual paraplegia comes on more rapidly, although not suddenly, and may increase to a considerable degree of weakness, although there is rarely absolute loss of power. The onset may be attended with spinal tenderness, and with hyperæsthesia or various subjective sensations in the legs, and these may be followed by defect of sensation. It is probable that the symptoms are due to myelitis of slight intensity. They usually pass away in the course of a few weeks. In very rare cases, paralytic symptoms are of greater severity. The weakness, commencing in the legs, may invade the arms, and when such extension occurs rapidly, the case becomes one of the so-called "acute ascending paralysis," and death has been known to occur in the course of a few days.

Limited atrophic paralysis occurs in some cases, especially during convalescence. It is sometimes very severe and may leave permanent disability. It is probable that in some cases the lesion is spinal, acute anterior poliomyelitis. There is loss of power, variable in extent, with rapid wasting and loss of faradaic irritability in the muscles most affected. Although a considerable amount of recovery always occurs, there may be permanent atrophy of certain muscles. This affection is more frequently secondary to typhoid fever than to any other acute specific disease. It is, however, probable that in many cases such symptoms are due, not to poliomyelitis, but to multiple neuritis. This was probably present in a case described by Alexander, in which typhoid fever, in a patient aged twenty, was followed by wasting of muscles with loss of electrical irritability and of the knee jerk, and paralysis of one vocal cord. The development of the palsy was accompanied by severe pains in the legs.* Recovery was complete. The neuritic nature of the symptoms is also sometimes probable when the atrophy is local. In a case recorded by Vulpian, during convalescence, pains in the right shoulder and arm were followed by paralysis, considerable in the deltoid, slight in the biceps and brachialis anticus, and still less in the triceps and scapular muscles. There was loss of electrical irritability, faradaic and voltaic, in the deltoid, but no affection of sensation. The distinction of the central or peripheral nature of many cases is very difficult.

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* De la Harpe, *Rev. méd. de la Suisse Rom.*, *1883*, No. 6.

† It is not uncommon for adults after typhoid never to regain the good walking powers they had before the illness, although there is nothing that can be called paralysis.

‡ *Lancet*, *1885*.

In very rare instances paraplegic weakness after typhoid fever has been associated with paralysis of the soft palate, but even these cases do not resemble closely diphtheritic paralysis. There is never the paralysis of accommodation, or the slow progress of paralysis from one part to another.†

Paralysis of cranial nerves has been seen in a few instances, and in two cases paralysis of the abductors of the vocal cords occurred in so complete a degree as to necessitate tracheotomy (Rehn, Villemin). Optic neuritis is a very rare sequel of typhoid fever. Localized neuralgic pains in the course of the spinal nerves are occasionally met with during convalesence. They have been known to be accompanied with spots of anæsthesia, and are then doubtless due to neuritis. Landouzy records a case in which a spot of such anæsthesia in the groin was permanent.

The muscular tremor, which is occasionally so conspicuous during the course of typhoid, may continue during convalescence, and it has been known to persist and to be followed by the symptoms of disseminated sclerosis (Chauffard, Ebstein, and Westphal). The tremor never goes on to paralysis agitans. In girls, hysterical symptoms are not rare during the course of the disease and during convalescence; contractures, anæsthesia, and even convulsions have been met with.

TYPHUS FEVER.—After typhus fever, symptoms have been met with very similar to those that are met with after typhoid. Paraplegic weakness is, however, less common; paralysis of cranial nerves occurs only in consequence of an attack of meningitis complicating the fever; while paralysis of the palate has not been met with. Bernhardt has recorded a case of neuritis of the musculo-spiral nerve, which may have been set up, as paralysis in this nerve so often is, by pressure upon it during the period of prostration and inertia. Optic neuritis has been observed after typhus, although even more rarely than after typhoid.

ERYSIPELAS is rarely followed by paralysis. In one remarkable case‡ paralysis of the soft palate and of the posterior crico-arytenoids (abductors of the vocal cords) followed facial erysipelas and one-sided pneumonia.

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* Alexander, *Deut. med. Wochenschr.*, *1886*, No. 31.

† True diphtheria sometimes complicates typhoid fever, and may be followed by characteristic paralysis, as in a case related by Dr. Murchison.

‡ Feith, *Berlin. klin. Wochenschrift*, *1874*, No. 49.

The paralysis of the larynx necessitated tracheotomy, and appears to have been permanent.

VARIOLA.—Spinal symptoms are more common in connection with smallpox than with any other of the eruptive fevers. The fact was associated by Trousseau with the severe pain in the back which occurs at the onset, on the ground that this pain may be accompanied by weakness, formication, and tingling in the legs, plantar pain, and retention of urine, and that these symptoms pass away with the pain in the back on the appearance of the eruption. More pronounced spinal symptoms may come on during the height of the disease, or especially during convalescence. There is paraplegic weakness, with or without affection of sensation, and varying much in degree. Recovery in severe cases may be incomplete. The symptoms probably depend upon myelitis, which, disseminated through the cord, was actually found by Westphal in one case; the gray and white substance was occupied by scattered spots of softening.* Acute atrophic paralysis has also been observed, usually due, no doubt, to poliomyelitis (as Roger was able in one case to demonstrate), but possibly sometimes produced by a primary alteration in the nerves. In a case of atrophy in one arm, Joffroy could find no change in the cord, but only an acute degeneration of the nerves. Ataxy of both arms and legs, persisting for a long time after the primary disease, but ultimately passing away, has also been described.† Acute ascending paralysis has been observed during convalescence, fatal in a few days, and, as in other cases, without recognizable lesion.‡ Occasionally paralytic symptoms extend to the cerebral as well as the spinal functions. The ocular muscles have been paralyzed, perhaps from neuritis. Smallpox has also been followed by symptoms of insular sclerosis.§

MEASLES.—Spinal symptoms may occur during and after measles, very similar in characters to those which follow smallpox. Those of acute poliomyelitis are the most frequent, but acute ascending paralysis has been met with (Bergeron and Liégeard). A remarkable case of disseminated myelitis has been recorded by Barlow.|| Hemiplegia, probably from thrombosis in a cerebral vessel, has also been met with. Optic neuritis has only been observed as a result of coincident meningitis.

SCARLET FEVER.—Spinal symptoms are rare after scarlet fever, while cerebral hemiplegia is not infrequent, and is probably due to embolism from scarlatinal endocarditis, or to spontaneous thrombosis. It may occur during or apart from kidney sequelæ. Convulsions, persisting as epilepsy, are also more frequent during or after scarlet fever than after any other acute disease (see p. 1080). Neuro-retinitis has been many times observed to follow scarlet fever, without any renal complication.¶

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* Foci of softening have also been described by Damaschino, and by Joffroy and Hayem.

† Manisolle, *Thèse de Poitiers*, ref. in *Virchow's Jahresb.*, *1880*, ii, 44.

‡ Bernhardt, *Berl. klin. Wochensch.*, *1871*, No. 47; Gros and Beauvais *L'Un. Med.*, *1884*, No. 131.

§ Long, Charcot.

|| *Med.-Chir. Trans.*, *1885*.

¶ In scarlet fever there is sometimes false membrane in the throat, like that of diphtheria, and the patients are often said to have had both diseases. My colleague, Dr. Barlow, whose

MUMPS.—Palsy of accommodation has been described, but was met with only in one case in an extensive epidemic.* Joffroy met with paralysis of the limbs in one instance; there was loss of electrical irritability and of reflex action, and the muscles were tender.† It is possible, however, that in each of these cases there may have been undiscovered diphtheria; in the second case, indeed, slight angina was observed.

MALARIAL FEVER.—During the course of severe malarial fever singular paralytic symptoms have been met with, especially hemiplegia and aphasia, which are said to pass away under the influence of quinine, and are, therefore, ascribed to an effect of the malarial poison on the nerve centres (Grasset). Graver and more lasting symptoms of the same class have been met with in cachectic subjects, and have been ascribed to obstruction of cerebral vessels by pigmentary matter.

DYSENTERY, when severe, has been followed by paraplegia, commencing with tingling in the skin, and pains along the course of the nerves, succeeded by motor and sensory paralysis, sometimes with muscular wasting. The arms may also be involved. These symptoms sometimes occur during the course of the disease. They are probably due to myelitis or to peripheral neuritis. In one case, extensive softening was found in the cervical and lumbar enlargements (Delioux).

DIARRHŒA.—Simple diarrhœa, whether spontaneous or the result of undue purgation, has been followed by spinal symptoms, usually transient, sometimes more severe. In women, hysterical symptoms also result from the peculiarly depressing effect of diarrhœa on the nervous system. Tetany is an occasional sequel. In children, exhausting diarrhœa sometimes causes cerebral thrombosis, or the symptoms of cerebral anæmia termed "hydrocephaloid" (see p. 761).

ACUTE RHEUMATISM.—No palsy is common in connection with acute rheumatism, except the embolic hemiplegia that is the result of the endocarditis. Myelitis, especially poliomyelitis, has been thought to be a frequent sequel, but the cases on which this opinion has been based have been for the most part those in which pains in the limbs have attended the onset of the spinal disease, and have given rise to an erroneous diagnosis of acute rheumatism. It is possible, also, that rheumatism and myelitis may both result from the same exposure to cold. Curious cases are, however, sometimes seen, in which inflammation and swelling of the joints of the legs, especially of the ankle joints, have been followed by paralysis, and it is doubtful what is the relation between the two, whether the arthritis was the result of the commencing spinal lesion, as it may be the result of acute and severe

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experience of these cases is very large, informs me that he has never known diphtheritic paralysis to follow this complication, and hence it is very doubtful whether the membrane is an indication of true diphtheria. It is possible, however, that there are two forms of this complication, one truly diphtheritic (see Heubner, *Jahrb. f. Kinderheilk.*, Bd. xiv, and Demme, *Bericht aus d. Jennerspital*, Berne, 1878 and 1879).

* Boas, *Kl. Monatsbl. f. Augenh.*, *1886*, p. 273.

† Joffroy, *Prog. Méd.*, *1886*, No. 47.

myelitis, or whether it was really of the nature of true rheumatic inflammation. Thus, a patient aged thirty, had subacute articular rheumatism of both ankle joints, with considerable pain, and this was followed by incomplete palsy of all limbs, disturbance of sensibility, partial atrophy of the muscles with the degenerative reaction, loss of the knee jerk, tremors and involuntary twitchings; the face and facial nerves were also involved, and some optic neuritis supervened. Remak, who observed the case, assumes that there was multiple degenerative neuritis, perhaps with central foci of sclerosis.*

DIPHTHERITIC PARALYSIS.

Diphtheria is frequently succeeded by paralysis of definite and peculiar character, such as is not met with after other acute diseases, or is met with only in extremely rare instances. The occurrence of paralysis after low forms of sore throat has long been known, and was described by the elder Chomel, as observed in an epidemic at Paris in 1748, by Ghisi in Italy, in 1749, and by Bard, at New York, in 1771. When diphtheria came to be distinguished from other forms of angina, it alone was found to be succeeded by this paralysis. Hence it is almost certain that the epidemic sore throats which, in the last century, were followed by paralysis, were also diphtheria. It is, indeed, believed by some (influenced chiefly by the assertions of Gubler) that a similar paralysis occurs, in rare cases, after simple or herpetic tonsillitis, but the evidence of this is not conclusive, and that adduced by Gubler is least so. It is true that paralysis does sometimes follow an attack of sore thoat, the diphtheritic nature of which has not been suspected. But the significance of such cases is lessened by the following considerations: (1) Slight cases of diphtheria, in which the false membrane is limited, say to a small spot on one tonsil, often escape recognition until their nature is declared by the origin from them of other unmistakable cases. (2) Such slight cases, as well as severe ones, may be followed by paralysis. (3) The supposed cases of paralysis, after simple angina are as rare as such sore throats are common, while paralysis occurs after a large proportion of the cases of true diphtheria. These considerations make it probable that all sore throats followed by paralysis are diphtheritic. Indeed, considering the above facts and the small amount of false membrane sometimes present, it seems more probable that diphtheria may occur without any false membrane, than that characteristic paralysis occurs after simple sore throats.†

Other acute diseases may be followed by paralysis, but in these cases the palsy presents characters for the most part altogether different from that which succeeds diphtheria. They have been already described.

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* *Neur. Centralbl.*, *1885*, No. 14.

† Cases, however, have been met with (see p. 1233) that suggest that a paralysis closely analogous to diphtheritic paralysis may occur as a primary affection without any preceding acute disease, and may run a course similar to that which succeeds diphtheria. I have seen one or two cases suggestive of this conclusion.

The frequency with which diphtheria is followed by paralysis seems to vary in different epidemics and at different ages; it has been variously stated at from 8 to 66 per cent. It is probably not far from the truth to say that, on an average, one-fourth of those who do not die from the primary disease subsequently suffer from paralysis. Its occurrence is not influenced by sex, but, according to the statistics collected by Landouzy,* age distinctly increases the liability to suffer. The sequel may, indeed, occur at any age, from two years upward, but adults furnish a larger proportion of the cases of paralysis than of diphtheria. Sixty-eight cases of paralysis were distributed as follows (Landouzy): Under 10, fifteen; 10–20, eighteen; 20–30, thirteen; 30–40, eight; 40–50, eight; over 50, six. From this it would seem that the tendency to paralysis is very slight in early infancy, and that it increases the older the sufferer. I have seen one severe case at the age of sixty.

The previous health of the patient seems to have no influence; paralysis does not occur more frequently in the weak than in the strong. Nor does the severity of the attack of diphtheria determine the occurrence of palsy; it is equally likely to follow attacks that are severe and those that are trifling. It occurs after diphtheria of the surface as well as after that of the throat. The celebrated alienist Griesinger died from paralysis succeeding a diphtheria limited to the wound produced by opening a perityphlitic abscess.

Time of Onset.—As a rule, the primary disease is over before the paralysis comes on; the most common time being the second or third week after the termination of the throat affection. Sometimes, however, it commences during the course of the diphtheria, from the fifth to the tenth day, and it has been known to commence as early as the second day. These statements apply to definite loss of power.

SYMPTOMS.—The onset of the paralysis is usually marked by no general symptoms, but now and then a slight elevation of temperature, and, in children, restlessness and irritability, may precede it, and they may also accompany its subsequent extension. The first part to be affected is usually the palate, and fluids are found to regurgitate through the nose. Vision for near objects is then interfered with, in consequence of loss of power of accommodation. These two palsies are almost invariable. Subsequently, and less frequently (in about half the cases), the limbs become weak, and often they are the seat of numbness or tingling, and sensation may be definitely impaired. Less frequent are paralyses of the muscles of the trunk, neck, and larynx, disturbed innervation of the heart, weakness of the external ocular muscles, of the bladder and rectum, and loss of sexual power.

The characteristic of diphtheritic paralysis is its gradual onset and progress in each part, and the way in which different and often distant parts are affected, one after the other. The loss of motor power is accompanied, and indeed preceded, by a loss of myotatic irritability, evinced, in the case of the legs, by the loss of the knee jerk. When the weakness is considerable, electric irritability of the muscles is usually changed, and there is moderate wasting. The fact that the knee jerk disappears before the onset of the palsy is part of a wider fact, that the knee jerk is often lost after diphtheria in cases in which no paralysis occurs. This was discovered by Bernhardt,* and has since been abundantly confirmed. He found the knee jerk was lost in no less than two-thirds of the cases examined (twenty-two in number), in which there was no definite palsy. The loss occurred usually during the second month, sometimes toward the end of the first month, *i. e.* three or four weeks after the onset of the primary disease. It both disappeared and returned first on one side, and in one case the loss was unilateral.†

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* "Des Paralysies dans les maladies aigues," Paris, 1880.

The paralysis of the palate is shown by the voice acquiring a nasal tone, and by the partial regurgitation of liquids through the nose during deglutition. The nasal tone is, of course, due to the cavity of the nose not being shut off during phonation; and hence, also, the explosive consonants are imperfectly pronounced, *p*, for instance, becomes *m*, and the patient is unable to distend the cheeks or to blow out a candle. The closure of the anterior nares at once removes this disability. Gargling, also, is no longer possible. On inspection, the palate is seen to hang more vertically than normal, and when the patient utters the sound "ah" it is not raised as in health. One side is sometimes lower than the other, but it is rare for one side to be paralyzed alone. There is usually distinct loss of sensibility in the palate; the patient does not know when it is touched, or the touch occasions no discomfort or reflex action, although, if the back of the pharynx is touched, evidence of sensibility is at once obvious. Very rarely anæsthesia has been observed without motor palsy. When the paralysis has lasted some time, the palatal muscles cannot be stimulated by faradaism as readily as in health.

Paralysis of the pharynx is not common, but may occur, and may be so considerable as to render swallowing almost impossible. The pharynx has even been relaxed to such a degree as to form a pouch in which food accumulates. In the larynx, paralysis of the upper part (superior laryngeal nerve) is more frequent than that of the vocal cords (inferior laryngeal nerve). In the former case the epiglottis stands erect against the base of the tongue, and does not descend over the opening, during the act of deglutition, in consequence of the weakness of the depressors. The upper part of the larynx is insensitive, although, when a foreign body reaches the vocal cords, pain is felt. Hence, food is apt to get into the larynx and to cause coughing. The voice is hoarse, probably in consequence of paralysis of the crico-thyroid muscle, but the vocal cords move as usual. In other cases there is paralysis in the region of the inferior laryngeal nerve, and phonation may be impossible. The laryngoscope then shows immobility of the cords and sometimes a preponderant weakness of abduction, so that the cords are not separated during inspiration.

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* *Virchow's Archiv*, *1885*, Bd. 99, p. 293.

† It has been said that the knee jerk is lost during the primary disease in most cases (McDonnell *Am. Med. News*, *1887*, p. 448), but this is not generally true. The difficulty of exact observation in a case of severe illness is very great. According to Money, the loss may be preceded by a brief exaggeration ("Treatment of Dis. in Children," p. 515).

The tongue is rarely paralyzed, but I have seen transient deviation to one side. Paralysis of the face is also infrequent, although the lips are sometimes a little weak; and there is occasionally distinct loss of power on one side. Very rarely there is considerable bilateral paralysis, which may involve all parts of the face, or be confined to the lower portion.

Special Senses.—Next to the affection of the palate, both in frequency and in time, is loss of power of accommodation of the eye, from paralysis of the ciliary muscle. This is always described by the patient as "failure of sight," but only vision for near objects is impaired. It has been alleged that the "far point" is more distant than under normal circumstances, but this statement is probably erroneous.* The degree to which sight is affected depends on the refraction of the eyeball, *i. e.*, on the degree to which the power of accommodation is habitually employed. With normal refraction, near vision is considerably interfered with; the patient, for instance, cannot read, while distant vision remains good. In hypermetropia this effect is still more marked, while if there is myopia vision may suffer little or not at all, because the patient does not need to accommodate. Before inferring the absence of paralysis from the absence of affection of sight, it is therefore necessary to ascertain that the patient is not myopic. Both eyes are always affected, although one may suffer before the other. The failure of accommodation reaches its height in a few days, and usually lasts for two or three weeks. The action of the pupil to light may be sluggish, but is rarely lost; usually there is contraction on an effort at accommodation, even when no accommodation is possible, but occasionally this associated action is lost with accommodation, although the light reflex remains. The external muscles of the eyeball are also sometimes affected. As the ciliary muscle is regaining power there may be undue convergence on accommodation from the increased voluntary effort affecting the internal recti in undue degree. The most common palsy is weakness of the internal recti conjoined with the paralysis of the ciliary muscle, so that the eyes tend to diverge, just as they often do in near-sighted persons who do not use accommodation. Occasionally there is paralysis of the external rectus on one side. I have once seen double ptosis, and paralysis of all the muscles supplied by one-third nerve has been observed. In two cases, almost all the muscles of both eyes became paralyzed. In one, the right eye, was quite motionless, and in the left the only action was in the external rectus; the eyelids also drooped; in the limbs there was ataxy and some loss of power. The ocular palsy lasted a month (Uhthoff). In the other case all the ocular muscles of both sides were paralyzed and the fields of vision were restricted. There was also paralysis of palate, right side of the face, and muscles of the neck, and ataxy of the limbs. The patient died; the morbid appearances will be mentioned presently.* I have also seen a patient who described, as part of the palsy after diphtheria at the age of twenty-two, double vision and double ptosis. Sometimes there is slight paralysis of one or another of the ocular muscles, varying from day to day.

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* See Schmidt-Rimpler, *Berlin. klin. Wochenschr.*, *1884*, No. 7.

There is not often any other obvious affection of sight than that which results from the paralysis of accommodation. Nevertheless, it is probable that a concentric contraction of the fields of vision is not uncommon; it is, as it were, masked by the effect of the cycloplegia, and so generally escapes notice. An example of it has been published by Jessop,† and it was present in the case of ophthalmoplegia mentioned above. It was found by Herschel in no less than five out of thirteen cases of diphtheritic cycloplegia examined.‡ No morbid change has been observed with the ophthalmoscope.§

Of the other special senses, hearing is never impaired, but I have twice known the sense of taste to be lost, and, in one instance, smell was impaired, although taste was normal.

Limbs.—The affection of the limbs succeeds that of the soft palate. The loss of motor power always comes on gradually, and varies much in degree, but is rarely or never absolute. It is usually bilateral and symmetrical; one side may be affected more than the other, but there is never limited hemiplegic weakness. The legs commonly suffer before the arms. The muscles become flabby and toneless; the invariable loss of the knee jerk has been already mentioned. The weakness may merely be sufficient to render the patient unduly fatigued with slight exertion, or it may be such that standing is impossible. When considerable, there is a peculiar gait that is very suggestive; the legs are moved as if they were at once heavy and limp, and there is not the aspect of stiffness so often seen in spinal paraplegia. If the affection is slight there may be no change in electric irritability, but when considerable weakness has existed for two or three weeks, there is generally a diminution of irritability to faradization, which may increase to almost complete loss. The muscles still respond to the voltaic current, and their reaction to it may be altered as in ordinary peripheral paralysis (see p. 67). The irritability of the nerve trunks is lessened to both faradaism and voltaism. Superficial reflex action is also diminished or lost.

In slight cases sensation may be unaffected, but it usually suffers when the motor paralysis is severe, and occasionally it is impaired when there is little motor weakness. Subjective sensations of "numbness," tingling, "pins and needles," "formication," etc., and sometimes hyperæsthesia, may precede diminution of sensibility, and are often complained of when there is no actual loss. When this exists it may involve equally pain and touch, or tactile sensibility alone; it is always greatest toward the extremity of the limbs. I have known anæsthesia to be confined to the fingers and thumb of one hand, in a case in which both tactile and painful sensibility had been lost over both legs. Sometimes the distribution of anæsthesia is peculiar; in one patient, tactile sensibility was lost on the palms and soles and preserved elsewhere, even on the dorsal surfaces of the hands and feet. The tips of the fingers may be alone affected. If there have been pains in the limbs, the loss of sensation may correspond to them in position. Muscular sensibility may also be impaired.

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* Mendel. For the reference to these cases, see p. 613. † *Trans. Ophth. Soc.*, *1886*.

‡ *Berlin. klin. Wochenschrift*, *1883*, No. 30.

§ Care must be taken not to mistake for this the hysterical amblyopia which, in girls, sometimes comes on during diphtheritic paralysis (see p. 1226).

Often there is clumsiness or definite incoördination of movement, over and above the weakness, and this may be marked when there is little or no loss of power. In some cases it is associated with cutaneous anæsthesia, but it is certainly independent of this, since it has been observed when sensation was unaffected. The incoördination may involve the arms or legs or both. In the legs the disorder may closely resemble locomotor ataxy, for which it is occasionally mistaken; the anæsthesia and absent knee jerk facilitate the error. Sometimes in the arms the disorder of movement resembles that of general paralysis of the insane—there are slight irregular twitching movements, which may render it difficult for the patient to take hold of an object.

The *trunk* muscles are less frequently affected than are those of the limbs, but they may become so weak that the patient is unable to turn over in bed. The paralysis of the intercostals may interfere seriously with the respiration, and unexpelled mucus may accumulate in the bronchial tubes and give rise to alarming paroxysms of dyspnœa, which in children are often excited by emotion. Duchenne asserted that the muscles of the bronchial tubes are paralyzed in these cases, but the fact rests on inadequate evidence, since there is usually sufficient weakness of the thoracic muscles to explain the symptoms. When the weakness invades the neck muscles the head cannot be supported, and the diaphragm sometimes ceases to act. Fortunately, the paralysis rarely reaches its height in the two sets of respiratory muscles at the same time.

Symptoms of *cardiac* failure are among the most grave, but among the less common, manifestations of the disease, and are supposed to depend on paralysis of the vagus. Irregularity of the action of the heart is not uncommon apart from graver cardiac symptoms. Of these the first indication is generally retardation of the pulse, which may afterward become unduly rapid, and irregular or intermittent. In some cases the retardation precedes the quickening of the pulse. In either condition fatal syncope may occur. According to Leyden, however, these symptoms ought to be ascribed to the degeneration of the muscular substance of the heart rather than to true paralysis of the pneumogastric. But it is more probable that the visible changes in the cardiac tissue are the result of the nerve lesion. Dyspnœa and irregular breathing are sometimes associated with the cardiac symptoms, and have been likewise ascribed to an affection of the vagus.

The *bladder* is affected only in very severe cases. There may be either retention, with or without overflow incontinence, or there may be simple incontinence. The anal sphincter seldom suffers, but the obstinate constipation sometimes met with has been ascribed to a paralysis of the muscular coat of the intestine. Loss of sexual power is not uncommon in adult males. Symptoms of vasomotor paralysis are never met with, nor is there any marked tendency to disturbance of the nutrition of the skin.

Complications.—Convulsions have been observed in a few very severe cases in which the paralysis has commenced during the course of the primary disease. They are to be regarded rather as a complication of the diphtheria than as part of the paralysis, and probably result, in most cases, from an organic cerebral lesion. Hysterical paralysis, motor and sensory, occasionally complicates a true diphtheritic palsy in those who are predisposed to hysteroid disturbance. Hysterical symptoms may also succeed the specific paralysis. This complication sometimes gives rise to much difficulty in diagnosis, as will be mentioned presently.

A very rare complication is sudden hemiplegia, evidently the result of a vascular lesion of the brain. Three cases have been mentioned by Mendel, two of which were fatal; in one of these a post-mortem examination showed a minute hemorrhage, the size of a cherry stone, damaging the internal capsule.*

Albuminuria is very common in diphtheritic paralysis, usually persisting from the primary disease. In severe cases, that are quickly fatal, it is seldom absent.

Course and Duration.—Diphtheritic paralysis varies extremely in its extent, severity, and course. In some cases the palsy is limited, slight and transient. In others, region after region is successively attacked; the parts which suffer first, as the palate and eye, may recover before the limbs are involved, and when these are improving, and a speedy convalescence is hoped for, paralysis of the trunk and respiratory muscles may come on and throw the patient into extreme danger. Irregular waves of palsy seem to flow through the body, sometimes quickly, sometimes slowly, and, determined as it is by influences that we cannot discern, its course can never be foretold. As a rule, however, the palsy does not return in a part which it has once left, and when local improvement has set in it usually continues, although other parts may become weaker. It has been said that when the paralysis commences before the primary disease is over, its course from one part to another is slow and often discontinuous, but when it commences some time after the primary disease it exhibits a much slighter tendency to remission (Sauné). To this rule, however, the exceptions are very numerous. In fatal cases death often occurs early, even within the first week from the onset. Sir William Jenner has pointed out that in the most severe and serious cases the interval between the primary disease and the onset of the palsy is usually short.

The total duration of the paralysis, both general and local, varies according to its extent, but is generally from six to eight weeks in cases that recover. A local paralysis lasts longer if other parts are affected than if it is the only manifestation of the disease. When the palate alone is affected, it usually recovers in two or three weeks, but if the limbs also suffer, the paralysis of the palate may last a much longer time. Loss of power in the limbs always endures longer than the affection of the palate or eye ; it rarely passes away in less than two months, and may last for four or six months. When definite improvement has commenced, it often proceeds rapidly, although the paralysis may have been severe and of long duration. Thus, in one case, paralysis of the limbs commenced in the seventh week, slowly increased until the twelfth week, remained almost complete until the twentieth week, when improvement commenced, and in four weeks more all trace of weakness had passed away. As power is regained, the electric irritability of the muscles becomes normal. The loss of the knee jerk is the last symptom to pass away, and often continues for several weeks after good power has been regained. Ultimate recovery is almost always perfect.*

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* *Neur. Centralbl., 1885*, p. 133.

Death may occur during the course of the paralysis from other sequelæ of the primary disease, especially from renal mischief. The palsy is fatal chiefly by three mechanisms : (1) Syncope from paralysis of the heart. (2) Asphyxia from paralysis of the muscles of respiration. (3) Exhaustion in consequence of inability to take sufficient food, due to paralysis of the pharynx, or to the anorexia and the repugnance to food produced by the liability for it to get into the larynx. The third mechanism is effective especially in children.

Pathological Anatomy.—The brain, spinal cord and nerves present, as a rule, no morbid appearances to the naked eye beyond a variable amount of hyperæmia or minute extravasations into their substance. Very rarely larger foci of hemorrhage are met with in the brain. Meningitis, with scattered spots of exudation around the spinal cord and medulla oblongata, was found by Pierret in one case, but in this the symptoms were different from those of ordinary diphtheritic paralysis, and death occurred on the second day. In the ordinary form of palsy the membranes are always healthy, except that there may be signs of congestion, and even, in some cases, hemorrhage about the nerve roots.† Nor are changes found in other organs, with the exception of the heart; when death has occurred with symptoms of cardiac paralysis, the substance of the heart may be pale, from degeneration of the muscular fibres.

The true pathological anatomy of the disease is revealed only by the microscope. The muscles have sometimes been found normal; in other cases, especially those of long duration, granular and fatty degeneration of the fibres has been found, variable in degree, even in the same part of the

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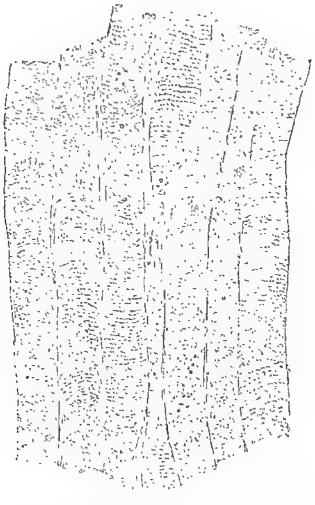
* In extremely rare cases some part, as the palate, has not recovered, and other paralyses have developed and persisted, apparently due to chronic disseminated myelitis. In one child a form of irregular bulbar paralysis (lips and palate) developed after diphtheritic paralysis at four, and persisted at eleven (Stadthagen, *Archiv. f. Kinderh.*, Bd. v). I have seen one patient in whom, a year after the diphtheria, and when all other symptoms had long ceased, there was considerable defect of power in the muscles moving the left foot, with diminution of faradaic irritability.

† Buhl, *Zeitsch. f. Biol.*, *1867*, p. 359.

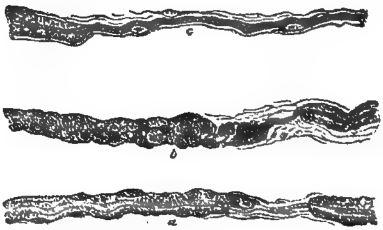
muscle and in different parts of the same fibre (Fig. 331). This change is usually most intense in the palate, but has been found also in the muscles of the limbs. In all severe cases degeneration is found in the nerves going to the paralyzed parts, either in the peripheral parts of the nerves, or in their whole extent, including the anterior roots. The posterior roots, on the other hand, are normal. The change not only corresponds to the palsy in distribution, but is proportioned to it in degree. It consists (see Fig. 332) in segmentation and breaking up of the white substance of the nerve fibres, with a multiplication of the nuclei of the nerve sheath, an accumulation of granule corpuscles among the remains of the fibres, and sometimes even a disappearance of the axis cylinders. There is not, as a rule, any inflammatory change in the interstitial tissue of the nerves. The alterations are very conspicuous in the nerves of the palate, where they were discovered by Charcot and Vulpian in 1862, and to these nerves it was thought the change was confined, until it was found in the anterior spinal roots by Buhl in 1867, and in the phrenic nerve by Liouville in 1872, but the wide extent of the change, and its correspondence with the position and degree of the paralysis, were only demonstrated by Déjerine in 1878.* The alteration (which can be best demonstrated by treating recent specimens with osmic acid) is thus limited, as a rule, to the nerve fibres. It is essentially the same as occurs in simple degeneration of nerves, and is often described as "parenchymatous neuritis." It is the more marked the longer the paralysis has lasted. Interstitial multiplication of nuclei has been seldom observed, except in the nerves

FIG. 331.



Diphtheritic paralysis. Fibres from a paralyzed muscle. Recent preparation. Granular and fatty degeneration.

FIG. 332.



Changes in the fibres of the anterior roots, picro-carmine preparation. (*After Meyer, Virchow's Archiv, Bd. 85.*)

a. Overgrowth of the protoplasm and nuclei of the sheath; the axis cylinder is continuous, although the medullary sheath is interrupted for a short distance.

b. Accumulation of granule masses, in places interrupting the axis cylinder, fragments of which can be seen between the globules of myelin.

c. A fibre in which the degenerating white substance ceases suddenly, leaving the axis cylinder only covered with the thickened sheath.

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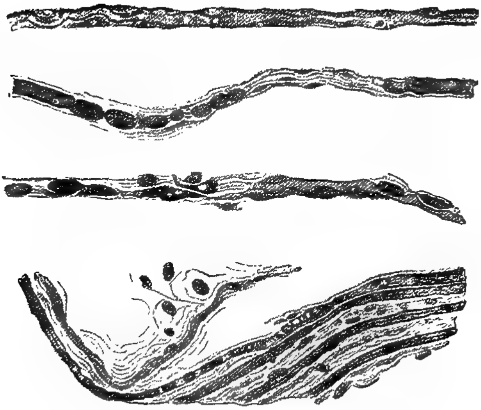
* Since corroborated by Meyer, Gaucher and others.

of the palate, but it was present in the case of fatal ophthalmoplegia mentioned on p. 1224; the nerves to the eyeball muscles presented indications of interstitial inflammation, as well as the acute degenerative changes in the fibres themselves. Minute hemorrhages were found in the sixth nerves. In one exceptional case Meyer found nodular swellings on various nerves, formed by cellular elements (Fig. 333) which separated the nerve fibres.

If the elements of the spinal cord are treated with osmic acid in the recent state, the motor cells may be found in a state of intense granular and fatty degeneration, such as is shown in Fig. 334.* In the hardened spinal cord, the white columns are always normal, and in some cases no change has been found in the gray substance. In other cases, however, the motor nerve cells of the anterior cornua have been found altered, either swollen and unduly homogeneous or vitreous in aspect, or smaller than normal, and usually with shrunken processes.† In rare instances collections of nuclei

FIG. 333.

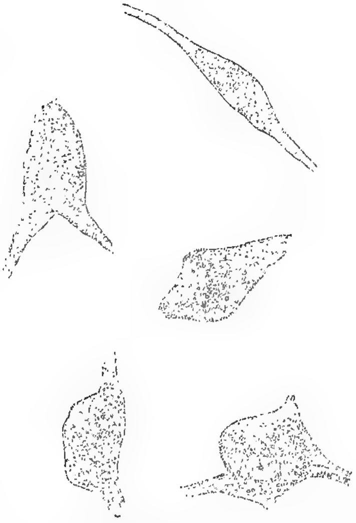


Interstitial and parenchymatous changes in the phrenic nerve; osmic acid preparations. (*After Meyer, loc. cit.*)

The three separate fibres show degeneration of the nerve fibres (segmentation of the myelin, etc.), with some increase of the nuclei.

The lower group of fibres is from one of the nodular swellings on the same nerve, and shows, in addition to the degeneration of the fibres, considerable increase in the interstitial tissue.

FIG. 334.



Motor nerve cells from the anterior cornu of the spinal cord, in a fatal case of diphtheritic paralysis. Recent preparation treated with osmic acid.

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* Accumulations of granules in the cells have been regarded as micrococcal by some writers.

† Vulpian, "Mal. du Syst. Nerv.," 1878; Déjérine, *Gaz. des Hôp.*, *1880*, No. 42; Abercrombie, *Trans. Internat. Med. Congress*, *1881*; Percy Kidd, *Med.-Chir. Trans.*, *1883*, p. 142. Kidd observed extensive vacuolation of the cells. The probable significance of this change has been already mentioned; it seems to occur in cells the nutrition of which is gravely altered. Kidd's figures of the vacuolated cells are almost exactly like those found by Sankey and myself in the dog, in chorea (*Med.-Chir. Trans.*, *1879*).

have been found in the gray substance. Of other changes which have been described, the significance is very doubtful; such are "rarefaction of the gray substance" (Vulpian) and an increase of nuclei around the central canal, often filling up its lumen, an appearance frequently seen in cords from healthy subjects, and which is probably without significance in this or any other disease.

Organisms have seldom been found in the nerve centres. Accumulations of micrococci in the vessels were described by Oertel,* and of rod-shaped bacteria by Klebs.† A remarkable case has been described by Buhl,‡ in which the nerve sheaths, etc., were infiltrated with bodies described as like those in the false membrane. In this case there were also numerous infarcts in the brain, as if from extensive vascular obstruction.

Pathology.—The alterations in the nerves agree perfectly with the indications afforded by the electrical reactions in severe cases.§ There is the loss of faradaic irritability which always indicates degeneration of the nerve fibres, and the persistence of voltaic irritability shows that the muscular tissue still preserves its contractile power. The alterations in the nutrition of the muscular fibres sometimes found are those that may occur in other forms of nerve degeneration, and correspond to this also in their inconstancy and variations.‖ Hence it is probable that the muscular changes are purely secondary to those of the motor nerve fibres. Degeneration of the nerve fibres, we know, always results either from their primary disease or from disease of the motor nerve cells, of which the axis cylinders of the fibres are the prolonged processes. This degeneration descends the motor nerves from the point at which it commences. In all known primary affections of the nerve fibres, the change begins in the distal parts and lessens as we approach the cord. Hence the fact that, in some cases, the change has been intense in the anterior spinal roots, makes it probable that this degeneration is the result of an alteration in the nerve cells. Such disease of the nerve cells has been found in severe cases. The change in the cells appears, it is true, to be slighter than that in the nerve roots, but this is not incompatible with the assumed relation between the two. We know that a transient and slight lesion of the cells often causes considerable degeneration in the fibres, and the fact that perfect recovery and regeneration of the nerves occurs in diphtheritic paralysis shows that its cause cannot be an actually destructive alteration. Changes of nutrition are, moreover, much more conspicuous in the nerves than in the cells.

But there is also another class of facts, which show that there may be a degeneration of the peripheral parts of the nerves, when the nerve roots are normal, and the gray matter and the cells, from which the fibres proceed, present no abnormal appearance. The case of ophthalmoplegia carefully investigated by Mendel is an instance of this; the centres presented no morbid appearance, although the alterations in the nerves to the eyeball muscles were profound. Recent research has shown that the nerves may suffer primarily in many diseases, when the gray matter of the cord escapes. When the peripheral extremities of the nerves are not primarily affected, they must suffer by secondary changes, and the condition of the muscles will thus be the same, whether the morbid process begins in the nerves, or in the gray matter of the cord.

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* *Ziemssen's Handb.*, ii, 1876, p. 608.

† Eulenburg's "Real-Encyclop.," iv, p. 167.

‡ *Zeitsch. f. Biol.*, *1867*.

§ The significance of the electrical reactions was pointed out as long ago as 1866, by Ziemssen (*Berl. kl. Wochenschrift*, *1866*, Nos. 43 and 44).

‖ Occasionally in injuries to nerves or in disease of the anterior gray matter of the cord the muscular fibres present very intense granular and fatty degeneration.

The fact that the lesion may begin in the peripheral nerves, makes it highly probable that in the cases in which the nerve cells of the cord are affected, the changes in the peripheral nerves are not entirely secondary. It is evident that if slight changes in the cells tend to cause slight secondary changes in the nerves, these may assume disproportionate intensity under an influence that tends to cause a primary degeneration in the fibres. This affords an explanation of the discrepancy between the amount of visible alteration in the nerve fibres and in the nerve cells, which has been noted by several observers.

Thus the facts of pathological anatomy suggest that the motor palsy depends essentially on acute alterations in the nutrition (revealed by changes in the structure) of the lower segment of the motor path (see p. 130), and that sometimes the cells and sometimes the fibres suffer primarily. The lesion is essentially "parenchymatous," seated in the nerve elements themselves. But in most intense changes of this character the elements of the interstitial connective tissue are secondarily involved in some degree. In only one recorded case were widespread interstitial changes so intense as to suggest a primary interstitial inflammation—the case in which Meyer found minute nodular swellings in the nerves. This case is exceptional, but there are some other facts of pathological anatomy that enable us to understand the occurrence of such a process. We have seen that in some acute cases there is an accumulation of bacillary or micrococcal elements in the vessels and in the nerve sheaths, and it is evident that we have here a process capable of explaining more or less random and excessive alterations outside the nerve elements themselves, in both the nerves and the nerve centres—alterations in the interstitial tissue, in the nerve sheaths, or in the vessels. This may also afford an explanation of the tendency to extravasation of blood that has been conspicuous in some cases, and has been more considerable in amount, and more peculiar in seat, than can be accounted for by the mere mechanical influence of asphyxial congestion. To the rule that the neuritis is not interstitial, there is, however, one common exception, that of the nerves of the palate. The palatine nerves are contiguous to the inflammation of the primary disease, and it is probable that they are influenced by the abundant growth of organisms in the false membrane, which often penetrate into the substance of the mucous membrane and even into the tissue beneath. Interstitial inflammation may ascend nerves, although the purely parenchymatous neuritis only descends them; hence it is not surprising that the palatine nerves should sometimes present evidence of inflammation through a considerable extent. Thus we must regard the essential lesion as one of the nerve elements, and the special implication of the connective tissue and the vessels as an associated and to some extent independent lesion.

The affection of the palatine nerves was long held to be the mechanism by which the whole of the nervous lesion was produced. It was thought that the inflammation ascends these nerves to the centre, and there spreads. But the discontinuity of the various palsies was always an objection, and, in fact, an insuperable objection, to this theory. The discovery of the lesion, and the demonstration that it also is discontinuous, have rendered the theory merely of historical interest.

The special susceptibility of the palate is not, however, altogether explained by the fact that the local inflammation may extend to the sheaths of the palatine nerves. A considerable time usually intervenes between the primary angina and the palsy of the palate, and the latter may be the first symptom of the palsy that follows diphtheria of a surface wound, when the throat affection is altogether wanting. From this it is evident that there is a special susceptibility of the centres or nerves of the palate to be influenced by the cause of the palsy. If this is true, it becomes a question whether the local inflammation has as much to do with the occurrence of palsy of the palate as the lesion of its nerves would suggest. The peculiar influence of some poisons on certain parts of the nerve centres is well known, and is illustrated in this disease by the paralysis of accommodation —a phenomenon as remarkable, if not more remarkable, than the paralysis of the palate. The lesion underlying the cycloplegia has not yet been discovered, and we do not know whether it is central or peripheral. It is easier to conceive that the isolated impairment of such a function is due to an influence on the centre, than that it is the result of peripheral nerve changes.

We are equally ignorant of the precise cause of the impairment of sensation. The analogy of the motor palsy suggests that it may be due sometimes to a central and sometimes to a peripheral lesion of the nerve elements. The cells in which the posterior root fibres end may be affected in the same way as are those from which the motor nerve fibres proceed. The peripheral limitation of the fields of vision, occasionally found, points to a central affection, and so also does such symmetrical distribution of anæsthesia as the affection of the palms and soles mentioned on p. 1225. We must remember that the influence, whatever it is, that causes recognizable alterations in structure, may also, in less degree, cause slighter and more transient impairment of nutrition and of function. The isolated loss of the knee jerk may be due to such an influence on the motor cells; when there is actual palsy, the motor lesion affords an ample explanation of the loss, since the symptom would certainly result from the structural changes discovered in these cases. An alternative explanation of the isolated loss is that there is an affection of the sensory muscle nerves analogous to that which probably causes the symptom in tabes (see p. 313). Distinct muscular anæsthesia has been observed in diphtheritic palsy, and an affection of these nerves, or of their central cells, affords the readiest explanation of the ataxy that is sometimes so conspicuous. According to this theory, the resemblance between post-diphtheritic ataxy and the condition of the tabetic patient is more than superficial, since we have seen that the incoördination of tabes must also be ascribed, in part at least, to an affection of these nerves.

The most important point in the pathology of diphtheritic paralysis, its relation to the primary disease, is still most obscure. For the remarkable facts that it almost always commences after the primary disease is over, and that it so often reaches its height two or three months afterward, there is at present no explanation. While the presence of organisms in the nerve sheaths and interstitial tissue suggests that the inflammation of these structures may be due to the irritation of an organized *materies morbi,* no appearances have been seen to suggest that this is the cause of the change in the nerve elements which is the essential cause of the palsy. Are these changes the result of some nerve poison left behind by the diphtheritic virus, a poison which multiplies in the body without causing general symptoms, and is certainly incapable of infecting another person? Or are they due to the diphtheria poison, bacterial or other, which acts on the nerve elements during the primary disease, although the effect on the nutrition of the cells and fibres does not show itself until some time after the virus has ceased its active ravages? Or are they due to some poison associated with that which causes the throat affection, of similar nature, but not necessarily proportioned to the latter—indeed, not necessarily coexisting with it—and producing its effects without pyrexia? To these questions at present no answer can be given. But it may be noted that the absence of any relation between the intensity of the diphtheria and of the subsequent paralysis accords best with the last theory, and this is supported, also, by a very remarkable series of facts recorded by Boissarie.* In a certain district of Paris there occurred a series of cases of severe diphtheria, and at the same time a series of cases of paralysis of the palate, eyes, limbs, heart, etc., perfectly like that which occurs after diphtheria, and accompanied by albuminuria. The remarkable fact is that in these cases of primary palsy there was no history of preceding sore throat, and in several of the cases distinct diphtheria followed the paralysis, which lessened during the throat affection. Some of the cases of primary palsy seemed to arise distinctly by infection.

DIAGNOSIS.—A definite attack of diphtheria, and the characteristic onset of the subsequent paralysis, render the diagnosis, as a rule, a simple matter. It is only when the nature of the preceding disease has escaped recognition that any real difficulty occurs. It must be remembered that an offensive discharge from the nose is sometimes the chief indication of diphtheria. But the onset by the affection of the throat and eye, and the subsequent progressive paralysis in the limbs, are so distinctive as to indicate at once the nature of the symptoms, and also that of the preceding sore throat, of which there is usually a clear history. In most cases of this character in which a mistake is made there has been a considerable interval between the throat affection and the palsy.* Greater difficulty is presented by those cases in which little attention has been paid, not only to the primary disease, but also to the early symptoms in the throat and eye, and in which these symptoms pass away before the limbs become affected. In such a case the symptoms may readily be ascribed to a primary disease of the spinal cord. The absent knee jerk and incoördination may simulate locomotor ataxy, or the weakness may be mistaken for simple paraplegia, or the wasting be ascribed to a subacute poliomyelitis. An important distinctive character is the manner in which the weakness spreads slowly from one part to another, often lessening in the part first attacked. In such cases careful inquiry will generally elicit a history of transient difficulty in swallowing and in reading, commencing not long after a sore throat. The change in electrical irritability is usually much slighter than in acute atrophic paralysis, while in the latter there is no affection of sensation, and there is a more sudden onset. The onset of diphtheritic paralysis is more rapid than is common in ataxy, from which, also, the marked weakness and the absence of much pain help to distinguish it. In simple paraplegia the knee jerk is usually excessive, and it is never absent unless there is marked wasting or anæsthesia in the thigh.

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* *Gaz. Hebd., 1881,* Nos. 20 and 21.

It is not often that there is any difficulty in distinguishing between diphtheritic paralysis and hysteria. The affection of the palate, almost invariable in the former, is never met with in the latter disease. But real difficulty may be occasioned by the fact that a true diphtheritic paralysis may be succeeded by hysterical palsy. Thus, in one case, paralysis of the palate and ciliary muscle had ceased, but was succeeded by general loss of power and of sensation, supposed, also, to be diphtheritic. The girl was lying in bed almost motionless, with universal anæsthesia and convergent strabismus from, it was supposed, a paralysis of both sixth nerves. Under emotional excitement, however, the limbs were moved with considerable force. The anæsthesia was universal in extent, a most unusual symptom in diphtheritic paralysis. Moreover, the eyes could be moved to right and left, but the convergence was maintained during the movement, and was thus clearly due to considerable spasm of the internal recti; such spasm is never met with in diphtheritic paralysis except in slight degree on an effort at accommodation. The diagnosis of hysterical paralysis was confirmed by the patient's rapid recovery under moral treatment. In such cases the knee jerk may give important guidance. Thus a girl presented herself with weakness of the left leg, and anæsthesia of both leg and arm, symptoms that had existed since an attack of diphtheria six months before. There was, also, loss of accommodation and of convergence. The latter was no doubt diphtheritic, but the knee jerk in the leg was normal, and this made it certain that the palsy of the leg was not diphtheritic, and probable that it and the anæsthesia were purely functional, an opinion confirmed by the previous history of the patient and the subsequent progress of the case. She had had a similar transient hemiplegic attack two years before, and faradaism removed the palsy in a few days.

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* Thus, a young man came to me with distinct symptoms of diphtheritic paralysis, the nature of which had not been recognized. He had lost a brother from diphtheria three months before, and the patient himself had suffered from a sore throat ten days before his brother was taken ill. He had not seen a doctor, considering that such affections "were better left alone."

The PROGNOSIS in diphtheritic paralysis is, on the whole, good, so long as there is not grave exhaustion from the preceding disease, evidence of cardiac paralysis, weakness of the muscles of respiration, or inability to take food. In each of these conditions there is danger. Considerable retardation in the heart's action is also a more serious symptom, and so is extreme frequency or irregularity. The failure of respiratory power is most grave when both the intercostals and the diaphragm become weak at the same time; the amount of mucus accumulating in the chest is an accurate indication of the degree of danger. Paralysis of the vocal cords is rarely a cause of death, because it is usually incomplete, but paralysis in the region of the superior laryngeal nerve is serious, because it is so often associated with pharyngeal paralysis; food readily enters the insensitive opening to the larynx, and may thus cause great distress, and even reach the lungs and set up a form of pneumonia. Paralysis of the muscles of the neck is a rather grave symptom, because paralysis of the diaphragm and pharynx so often occur in conjunction with it. The sooner the paralysis comes on after the primary disease the greater is the danger to life. If a serious form of palsy (*e. g.*, that of the heart) commences during the first week of the affection, the chances are much against the recovery of the patient.

The duration of an attack is difficult to foretell. As a rule, the more severe the palsy the longer will it last. If the paralysis of the palate is considerable and lasts some weeks, it is improbable that the limbs will escape, or that the attack will be over in less than two months. If the legs become very feeble, the arms will almost certainly suffer also. On the other hand, if the paralysis of the palate is brief in duration, there is a fair prospect that the limbs may escape. It must, however, be remembered that complete recovery from the early initial symptoms does not preclude the occurrence of others, but it is not probable that, in such a case, they will be severe.

The prognosis is more serious in children than in adults, chiefly on account of their smaller reserve of strength, and the greater difficulty of feeding them if complications interfere with the ordinary method.

TREATMENT.—Of first importance in the treatment of diphtheritic paralysis is restoration and maintenance of the patient's strength, already weakened by the previous disease. For this end feeding is at once the most important and the most difficult measure. Easily digested nourishment should be given at frequent intervals, with wine or brandy if the pulse indicates it. When the palate is paralyzed, pulpy foods are swallowed much better than liquids, and the affection of the palate does not usually interfere with a due amount being taken. A much more serious obstacle is the paralysis of the pharynx or of the epiglottis and upper part of the larynx. In the former, deglutition may be difficult or impossible; in the latter, food cannot be kept out of the larynx and attempts to swallow occasion so much distress that mental repugnance is added to the physical difficulty, and it may be impossible to give food in the ordinary way. It is indeed inadvisable to do so if particles of food and drink constantly get into the larynx, on account of the danger of pulmonary complication. Food must therefore be injected into the rectum, or given by means of a small œsophageal tube or large gum-elastic catheter. A flexible tube passed through the nose does not answer so well in these cases, because the paralysis often extends to the lower part of the pharynx, and it is absolutely necessary that the extremity of the tube should be below the paralyzed portion. Which method, rectal or œsophageal, should be adopted, must depend on age and condition, but it is absolutely essential that the patient should not be allowed to pass more than twelve hours without food being given, in sufficient quantity, by one or the other method. It is unwise, in the case of children especially, to wait in the hope that the child may presently be induced to take food. Few cases of severe diphtheritic paralysis can survive forty-eight hours' complete fasting, and the danger of producing exhaustion by the procedure of feeding is greater the longer the abstinence has continued.

The variable course of diphtheritic paralysis, and its ultimate tendency to recovery, render the influence of drugs upon it very difficult to ascertain. There is no evidence, at present, that any agent has a considerable influence on the morbid process itself. Tonics, especially iron and quinine, often seem to be useful. Strychnine has been largely employed, and sometimes seems of actual service. But it is certainly powerless to neutralize the morbid process in its early stages, and seems to be without influence on the spread of the disease. Moreover, it is not wise to give large doses of a drug that stimulates the nerve cells so powerfully. Belladonna in large and frequent doses has been thought by Abercrombie to do good.

Of local treatment for the paralysis the most important are rubbing and electricity. The slowly interrupted voltaic current should be used, to which alone the muscular fibres are capable of responding if the paralysis is severe. The current should, if possible, be of such a strength as will cause the muscles to contract. In the case of children, if an adequate strength causes severe emotional disturbance, it is far better to be content with a weaker current, as strong as can be borne without distress or alarm. The utmost good electricity can do is very small compared with the harmful influence of a daily fright.

Among special symptoms which may require treatment, the paralysis of the pharynx has been already considered. The loss of accommodation scarcely calls for treatment, and it is doubtful whether anything can be done for it. The application of eserine (a half per cent. solution in water) twice a day has been recommended by Herschel, but there is much doubt whether it has any influence on the duration of the affection. If there are symptoms of cardiac failure, the patient should be kept in the recumbent posture, since fatal syncope has occurred on sudden rising. For undue frequency of action small doses of digitalis may be given, the effect being carefully watched. Duchenne recommends faradaism to the præcordial region as a powerful means of stimulating a failing heart, and several other French writers have endorsed his recommendation.

Failure of the muscles of respiration is rarely so complete as to cause death directly, but when mucus accumulates in the chest, suffocating paroxysms are apt to occur, which are attended with some danger. If life is threatened in one of these, artificial respiration may carry the patient over the attack, and Duchenne recommends reflex stimulation of the respiratory centre by faradaism to the back of the chest. An account of a case in which the method was employed with success has been given by Millard.* The patient was a man aged thirty, and paralysis of the palate, diplopia and weakness of the limbs had existed for about three weeks, when suddenly distress in breathing came on; expiration was short and sudden, mucus accumulated in the air passages, causing a loud tracheal râle; the patient could not expel it, and asphyxia seemed imminent. Duchenne was called in, and, noting that the diaphragm acted well, and that the source of the trouble was the deficient expiration, faradized the skin at the back of the thorax, and thus "rapidly reëstablished the expiratory power, and caused the immediate expulsion of a quantity of bronchial mucus. The patient was immediately relieved, and, the application being repeated during the next few days, all alarming symptoms passed away, and he ultimately recovered."

HYDROPHOBIA.

Hydrophobia is the name given to a malady, when it affects man, which is called rabies when an animal is the subject of it. It is a specific disease, always due to a specific poison, always contracted from an animal, and, like syphilis, transmissible only by inoculation. It is manifested by symptoms that are chiefly due to a disturbance of the nervous system, and is almost always fatal.

RABIES IN ANIMALS.

Before describing the disease as it occurs in man, it will be well to give a brief outline of the characters of the affection in animals. The fullest description of it is that given by Fleming in his work on the disease.† The malady in animals, as in man, is invariably, or almost invariably, due to inoculation by a bite. It is the opinion of Fleming that it arises spontaneously in very rare instances, but this is not certain.‡ It occurs in dogs, wolves, foxes and cats, and by these it is commonly transmitted, especially by the first two. It occurs, also, in the herbivora and some rodents, but it is probably transmitted by them only in very rare instances. In the dog the first symptom is dullness, with an indisposition to move. Then the animal becomes shy and suspicious, or threatening in manner, and afterward irritable and restless, with a strong tendency to bite. Throughout the early period there is a depraved appetite; ordinary food is rejected, and hay, straw, cloth, wood and cinders, and even the animal's own hair, are eaten. This is a very important characteristic, because the presence of such substances in the stomach constitutes one of the most important signs by which the disease can be recognized after death.* The bark usually becomes changed into a peculiar howl, which begins with a short, low note and ends with a long higher note; it has a peculiar metallic ring. There may be drooping of the lower jaw and weakness of the muscles of deglutition; these may prevent the animal swallowing, but there is no dread of water. Ultimately palsy always comes on; first manifested by unsteadiness of movement, then by increasing weakness of the legs, until the creature cannot stand. It then usually becomes comatose and dies. A distinction has been made between "dumb or paralytic rabies" and "furious rabies," but the difference depends only on the degree of excitement; all cases, if permitted to run their course, end in paralysis. After death, distinct changes are often to be found in the nerve centres. There is congestion of membranes and in the cortex, with accumulations of lymphoid cells about the vessels, especially intense beneath the lower part of the fourth ventricle, where I have found the tissue in a state indistinguishable from that of acute inflammation. The changes are essentially the same as those to be described as often present in man. There may be minute extravasations, and sometimes hemorrhage visible to the naked eye. Similar but usually slighter changes are often found in the gray matter of the spinal cord. The larynx and trachea are usually congested, and so also are the lungs.

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* Landouzy, "Des Paralysies dans les maladies aigues," 1880, p. 87.

† Some additional facts, observed during a long series of investigations, are described by Dowdeswell (*Proc. Royal Society*, vol. xliii, p. 48). His description has been followed in the above account.

‡ Ibid., p. 450.

In the stomach, besides the peculiar contents already mentioned, minute extravasations are commonly seen in the mucous membrane. The salivary glands present no constant changes nor do other organs.

In the rabbit, according to Dowdeswell, the symptoms are dullness, followed by excitement, transient pyrexia, and progressive paralysis, which is the cause of death. The post-mortem changes are similar to those in the dog, but the stomach contains ordinary food.

Pasteur has discovered that the virus exists, after death, in the central nervous system as well as in the salivary glands, and Dowdeswell has found that it is also abundant in the peripheral nerves. Inoculation with these tissues produces the disease even more certainly than does that of the salivary glands. Pasteur has also proved that, in animals, the disease is produced far more surely, and with a more uniform incubation period, when the inoculation is made beneath the dura mater, by trephining the skull, than when it is subcutaneous. The period of incubation is very variable in animals, especially when the inoculation is subcutaneous. In intracranial inoculation it is generally about seventeen or nineteen days, and this whatever be the source of the virus. By passing the virus through a series of rabbits, the period becomes shortened to six or seven days, and remarkably uniform. The poison exists in the tissues only toward the end of the period of incubation. Nothing is yet certainly known of the nature of the virus. It is supposed by most observers that it must be a micro-organism, but attempts to discover and isolate it have been only partially successful.*

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* This sign has, indeed, been regarded as pathognomonic, and is usually accepted and employed as such. Doubts have, however, been thrown on its value. Gibier, *Gaz. Heb.*, *1884*, No. 29, etc., states that he has repeatedly failed to obtain evidence of the rabid state of animals in whose stomach hay and straw were found, when he employed the test subdural inoculation. Mr. E. Batt, Vet. Surgeon to the Brown Institution, informs me that this feature is not uncommon apart from rabies, and that it is very difficult to feel confident, from post-mortem signs alone, that an animal has died from this disease.

RABIES IN MAN.

Human rabies, or hydrophobia as it is commonly called (from the frequent dread of water), is invariably acquired from a rabid animal, and is almost invariably due to inoculation with the saliva. It never arises spontaneously in man. The inoculation is commonly effected by a bite. In nine-tenths of the cases the disease is contracted from a dog, in a few cases from cats, very seldom from a fox or a wolf. It has been due to a wound received in the dissection of a rabid animal.† It is not certain that the inoculation with the blood of a rabid animal will produce the disease. The activity of the poison appears to be rapidly destroyed by decomposition. After post-mortem decomposition has commenced, and cadaveric rigidity has passed away, it is said that the disease can no longer be produced by inoculation even with the saliva. It is certain, however, that the saliva, dried before decomposition, retains its virulence for a longer period, certainly for days.

A bite is more effective if upon an uncovered part of the body, as the face or hand, than if inflicted through the clothes, by which the saliva may be wiped from the tooth. Children are often bitten about the face, and a large proportion of those who are so bitten are attacked. It is said that a bite inflicted immediately after a preceding bite is less likely to infect, as the teeth may have been cleaned by the preceding bite. For inoculation, the poison has to come in contact with the blood or fluids of the body, and this is sometimes effected without an actual bite. The disease has been produced by a rabid dog merely licking a scratch upon the hand, and it has resulted from the teeth having been used to loosen a knot upon a rope with which a rabid dog had been tied up. A person has been inoculated by a bite from a healthy dog inflicted immediately after it had been fighting with a rabid animal, the saliva of which was no doubt inoculated. The malady has followed the scratch of a cat, probably by the animal's saliva having thus been introduced.

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* At least, so far as positive results are concerned. Gibier (*Comptes Rend., 1884*, vol. 98, and *Thèse de Paris, 1884,*) and also Dowdeswell (loc. cit.) found an organism in abundance in the central nervous system in some cases; it was extremely difficult to stain and recognize, and this, if it was really the microbe of rabies, explains the frequent failure to discover it. Dowdeswell obtained some cultivations from it, and one animal inoculated with the cultivated organism seemed to be protected from the effects of the active virus (*Lancet, 1886*, vol. i, p. 1112).

† For an instance see Bollinger, *Ziemssen's Handbuch*, Bd. iii, p. 542.

It has been said that hydrophobia has been produced by the bite of an animal not suffering from rabies. This is opposed to all that we know of the origin of other diseases. The statement rests upon some facts which seem to establish that hydrophobia may result from the bite of a dog which did not at the time, or for several weeks afterward, present the recognized symptoms of the disorder. The best explanation of these facts is probably that in some rare and exceptional circumstances, rabies may affect a dog as a transient insignificant malady, communicable, however, by inoculation.*

Not all those who are bitten by a rabid animal contract the disease. The difficulty of ascertaining the precise proportion is very great, on account of the frequent uncertainty as to the exact nature of the affection of the dog. It has been calculated that, of those bitten by certainly rabid dogs, 47 per cent. suffer; of those uncauterized, 83 per cent.; of those promptly cauterized, only 33 per cent. On the other hand, of persons bitten by dogs merely suspected to be mad, only 8 per cent. suffer.† The immunity of some persons is perhaps due to bites having been inflicted through the clothes, or to the animal's teeth having been cleaned by preceding bites, but the poison probably varies in virulence, and it is possible that there are individual differences in susceptibility, such as apparently exist in dogs.

The incidence of rabies is determined by exposure to the risk of bites, especially on uncovered parts. Hence more males than females suffer, the proportion being about four to one. The largest number of cases in males occur during the middle period of life, but very few adult women are affected. Children of both sexes often suffer; they are much exposed to the risk of bites on the face and hands from straying dogs, and no less than two-fifths of all cases are under fifteen.‡

The period of incubation varies between wide limits, and is longer and more variable than that of any other known acute specific disease. The common period is from six to ten weeks. Bauer has calculated the average period of 510 cases to have been seventy-two days, and rather longer in men (eighty-five days) than in women (sixty-five days). It is shorter when it is on the head or neck than when on the limbs.* In at least half the cases the disease develops in from one to three months after the bite. In a few cases the period is less than a month, the shortest observed having been about twelve days. Cases are not uncommon in which the period is more than three months—six, nine, and in some cases even twelve or eighteen.† Instances have, indeed, been recorded in which five, ten or twelve years are said to have passed; most authorities consider that in such cases there has probably been a second unnoticed infection. It is, however, certain that the disease may occur after an interval of a year or of eighteen months. It is difficult, therefore, to deny the possibility that an interval of several years may elapse. It is almost as difficult to explain an incubation period of one year as of five years.

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* Pasteur has observed apparent recovery in dogs, and in some cases of the kind a relapse of the disease occurred—once after an interval of some months.

† Bollinger, loc. cit., from the statistics of Tardieu, Thamhayn, and Bouley.

‡ During the twenty-five years ending 1872 (*Reg.-Gen. Rep., 1875*), 299 males and 74 females died from hydrophobia. 89 males and 33 females were under 15, = one third of the males, and four-sevenths of the females. The greater relative male liability is least (but still considerable) in childhood, and increases up to 45; the ratio between the sexes is 2 to 1 in the first five years, and 3 to 1 in the third: during the thirty years 15–45 it is 8½ to 1. In later life the preponderance of males is not so great.

SYMPTOMS.—During the period of incubation there are commonly no symptoms. Occasionally pain or unpleasant sensations are felt at the seat of the wound, explicable in part (but not altogether) by the attention which is directed to it. Mental depression is occasionally conspicuous, but is commonly the result of the sufferer's knowledge and anticipation of the possible consequences of the bite. At the onset of the acute symptoms, there may be considerable local pain, occasionally radiating up the limb. It may commence a few days or a week before other symptoms. Even more frequently all local symptoms are absent. Usually the first indications of the impending malady are a sense of general malaise, mental depression, disturbed sleep, and some discomfort about the throat—an occasional sense of choking, or a little difficulty in swallowing liquids. The attempt to drink occasions some spasm in the pharynx, which increases in the course of a few hours, and spreads to the muscles of respiration, causing a short, quick inspiration, a "catch in the breath," resembling that which is produced in health by an affusion of water. This increases in severity to a strong inspiratory effort, in which the extraordinary muscles of respiration, sterno-mastoid, scaleni, etc., and even the facial muscles, take part; the shoulders are raised, and the angles of the mouth drawn outward. As the intensity of the spasm increases, so does the readiness with which it is excited. It may be caused by the mere contact of water with the lips, and a state of cutaneous hyperæsthesia develops, so that cutaneous impressions, such as a draught of air, which normally excite a respiratory effort, bring on the spasm. The mere movement of air caused by raising the bedclothes may be sufficient. The patient is often unable to swallow the saliva, which is usually abundant and viscid, so that it hangs about the mouth and is expelled with difficulty; this greatly increases the patient's discomfort. Vomiting is common, a greenish-brown liquid being ejected. The attacks of spasm are very distressing to the patient, and the mental state which they occasion increases the readiness with which the spasm is produced, and in some cases the mere sight of water or the sound of dropping water will cause an attack. It may even be excited by visual impressions which cause a similar sensation, as the reflection from a looking-glass, or even a strong light. The sufferer's horror and dread of these excitants becomes intense. Thus this disturbance in the act of swallowing liquids, which constitutes, as it were, the first symptom and keynote of the disease, spreads, on the one hand, to mental disturbance, and on the other to extensive muscular spasm. In each of these directions further symptoms commonly develop. The spasm, at first confined to the muscles of deglutition and respiration, spreads to the other muscles of the body, and the paroxysms, at first respiratory, afterward become general, and assume a convulsive character, although still excited by the same causes. The convulsions may consist in general muscular rigidity, sometimes tetanoid in character, with actual opisthotonos, or they may be coördinated and closely resemble hysteroid convulsions. On the other hand, the mental distress passes into disturbance, in which, at times, the balance of reason is lost, and the distress gets command over the mind. The horror with which the attempts to drink and the causes of the distress are regarded, becomes transferred to the attendants by which they may have been produced. Actual delusions occasionally supervene, and there may even be wild delirium. The mental disturbance is most intense during the paroxysms of spasm, and the frenzied patient may spit his saliva at those about him, and often attempts to bite them with his teeth, making occasionally strange noises in his throat which have been thought to resemble the barking of a dog. The sight of a dog has been known greatly to intensify the mental excitement, and this, strangely enough, in cases in which the sufferer had no suspicion of the nature of his affection.

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* Bauer, *Münch. med. Wochensch.*, *1886*. If 27 doubtful cases of alleged very long incubation were included, he found the average of 537 cases to be 126 days. The period presents very little variation in the case of bites by different animals.

† Many well-authenticated instances of eighteen months' duration are on record. One is described by Tachard, *L'Un. Méd.*, *1885*, p. 953.

The mental disturbance is not always thus subordinate, as it were, to the other symptoms. There may be a variable amount of mental derangement almost from the first. There may be an unnatural suspicion, and some manifestations of ill temper, succeeded by a curious mental restlessness and loquacity, and some incoherence in the sequence of ideas. Delusions and hallucinations are usually late symptoms, but occasionally some false ideas are manifested early in the affection. Throughout the symptoms there are sudden variations in the mental state, which is almost characteristic of the disease; a complaint, perhaps couched in indignant language, is succeeded by an apology, and the frenzied patient may alternately attempt to bite his attendants and beseech them to keep out of his way, or to hold him fast, that he may not hurt them. Similar mental disturbance may be present in children, although its details necessarily differ.

As the mental disturbance increases, the respiratory spasm and difficulty in swallowing sometimes lessen, and may even cease. The convulsive attacks may also cease. The patient may die apparently exhausted by the attacks of fury, or, if life is prolonged long enough, the mental and muscular excitement may give place to a state of paralytic exhaustion, which seems to represent the paralysis that is so prominent in animals. Coma may come on, but the patient usually dies within a few hours of the diminution in the symptoms of excitement. Death, however, sometimes occurs earlier, from asphyxia during a violent paroxysm of respiratory spasm, or from sudden failure of the action of the heart, a result that is explained by the lesions found in the medulla oblongata.

Among occasional symptoms priapism deserves mention; it is not very common, but has attracted attention from ancient times.

The temperature is almost always raised. At the outset the elevation is trifling, and occasionally, throughout the disease, it remains moderate, 100° or 101°. More frequently, as the symptoms increase, so does the pyrexia, and it amounts to 103°, 104°, or 105°, and may even reach a still greater height. The urine very often contains albumen, sometimes as much as a quarter or a third. Sugar has been found in a few cases.

The duration of the disease has varied in fatal cases from twelve hours to ten days. The usual duration is from two to four days; one-tenth of the patients die in the first twenty-four hours, three-fifths die within three days. Cases exceeding four days in duration are rare.

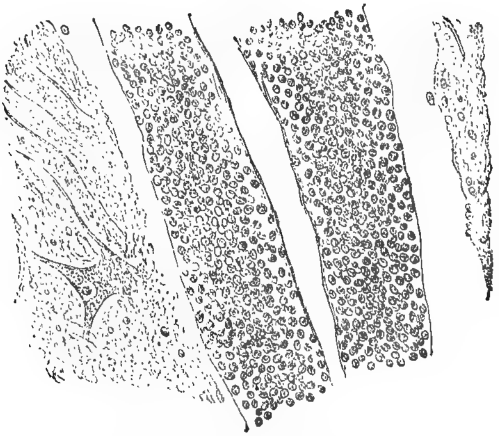
The course of the disease has been divided into two stages, the first distinguished by the respiratory spasm, the second by the mental disturbance and convulsions. By others, an initial stage of depression has been distinguished from the later stage of excitement. The final exhaustion is also regarded as a third or paralytic stage. But these stages are often not well marked; the elements that distinguish them may be combined in various degrees, and those of the later period are sometimes conspicuous from the first.

The special predominance of certain symptoms sometimes impresses a definite character on the attack, so that certain varieties may be distinguished. Their recognition is of some practical value, since it may prevent some of the frequent diagnostic doubts. The most important of these varieties depend on the predominance of mental symptoms on the one hand and of convulsions on the other. Delirious and mental excitement may exist from the onset of the affection, and in such cases the respiratory spasm may be less pronounced in the early period than is usually the case. The special character of the mental derangement varies greatly according to the mental condition of the patient and to the amount of special anxiety he has previously felt. There is almost always very marked mental distress, and the dread of the disease rather determines the direction of the morbid emotion than causes it. If there has been no apprehension of hydrophobia, as is often the case in children and occasionally in adults (especially of the lower classes), some peculiar delusion may be associated with the emotion, and may seem to be its cause when it is probably its consequence.

In other cases the convulsive symptoms predominate; the spasm induced by attempts to swallow quickly spreads to other muscles besides those immediately concerned, and the tetanoid character may be early assumed. In some instances, again, hysteroid convulsions occur almost from the first, and the respiratory spasm or mere emotion excites the coördinated convulsive movement seen in severe hysterical fits. This feature of the spasm may lessen as the disease develops.

In animals, as we have seen, symptoms of paralysis are much more prominent than they are in man, and sometimes they give a special character to the attack. The only example of a true paralytic form hitherto observed in man is that of a patient (Goffi) who died in St. Thomas' Hospital, and in whom the disease probably resulted from the inoculations of the Pasteur treatment (see p. 1250). The symptoms closely resembled those of "acute ascending paralysis."* The nature of the case would not have been suspected had not the inoculation test proved its character. This case suggests that some other instances of this mysterious form of palsy may have been the result of rabies—a possibility that should be kept in mind by those who meet with examples of this paralysis.

FIG. 335.



Hydrophobia; perivascular sheath distended with leucocytes; from the hypoglossal nucleus.

PATHOLOGICAL ANATOMY.—The blood is generally fluid, as in many acute diseases. The throat and pharynx are commonly congested, and so, in many cases, are other organs, especially the spleen and kindeys. The brain and spinal cord often present conspicuous congestion. The most important morbid changes are revealed only by the microscope,† and are found in the central nervous system. They are very variable in their degree, and are sometimes, although seldom, absent. Of nine cases I have examined, morbid changes were distinct in seven. These were indications of vascular disturbance; dilatation of small vessels, accumulations of leucocyte-like corpuscles around them and into the tissues, clots in the small vessels evidently formed during life, and minute hemorrhages. These changes are met with in various parts, especially in the cortex of the hemispheres, the medulla oblongata, and the spinal cord. They are always most intense in the medulla, between the eminentia teres above and the decussation of the pyramids below, and especially in the neighborhood of the pneumogastric, hypoglossal, and spinal accessory nuclei. The accumulations of leucocytes about the vessels is a very conspicuous change; they surround the outer wall, and may be so numerous as to fill up the whole space within the lymphatic sheath (Fig. 335); they may extend along the vessel for a considerable distance, and even pass into the adjacent tissue (Fig. 336). The nuclei contain a much larger number of such corpuscles than normal, and in places they may be aggregated and form dense masses (Fig. 337), which, since the corpuscles may be regarded as identical with pus cells, are, in fact, "miliary abscesses." These collections may sometimes also be seen outside the gray matter, scattered at random, as it were, especially about the root fibres of the nerves (Fig. 337). The thrombi within the vessels fill their

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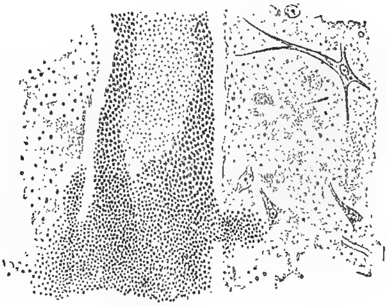
* See the *Brit. Med. Journal*, vol. ii, p. 830.

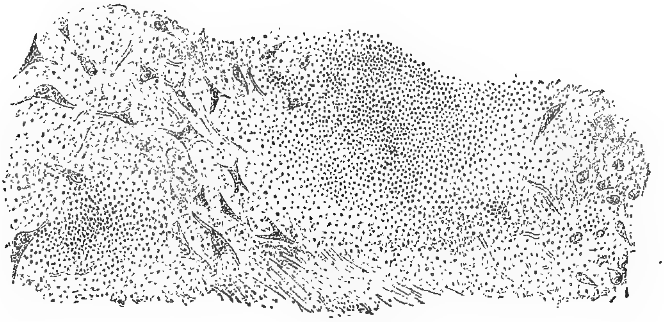
† They have been investigated and described by Coats, Clifford Allbutt, Benedikt, myself, and others. The figures here given are from a paper of my own published in the *Path. Trans.*, vol. xxviii.

FIG. 336.



Hypoglossal nucleus; leucocytes around a vessel, and extending into the adjacent tissue.

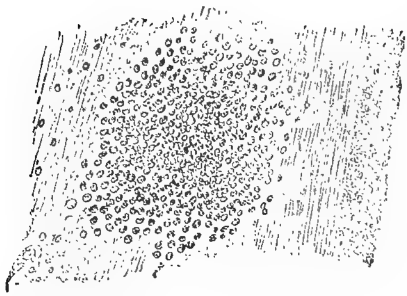
FIG. 337.



Cells of the hypoglossal nucleus on the right, of the pneumogastric nucleus on the left. Dense infiltration of the tissue with leucocytes.

cavity, and may present curved lines as if they had been exposed to the pressure of the blood. At the seat of these clots the walls of the vessel may be distinctly thickened. The nerve cells sometimes appear swollen and slightly more granular than under normal circumstances, but their alterations are trifling in the hardened specimens in which they are commonly examined. The hemorrhages are usually small, and only rarely sufficiently large to be visible to the naked eye. In the spinal cord the alterations are usually much slighter, and are confined to encrustation of the vessels with leucocytes, and to an increase in the number within the gray matter. I have seen them in one case so intense in the anterior cornua that its aspect resembled that of acute myelitis. The "miliary abscesses" are very seldom seen in the cord.

Fig. 338.



Accumulation of corpuscles ("miliary abscess") in the fibres of origin of the hypoglossal nerve.

Outside the nervous system leucocytal infiltration has been seen in the salivary glands and in the kidneys.*

Pathology.—Beyond the fact that the disease is the result of an animal poison, which is not volatile and can only be transmitted by inoculation, we know nothing of its nature or mode of action of its cause. From the variability and long duration of the period of incubation it has been thought that the poison is not at once generalized, but lies in the wound, probably undergoing some process of development, by which, at a future time, it passes into and affects the system. But neither this nor any other theory really explains the extraordinary variations in the incubation period. Nor is it easy to understand why the virus should remain in the wound, when it has as much opportunity for rapid passage into the system as other poisons of the same class. Moreover, cauterization of the wound more than a quarter of an hour after the bite usually fails to prevent the development of the disease. Even excision a few days after the bite is often ineffectual.

The symptoms indicate a primary action on the nerve centres, especially on the respiratory region of the medulla, and extending in a slighter degree to the brain above, and to the spinal cord below. Death is due to the effects of the over-action of the respiratory centres, or to the exhaustion consequent on the over-action of the brain, or to the secondary effects of the damage to the vital structures of the medulla. This opinion, based on the symptoms, is confirmed by the changes revealed by the microscope. The vascular changes and leucocytal infiltration are so intense in some cases as to amount to the anatomical signs of inflammation. Their variability in degree in different cases, and the fact that they may be in some extremely slight, and even in rare cases altogether absent, show that they are to be regarded as effects, rather than as causes, of the disturbance of the nerve elements. These must be ascribed to the influence of a poison reaching the brain through the blood, but accumulating and perhaps developing there, and causing an intense derangement of function. The morbid appearances in the nerve centres have been thought to resemble those of tetanus, but they differ in the far greater tendency to the extravasation of leucocytes, and in the special intensity of the changes in the respiratory region of the medulla.

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* Coats, Hale White, etc.

The primary effect on the medulla appears to be to greatly increase the excitability of the respiratory centre to reflex impressions, and, as the extraordinary muscles of inspiration (and not the diaphragm) are chiefly excited by reflex influences, the inspiratory efforts are of the "costo-superior" type. The centre for these movements is intimately connected with that for deglutition, and the region of the medulla in which the changes are most intense is that in which the glosso-pharyngeal, pneumogastric, and hypoglossal nerves are situated. The effect of the poison seems to be chiefly on these centres, just as other organic poisons seem to have specific actions on other parts of the nervous system. The "diminution of resistance" that results may spread to the structures through which tetanoid convulsions are produced. The vascular changes that are revealed by the microscope (and, as we have just seen, must be regarded as secondary results of the functional over-action*), are somewhat random in distribution; the exact points at which they occur are apparently influenced by conditions which are, so far as the disease is concerned, accidental, the general poison only determining the region involved. When they do occur, however, the leucocytal infiltration may be so great as to break up the tissue, and to constitute a minute point of suppuration, and if the point thus affected is important, grave consequences may ensue. We can understand thus, by these secondary changes in the nucleus of the pneumogastric, the occurrence of cardiac failure out of proportion to the other signs of exhaustion. It is probable, also, that the cortical excitement acts upon the medullary centres and increases their disturbance.† We know how much the functions of the pneumogastric and of the respiratory centres are under mental influence; the sigh, and vomiting from disgust are examples. On the other hand, it is possible that the disturbance of the medulla may determine the direction of the first cerebral derangement, and may cause the peculiar effect of the sight of water.

Some of the mental and physical disturbance of hydrophobia not only resembles but seems to be identical with that of hysteria. The convulsion may be distinctly of this character, and the frenzied horror at the sight of water has a close parallel in the maniacal stage of many hysterical attacks. In connection with the barking and biting of the hydrophobic patient, it should be remembered that both actions may occur during the attacks of pure hysteria; the biting is especially common.‡ There is nothing in itself extraordinary in hysteroid disturbance forming part of the functional disturbance of the brain that results from a different cause.

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* Hence it is incorrect to speak of inflammation of the medulla as the effect of the poison and the cause of the symptoms, and to make a comparison, as has been done, between the condition of the medulla in hydrophobia and that of Peyer's patches in typhoid fever.

† Putnam, *Boston Med. and Surg. Journal, 1879.*

‡ See the description of this disease.

Diagnosis.—The symptom of greatest value, as indicative of the nature of the affection, is unquestionably the peculiar respiratory spasm excited by attempts to swallow liquids. The history of a bite usually gives significance to definite symptoms, but occasionally misleads when these are equivocal.

In certain diseases of the throat and chest, sudden dysphagia may occur, which might give rise to the suspicion of hydrophobia. The difficulty has been described in œsophagitis and in pericarditis. The cases are those in which the dysphagia is not due to pain, but is a reflex difficulty, a throat spasm. Such cases are, however, very rare, and the history and other symptoms will be distinctive, if ordinary care is used. It is more likely that these affections will be mistaken for hydrophobia than that the latter disease should be overlooked. Cases in which the mental disturbance preponderates may be distinguished from simple insanity by the associated (significant even if slight) spasm, and by the onset after a suspicious bite. It is possible that there is a form of disease in which spasm is absent. The distinction of such cases from simple insanity may be most difficult and only to be made by the extremely rapid course of the affection. No patient ever passed from mental health to a state of imminent danger in two days, in consequence of simple insanity.

Organic brain diseases, meningeal hemorrhage, for instance, occurring in a person who had been bitten by a dog, has been mistaken for hydrophobia. One case is recorded in which the mistake was only discovered at the post-mortem examination. The character of the convulsions and delirium, and the absence of respiratory spasm, should prevent the error. From tetanus, hydrophobia is distinguished by the late period after the bite at which the symptoms develop. A difficulty can scarcely arise (except in the case of idiopathic tetanus) unless the patient has received some recent wound of another nature. The respiratory spasm and aversion to liquids, in the one case, and the early trismus in the other, almost always suffice for the diagnosis. If the symptoms of hydrophobia are present, the association of general tetanic spasm with the respiratory disturbance is quite compatible with the hydrophobic nature of the case.

Symptoms bearing a superficial resemblance to hydrophobia sometimes occur in those who have been in great dread of the disease in consequence of a bite from an animal possibly or certainly rabid. The knowledge that difficulty in swallowing is a symptom of the disease determines the occurrence of spasm in the throat on deglutition, perhaps analogous to the "globus hystericus." The patient's fears are intensified as the spasm increases in degree, and a dread of water may come on. Hysterical convulsion may be added, and the group of symptoms produced may deceive even the medical attendant. In this "lyssophobia," or "pseudo-hydrophobia" as it has been termed, the most important diagnostic element is the absence of all true respiratory spasm; there is not the peculiar "catch in the breath" that characterizes the genuine disease. There has always been much preceding mental alarm, but too much weight must not be placed on this, because great anxiety of necessity often precedes true hydrophobia.* The symptoms of the spurious form commonly soon subside if the mind of the patient can be tranquillized, or if the patient has recourse to some method of treatment in which he can be induced to place confidence. An instance of this is afforded by the case of Dr. Buisson, who after receiving on a scar some saliva from a patient who was suffering from hydrophobia (real, or more probably supposed) thought himself suffering from the disease. He took a vapor bath and was well. He afterward treated forty cases of the same character successfully in this manner. But there are other cases, regarded as spurious, in which the mental disturbance is intense, spasm distinct, and the patient passes into a state of exhaustion and dies in a few days. It is possible that many of these cases are genuine, although they have been published as spurious.†

I have more than once known a first attack of hysteroid convulsion, in a lad who had been bitten by a dog some time before, to be mistaken for hydrophobia, on account of the barking and biting and general convulsion. The absence of the respiratory spasm, and the intermitting character of the disturbance, should prevent error, which, indeed, can only occur if the characteristics of severe hysteria are not known.

PROGNOSIS.—Attacks of hydrophobia differ in their intensity and in the rapidity of their course, but at present all we can say is, that in any given case, however mild, only one issue can be looked for. It is to be hoped that the future may render the prognosis less grave, although we seem as far as ever from an actual cure for the developed disease. Still, we may echo the hope expressed by Boerhaave nearly two hundred years ago, "Nor ought we yet to despair of finding out, some time or other, a peculiar antidote for this poison, seeing we have succeeded in poisons thought formerly as destructive."

TREATMENT.—*Preventive.*—The prevention of a disease is important in proportion as its treatment is ineffectual; and the fatality of hydrophobia renders its prophylaxis of paramount importance. The prevention of hydrophobia is the prevention of rabies, and this could be readily effected in Great Britain on account of our insular position. The enforced muzzling of all dogs for a period of one year would almost certainly stamp out the disease.* It cannot be doubted, also, that the number of dogs is vastly in excess of any real need of the community. Most cases of hydrophobia are due to animals kept for pleasure, not for use, and often ill kept. More might also be done to make those who keep dogs familiar with the early symptoms of rabies, and to render it a criminal offence to permit a dog that presents such symptoms to be at large.

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* There has more often been a tendency to regard the genuine disease as imaginary than to mistake the spurious for the genuine. This tendency is especially marked among critics who have not seen the case, who do not scruple to cast doubts on the nature of even fatal cases. It is not certain that death has ever occurred from mere "lyssophobia."

† The diagnosis of the spurious from the genuine form of the disease is thus a matter on which there is very wide difference of opinion, which involves also the diagnostic indications. This difference will probably be lessened considerably now that we have an apparently crucial test in the results of inoculation. Two fatal cases, described as "pseudo-hydrophobia," have been published by Broadbent (*Clin. Soc. Trans.*, Feb. 23d, 1883), the spurious character of which appears to me to be open to question. In one case, it is true, the bite was received five years before, but this ought not to be allowed to *determine* the diagnosis (see p. 1241).

Until this wise and humane measure, the universal use of the muzzle, is adopted, the prevention of hydrophobia centres in the method devised by Pasteur, and in the cauterization of the bite. Pasteur's system of prevention depends on the fact that by drying the spinal cord of rabbits that have been rendered rabid by inoculation, the virus becomes less active ; the subcutaneous injection of an emulsion of such a cord (dried for fourteen days) causes no definite symptoms of the disease, nor do subsequent inoculations or injections made with cords dried for a less time. After such inoculations an animal is "protected," and does not suffer from rabies if afterward bitten by a rabid animal, or even if inoculated with the virus in its most active state.† These conclusions have been confirmed by Horsley,‡ in a series of experiments on dogs, which seem to be free from any source of fallacy. In these experiments the preventive treatment was carried out before the infection. Pasteur further believes that such a course of injections is effective in preventing the occurrence of the disease in a person who has been already inoculated with rabies, and he has treated thus many thousands of bitten persons. The efficacy of the treatment has been the subject of much discussion, and so, also, has its safety. Two facts only are certain. First, the danger that rabies may be communicated by the treatment is very small. There is, indeed, one case in which there can be no doubt of this result, the case of the man employed at the Brown Institution, who was bitten by a rabid cat, and died of paralytic rabies in St. Thomas' Hospital (see p. 1244).§ But this case only brings into relief the immunity with which the course of treatment has been pursued in an enormous number of cases.* The other fact is that the treatment is not invariably successful. In several instances rabies has developed at about the same time, after the original bite, that it might be expected to develop had the treatment not been adopted, and its severity has not apparently been modified. The efficacy of the treatment has therefore to be judged from statistics, and the difficulty of drawing a conclusion is very great. Of a given number of persons bitten by a mad dog, only a proportion, and sometimes only a small proportion, suffer afterward from the disease (see p. 1240). But the proportion who have suffered after undergoing the Pasteurian treatment seems to be far smaller than it would be in a like number of persons who had not been thus treated. Up to the end of 1886, 2682 persons had been inoculated, and the subsequent mortality from rabies among them was between 1 and 1.2 per cent. But even allowing largely for doubtful bites, it is estimated that not less than 5 per cent. of such a series would have died without the treatment.† We cannot feel sure that in all cases the animal that inflicted the bite was actually rabid, and hence a new source of uncertainty is introduced, the influence of which cannot be accurately assessed. The Committee on Hydrophobia investigated the facts of ninety unselected cases treated by Pasteur, and in two-thirds of these there was evidence which satisfied the Committee that the dog was rabid. In no less than twenty-four, the bites were on naked parts, and the wounds were not cauterized or treated in any way likely to prevent the action of the virus. Every one of these ninety patients had continued well, although all had been bitten more than six months, and most of them more than a year before. In such a number of cases of this character it is calculated by the Committee, on the lowest estimate, that not less than eight would have suffered from hydrophobia. The conclusion from the cts at present available seems, therefore, to be that the treatment is of great lue, although it does not secure absolute safety, and that the method is not tended by more than an extremely small risk of the production of rabies.

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* That such a measure is not adopted is a national disgrace, which is accentuated by the fact that the Government derives part of its revenue from a tax upon dogs. The opposition to the use of the muzzle is one of the strongest developments of morbid sentiment. There are apparently thousands of well-meaning people who would prefer that hundreds of dogs should perish every year of a painful malady, that many human lives should be annually lost, and scores of persons should be subjected for months to acute mental agony—rather than that dogs should be made to wear an apparatus which causes them a trifling annoyance for a few days. This perverted sentiment ought to be met with universal abhorrence, as a disgrace to humanity.

† Pasteur puts forward a theory which has, however, nothing to do with the accuracy of his facts. He believes that the virus is a microörganism, which produces, in its development, some substance by which its growth is checked, as in the growth of yeast by the alcohol produced. He thinks that the spinal cord contains both the virus and the antidotal product, and that the antidote is much less influenced by the process of drying than is the virus.

‡ "Report of Committee of Inquiry into M. Pasteur's Treatment of Hydrophobia," 1887.

§ The result has been ascribed to the circumstance that the man was almost constantly drunk while undergoing the treatment.

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* It has been suggested that some of the deaths from the ordinary form of rabies, after the treatment, were due to the inoculations, but the case referred to renders this doubtful. It suggests that the inoculation with the rabbit's cord would produce the paralytic, and not the ordinary form.

† "Report of Hydrophobia Committee," p. 3. Some further facts are very important. Of 233 persons bitten by animals in which rabies was certain (either by inoculation from the spinal cord or from the occurrence of rabies in other animals or persons bitten) only four died, while without inoculation forty would probably have died. The mortality among persons bitten by rabid wolves is exceedingly high; the bites are generally severe, and prompt cauterization is impossible; forty-eight persons so bitten were treated; a mortality of thirty would have been almost certain, but only nine died, and in three of these the symptoms commenced while under treatment. One very striking group of cases is that of six children bitten at Bradford, January 24th, 1886, by a dog proved to be rabid by experimental inoculation, and by the fact that another person who had been bitten by the same dog died of hydrophobia on March 4th of the same year. The six children who were treated were perfectly well at the date of the report, eighteen months after the bites. A more crucial test could hardly be devised. Almost as strong is the instance of three children bitten at Shipley, Yorkshire, in March, 1886, and treated by Pasteur, all of whom remained well a year later, while another person bitten by the same dog and not treated, died from hydrophobia seven weeks after the bite.

Treatment of the Bite.—The poison is deposited in a wound, and if it can e destroyed before it passes into the blood, the disease will be prevented.[*] he measures to be adopted for this purpose belong to the province of surery, but may be briefly mentioned. If the wound is on a limb, the irculation should, if possible, be arrested by a ligature around the limb pplied immediately. Free bleeding should be encouraged and the ound should be freely washed. These measures are most important, ecause it is not often that the cautery can be immediately applied. The use f these measures in the case of bites of every kind ought to be part of the ducation of children. The wound may be sucked, if there is no abrasion the mouth, which should be washed out each time. Absorption of poison rough a mucous membrane (to judge from experiments on the conjuncva) occupies several minutes. As soon as possible, the wound should be oroughly cauterized by the actual cautery, nitric acid, or nitrate of silver. henever practicable, the bitten part should be excised.

The treatment of the developed disease has to be directed chiefly to the iminution of the suffering of the patient. At the same time, recovery canot be said to be impossible. It is certain that cases have recovered in the ast, and it seems therefore certain that cases will recover in the future. owever slender may be the hope of doing more than lessen suffering, the atient should not be given over into the hands of death, even indirectly, y efforts exclusively directed to the euthanasia, still less ostensibly, as by the ather beds of old.[†]

The pathological lesions show that the functional nervous excitement has rganic consequences, and it is important, in order to lessen these, and to aintain the strength of the patient, that whatever excites the paroxysmal isturbance of the nervous system should be carefully avoided. A dimly-ghted, quiet room should be secured, and only the necessary attendants hould be admitted into it. Every sight or sound likely to disturb should e avoided, and it would probably be wise, if the spasmodic dysphagia is reat, to feed only by peptonized enemata.

The drugs that have been employed in the treatment of the disease are innumerable, and embrace (besides most known sedatives) a number of alleged specifics, all of which have been abundantly proved to be useless. The treatment employed in the few cases in which recovery has taken place has been found powerless in all other instances.

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[*] This is the common theory of cauterization, but is hardly consistent with the assumed calization of the poison in the wound during the period of incubation. It is, however, ertain that cauterization is of little value unless it is almost immediate. Promptness is of ore importance than the agent employed. A smoker's "vesuvian" would probably be uite effectual if instantly used.

[†] It is curious to note how the name of the disease preserves the fact that the horror of ater was formerly regarded not only as a characteristic of the disease, but as the predomiant element in its pathology, which afforded the chief therapeutical indication. "In the tervals" (of venesection, says Boerhaave) "he must be blinded and thrown into a cold ond, or be made wet with the continual throwing of water upon him, till he doth not seem ny more to be afraid of water, or but little," a final reservation that was at least prudent.

One case is said to have recovered under the influence of mercury, but this has been tried since in numberless instances without result. Curara, however, has been credited with three cures (Offenburg, Polli, and Watson), of which one case, that of Offenburg, seems undoubtedly genuine. The drug was first recommended half a century ago by an Englishman, Sewell,* but tried in small doses it failed. It arrests the hydrophobic spasms by paralyzing the motor nerves, and this may render artificial respiration necessary. There seems to be a remarkable tolerance of curara in this disorder. The dose commonly employed has been from $\frac{1}{16}$ to $\frac{1}{2}$ of a grain repeated every quarter or half an hour until there is general muscular paralysis, and renewed as this effect passes of. Polli administered in all three grains in five and a half hours to a child twelve years old. Even grain doses have failed to cause paralysis.† One case, probably genuine, has been recorded, in which recovery occurred under the use of Calabar bean and hypodermic injections of morphia (Nicholls). Bromide of potassium has very little influence; if given at all it should be in large doses, one or two drachms. Chloral gives some relief and, in a case recorded by Sansom, life was prolonged for ten days under its use. It is probable that the combination of chloral and morphia, which has a special influence on the respiratory centre, deserves further trial, and on the whole constitutes, as far as is at present known, the wisest treatment.

Tracheotomy was suggested by Marshall Hall as a means of averting death during an asphyxiating attack of spasm, but it is probably powerless. The spasm involves the chest wall, and according to Pitt,‡ the glottis is widely open during the attack.

Of the various remedies that have been held in popular esteem or vaunted as private specifics, little need be said. They furnish a curious chapter in the history of charlatanism and credulity. "Hitherto," we may still say with Boerhaave, "we have not met with any one which deserves credit enough to be entrusted with the life of people thus miserably afflicted, for no one is known whereof the experiments be certain, but they owe their birth either to speculation, or they have been copied from others, and taken upon trust." The list of reputed remedies that he gives has received a long addition since his time. Persons who fancy that they are suffering from the isease are cured by these measures, and their recommendations are repeated ith credulity by those whose readiness to express an opinion is inversely roportioned to their capacity for forming a judgment. Foremost among ese measures is the vapor bath of Buisson, the origin of which has been lready mentioned (p. 1249), and which every few years makes its reappearnce in the public papers.

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* And also by Waterton, the naturalist, who brought to England a supply of the drug (see "Dolan's Hydrophobia," 2d ed., p. 170).

† But it is possible that in some cases the agent has not been very active. Thus, in a case recorded by Bristowe, in which as much as 1½ grain was injected at a time, and 8 grains in twenty-four hours produced very little effect, the curara "had been in stock some time, and there was reason to believe that it had deteriorated in quality" (*Brit. Med. Journ.*, April 28th, 1885). The activity of the specimen should always be tested.

‡ G. N. Pitt, *Med. Times*, June 20th, 1885.

The saliva of persons suffering from hydrophobia has been proved to be apable of communicating the disease to animals. Hence, the attendants ould be cautioned to have no uncovered abrasions on the hands, and to ash from the eyes and face any saliva that may have been spit on them. f they are bitten by the patient, the wound should be treated as if it had een inflicted by a rabid animal. These precautions remove all danger, and ny anxiety that is felt may be removed by the assurance that of the thounds of persons who have attended on patients with hydrophobia, no authenic instance has been recorded in which the malady was thus contracted.* or has it ever been known to have been acquired through making a post-ortem examination on the bodies of persons who have died from the isease.

METALLIC POISONING.

Many metals, when taken into the body, cause chronic disturbance of the ervous system, in addition to the acute symptoms that are produced by a onsiderable dose of the poison. The latter are not considered here, since hey belong to the province of toxicology. The more chronic disturbance enerally results from the often-repeated entrance of small quantities of the etal, and very seldom succeeds a single large dose. The chief metals that fluence the nervous system are lead, arsenic, silver and mercury.

LEAD POISONING.

Etiology.—The occurrence of symptoms of lead poisoning seems to be, o some extent, determined by individual peculiarities. Of a given number f persons, all of whom have been exposed to the same influence, and xhibit indications of the presence of lead in the system, some will suffer onsiderably, some slightly, others not at all. Little is known of the conitions which thus influence the result, but it is probable that the weakly ffer more than the strong, and that those who inherit a tendency to gout ffer more than others. Most cases of lead poisoning occur in males, but is is solely in consequence of the larger number of males engaged in occuations which involve exposure to the poison.

The duration of exposure before symptoms occur varies between wide mits, and is chiefly influenced, no doubt, by the amount of lead daily osorbed. When this is large, severe symptoms are often induced in a few weeks. Some of the most acute cases occur in young persons whose exposure has been brief. It is probable that, when the daily dose of the poison is small, a certain amount of tolerance is sometimes established, which may be ultimately overcome in consequence of the accumulation of lead in the system, or in consequence of some other impairment of the general health.

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* Indeed, even the danger of bites seems small. A child, whose malady was verified by e inoculation test, bit two persons whose wounds were uncauterized, and at the end of six onths they were still well (*Riv. Clin. di Bologna*, Aug., 1886).

The sources of lead poisoning are very numerous, but may be divided into two groups, the *industrial* and the *accidental.* Of the former, the most potent are lead works, especially those in which white lead is prepared, but more frequent are the various industries in which prepared lead is used. Painters, plumbers, type founders, compositors, and those who glaze pottery with lead, are the most frequent sufferers. Glass grinders may also suffer, lead being a constituent of most kinds of glass. The *accidental* sources are extremely numerous; the most frequent is the contamination of drinking water by leaden pipes, or by lead-lined cisterns.* Acid fruits, cooked in glazed earthenware vessels, have become charged with lead; shot left in wine bottles, after cleaning, has been dissolved by the wine; snuff may be contaminated in consequence of being packed in lead paper. Lead enters into the composition of some hair dyes and cosmetics, and cases of lead poisoning have occurred from their use. Lastly, symptoms of chronic poisoning have occurred from the continued medicinal administration of lead.

The most frequent path by which lead enters the system is the alimentary canal, not only by the accidental contamination of articles of food, but also through uncleanliness. Workers with lead often neglect to remove all trace of lead from their hands before taking food, and thus become poisoned. This is shown by the influence of enforced cleanliness, which lessens the amount of lead poisoning among such artisans. Lead may, however, enter through the skin, as in poisoning from the use of cosmetics and hair dyes; it may be dissolved by some of the organic acids secreted by the cutaneous glands, or get through the skin in consequence of the friction often employed in rubbing hair dyes into the roots of the hair. Lead poisoning has been produced in children by the continued use of Goulard's lotion. Lead may be thus absorbed by those who work with the hands immersed in a preparation of the metal. It may certainly enter by the mucous membrane of the nose (in the case of poisoning by contaminated snuff) and probably also by that of the other air passages; inhaled particles are dissolved by the alkaline secretion, or enter the tissues and are dissolved by the carbonic acid contained in the blood. No doubt, however, some lead, entering by the air, is carried by the buccal secretions to the stomach.

It is commonly believed that, however lead enters the system, it acts on e tissues through the blood, permeating the whole body and influencing iefly certain structures. Unquestionably, most of the symptoms must be us produced. But lead has been said also to have a local influence on the art where it comes in contact with the skin. This opinion, although based n some curious facts, has not received general confirmation.*

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* Various chemical peculiarities of water will make it take up lead more readily. For particulars, the reader is referred to works on hygiene. A curious instance is related by Thorne, in which lead poisoning at Sheffield was due to the presence in the water of a peculiar acid derived from the soil (*Practitioner*, Dec., 1886, p. 465).

The lead which has entered the system collects in the various tissues and rgans, but not in the same degree in all. In dogs, to which lead is given food, the organs have been found to contain the metal in the following rder: bones, kidneys, liver, spinal cord, brain, muscles, intestine; and lsewhere only a trace (Heubel). It is doubtful, however, whether this rder is always the same in man. In one case, for instance, much more ad was found in the brain than in the liver, and none in the spinal cord Troisier and Lagrange); while in another case the liver contained much nd the brain none.† In one acute case lead was found in the spinal cord, ut none in the brain (Comby).

Lead is excreted by the liver, the kidneys, and also, it is said, by the ntestinal mucous membrane. Lehmann found that when it was injected nder the skin of an animal, almost all passed away by the bile.‡ Once ombined in the organs, it is very slowly eliminated, and its presence may e detected years after it has ceased to be taken in. It probably exists in hem in some combination with albumen.

Symptoms.—Disturbance of the general nutrition usually precedes other ymptoms, although it may escape observation. There is anæmia, often onsiderable, and depending on an actual diminution in the number of blood orpuscles. The hæmoglobin is reduced in proportion to the corpuscles, nd the anæmia of lead thus differs from that of chlorosis, in which the eduction is much greater in the hæmoglobin than in the corpuscles. Lead næmia resembles in this respect pernicious anæmia, and the resemblance s not merely superficial. The reduction in the number of the corpuscles is robably due to the accumulation of lead in the bones, the medulla of which s known to be one of the chief blood-making tissues of the body. Atrophy nd degeneration of the bone marrow has actually been found in lead poioning (Raimondi). The muscular strength lessens under the influence of he anæmia. As a rule the temperature is normal, but now and then there s a slight continuous pyrexia for which no other cause than the lead poisonng can be discovered. I have seen one case in which the temperature was onstantly 99°–100°, and three others with slight fever have been described y Renaut. When acute nervous symptoms occur, elevation of temperature s frequent.

Lead is excreted in part by the kidneys, and the urine undergoes alterations n its composition, the amount of uric acid being diminished. Hence the ubjects of lead poisoning often suffer from gout when they have reached middle or later life,* and the kidneys may become diseased, as in gouty subjects—indurated and granular. Apart from actual kidney disease, albumen may be present in the urine at times, as, for instance, during attacks of colic (Seguin). We do not know whether the lead poisoning causes the renal degeneration directly, or indirectly by the induced gout. But the effect is one of extreme importance, since it may lead to cardiac and arterial disease and thus to cerebral hemorrhage. By this mechanism, more frequently than by any other, lead poisoning causes death. Both renal disease and arterial degeneration are probably more common in saturnine than in ordinary gout.† The continued action of lead is not necessary for the production of kidney disease, and this may develop without any actual attack of gout. A gentleman suffered from wrist drop due to lead poisoning from drinking water; his urine was then normal. There was no further ingestion of lead and he recovered perfectly, but six years later presented all the signs of organic kidney disease.

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* The facts that suggest a local influence are mentioned at p. 1264.

† Pye Smith, in Fagge's "Manual," ii, 163.

‡ Lehmann, in *Diss. Berl., 1882.*

A characteristic symptom of lead poisoning is a narrow, bluish-black line on the gums, close to the teeth, distinguished by its narrowness and tint from the common zone of purplish congestion. The lead line usually forms where the gum is not in perfect union with the tooth, and the line, as seen, is the edge of a layer of deposit beneath the separated surface of the gum (Cras). It consists of sulphide of lead, deposited chiefly outside loops of blood vessels, and hence, when viewed with a lens, it often has a punctate appearance. The sulphur comes from albuminous matters which accumulate between the gum and the tooth (Tomes). If the gums are in perfect union with the teeth, and these are kept scrupulously clean, the lead line may be visible only on the projections of gum between the teeth, and it may even be absent entirely. A punctate deposit of sulphide of lead may form in the mucous membrane of the lip, upper or lower (as Garrod first pointed out). It is only seen when there is an accumulation of tartar on the teeth, with which the stained lip was in contact. A low form of stomatitis (accompanied by periostitis) has been occasionally observed in lead poisoning (Gubler, Anguelo).

Severe abdominal pain, "lead colic," is another frequent symptom. It is a paroxysmal pain, felt chiefly in the region of the umbilicus, sometimes over a more extensive area, and accompanied with obstinate constipation. During the paroxysms there is retraction of the abdominal wall, sometimes vomiting, and usually retardation and increased tension of the pulse. The constant constipation proves that the pain is not (as has been sometimes maintained) in the abdominal wall. Another theory regards it as pure neuralgia of the sympathetic plexuses, accompanied by an arrest of the intestinal contractions. The balance of evidence, however, is in favor of the theory, implied in the name, that the pain is a true "colic," the result of spasmodic contraction of the bowel, probably the colon—a contraction which is tetanic and not peristaltic, and therefore arrests instead of promoting the movement of the intestinal contents. The constipation is probably facilitated by a diminution in secretion from the mucous membrane, degenerative changes in which have been proved to occur in lead poisoning. Such tetanic contraction has been observed in animals under the influence of lead, but the mechanism by which it is produced is uncertain. It has been ascribed to the influence of lead on the sympathetic ganglia (Tanquerel), especially the mesenteric and cœliac plexuses (Romberg, Eulenburg), on the nervous ganglia within the intestinal wall (Harnack), on the muscular fibres of the bowel itself (Hitzig), and it has been supposed to be connected with the accumulation of sulphide of lead in the mucous membrane, sometimes visible after death.* At present there is no evidence to show which of these theories is correct, and it is therefore uncertain whether lead colic should or should not be classed among the nervous effects of lead poisoning. The retardation, and the increased tension of the pulse associated with it, are doubtless, in part at least, reflex effects of the strong afferent impression.

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* This was originally pointed out by Garrod, and has since been recognized almost universally. The clinical evidence of it is such that acquired gout increases very much the probability of lead poisoning in a doubtful case. It is not often met with, however, in young persons.

† Loriner, *Brit. Med. Journal*, July 24th, 1886.

Nervous Symptoms.—The definite nervous symptoms produced by lead poisoning are of three chief classes: (1) pains in various situations; (2) local paralyses with wasting of muscles; (3) cerebral symptoms, convulsions, delirium, coma; sometimes hemiplegic weakness, and hemianæsthesia.

(1) *Pains in the limbs* are very common, and may be referred to the muscles or the joints, or may be vague in seat. They are usually dull, aching, "rheumatic" pains, but sometimes sharper and neuralgic in character. They have been called "saturnine arthralgias," an unsuitable name, because (apart from gout) the joint pains are not usually prominent. There is often tenderness of the muscles, and occasionally tingling of the extremities, very rarely areas of anæsthesia; I have seen, for instance, diminished sensibility to touch in the back of one forearm and the front of the opposite leg. It is probable that such symptoms are sometimes the result of neuritis. Muscular "stiffness" may be associated with the pain, and now and then there is painful cramp in the calves. When the pains have a well-marked neuralgic character, in the arm or leg, sharp darting pain may occur along the course of the nerves, and exacerbations may be excited by pressure on the nerve trunks (Dreyfour).

(2) *Local muscular paralysis* with wasting is a very common consequence of lead poisoning. It presents two forms. In one, the most frequent, loss

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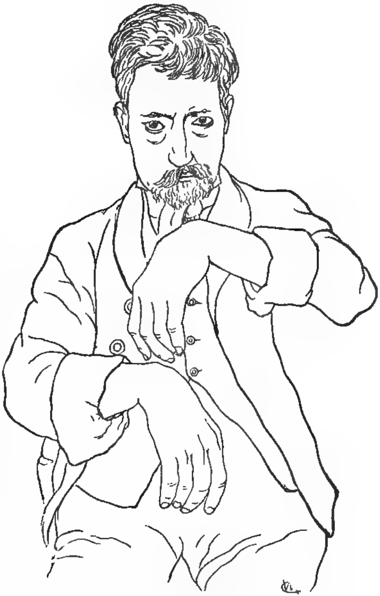
* Maier (*Virch. Arch.*, Bd. 90) found degeneration of the nervous ganglia in the wall of the intestine, and in the abdominal sympathetic, in rabbits in which chronic lead poisoning had been experimentally produced. He also found similar changes in a case of saturnism in man. The cœliac plexus was surrounded and infiltrated with connective tissue, and the sympathetic cells were in part atrophied, but the fibres were little changed.

of power precedes wasting, and the muscles present the "degenerative reaction," *i. e.* loss of faradaic irritability (from acute degeneration of the nerve fibres) and preservation of the voltaic irritability of the muscular tissue, just as in a traumatic lesion of a nerve. The common seat of this form is the extensor muscles of the wrist and fingers, and "wrist-drop" results. In the other form of saturnine atrophic paralysis the weakness and wasting come on simultaneously and proceed *pari passu;* faradaic and voltaic irritability are both lessened in proportion to the wasting, just as in progressive muscular atrophy. This form may affect any part, but is especially common in the small muscles of the hand. Between these two, intermediate forms are sometimes met with, in which a slight increase and qualitative change in voltaic irritability accompanies a moderate diminution in that to faradaism. We may distinguish the two varieties as the "degenerative" and the "primary atrophic" forms.

FIG. 339.



Wrist-drop from lead poisoning. (*From a photograph.*)

In the first, not only is the wasting secondary to the weakness, but some muscles may be merely weak, and may recover without undergoing atrophy. In the muscles at the back of the forearm wasting is almost invariable. This extensor paralysis is usually bilateral, although one arm is often affected a little earlier than the other. The right arm is the first to suffer, perhaps in consequence of the greater use of the right hand, since the left arm has been first affected in a left-handed man.* The rule is not invariable: I have known the left hand to suffer first in a right-handed patient. The interval between the affection of the two arms may be a few days, a few weeks, very rarely, several months; occasionally both are affected together. The paralysis usually comes on in a subacute manner, reaching a considerable degree in a few days or one or two weeks. The onset is more rapid in second than in first attacks, but it is never sudden, *i. e.* there is never immediate considerable paralysis. The first difficulty is in the extension of the fingers, often of the two middle fingers, sometimes of the first and second; but it soon extends to the others. The loss is that of extension at the metacarpophalangeal joints (by the long extensors); if the first phalanges are passively straightened, the distal phalanges can be extended without difficulty by the unaffected interossei and lumbricales. The thumb also suffers; extension of the phalanges (by the E. primi and E. secundi internodii pollicis) is lost, while its metacarpal bone can be still extended (or rather abducted) by the E. ossis metacarpi pollicis (abductor pollicis of the Germans). The weakness of the extensors of the fingers is usual greatest toward the ulnar side; the first finger may be extended best, and the little finger least. Soon the extensors of the wrist become weak; sometimes the radial, sometimes the ulnar extensor suffers first, with a corresponding defect in the lateral movement of the hand, and deviation in the attempt to extend it. The hand ultimately "drops," and cannot be brought up to the level of the forearm. As long as the special extensors of the wrist retain power, this joint can still be extended when the fingers are flexed so as to close the fist, although the wrist cannot be extended when the fingers are also extended. The reason for this seems to be physiological. When in health the hand and fingers are both extended; the special extensors of the wrist act very little, if at all; the movement at the wrist joint is effected by the long extensor of the fingers. If, however, the fingers are flexed, the extension of the wrist is effected by the special extensors. These facts may readily be verified by placing a finger on the tendon of the E. carpi ulnaris or radialis, during these movements. When the two muscles are paralyzed the wrist cannot be extended, even with the fingers flexed.

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* Bernhardt, *Deut. Arch. f. Kl.*, Band xxii, 363.

FIG. 340.

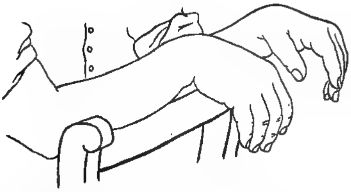
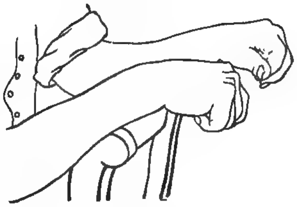


FIG. 341.



Wrist-drop from lead poisoning. Fig. 340 shows the maximum extension of the wrists and fingers; Fig. 341, the extension by the proper extensors of the wrist when the fingers are flexed.

The flexors of the fingers are unaffected. Nevertheless their action is seriously impaired by the inability of the extensors of the wrist to coöperate with them, and to maintain the extension of the carpus necessary for forcible flexion. Hence flexion of the fingers flexes also the wrist, and the course of the tendons is thus so shortened that the maximum contraction of the flexor muscles exerts but little force. The repeated flexion of the carpus produces a slight displacement backward of the carpal bones, and probably, also, a distention of the synovial sacs connected with them, so that a prominence forms over the carpus on the dorsum of the hand. It often alarms the patient, but is of no real consequence.*

In most cases of wrist-drop the paralysis is limited to the muscles mentioned—the common extensor of the fingers, the extensor indicis, the extensors of the phalanges of the thumb, and those of the wrist. All these muscles are supplied by the musculo-spiral nerve. But the supinator longus, also supplied by that nerve, almost always escapes, and so also, as a rule, does the extensor of the metacarpal bone of the thumb.

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* This swelling is sometimes termed "Gubler's tumor."

The affected muscles rapidly waste, and the back of the forearm becomes flattened, rendering the integrity of the supinator longus still more striking by contrast. The electrical reactions, as already stated, are those of nerve degeneration (see p. 65). There is loss of all irritability in the nerve, and loss of faradaic irritability in the muscles, while that to voltaism is preserved, for a time increased, and is commonly changed in quality. The anodal closure contraction occurs as readily, or more readily, than the kathodal closure, and a continuous tetanic contraction is readily produced during the passage of the current. Such a qualitative change may even precede the onset of the paralysis (Erb). Thus, in one early case, in which one arm only was affected, I found in it the early anodal contraction, while in the other, which was not paralyzed, the only change was the occurrence of tetanic contraction with an unduly weak current. The excessive degree of voltaic irritability passes away in the course of a few months, but some irritability remains for a year or two, even when there is no return of power, and it can often be quickly increased by sedulous treatment.

Although lead palsy is usually limited to these muscles, it occasionally invades the upper arm. The deltoid suffers most frequently, and sometimes before the forearm muscles. Occasionally the biceps and brachialis anticus suffer, very rarely the triceps. In severe cases, those muscles in the forearm may suffer which usually escape—the extensor ossis metacarpi pollicis, and even the supinator longus. Very rarely the supinators suffer early. Occasionally the small muscles of the hand are involved, but it is more common for their affection to be of the second variety, the primarily atrophic. Different parts of a muscle may be affected in different degree; thus the fingers may be variously paralyzed by the greater affection of certain parts of the common extensor, and one part of the deltoid may suffer more than another.

In the legs paralysis sometimes occurs, analogous to that just described in the arm. The muscles chiefly affected are those homologous with the forearm muscles, viz.: the long extensor of the toes, and the peronei muscles supplied by the peroneal nerve. But the tibialis anticus, although supplied by the same nerve, usually escapes, like the supinator longus in the arm. The affected muscles present the degenerative reactions already described. Slight deformity of the foot, talipes equino-varus, may result.

Occasionally muscles in which there is no wasting may be weak, especially the muscles of the upper arm and thigh (including the ileo-psoas). In such muscles there is often (although not invariably) a slight indication of the qualitative change in voltaic irritability already described (Erb). Now and then there may be universal loss of power, incomplete in degree. Incontinence of fæces and urine is occasionally observed. I have once met with persistent loss of power over the spinchter ani associated with anæsthesia of the skin about the anus and coccyx.

The second form, characterized by primary atrophy, occurs especially in the intrinsic muscles of the hand, but is sometimes extensive and irregular in its distribution, affecting many muscles in all four limbs. The wasting is slow, and accompanies, instead of succeeding, loss of power. Fibrillary twitchings are common, as in progressive muscular atrophy. The muscles still act to faradaism, although the irritability is lowered in proportion to the wasting; the voltaic irritability is usually lowered in a similar manner, but it is sometimes a little greater than the faradaic, and there is often a qualitative change. This form usually accompanies the degenerative extensor paralysis, but it may occur alone. It is much more obstinate than the degenerative form, and often persists long after the latter has recovered; it may indeed be permanent. For instance, a gentleman, poisoned by drinking-water, suffered complete wrist-drop on both sides, with wasting and loss of faradaic irritability in the muscles, the voltaic irritability being preserved. There was transient weakness, without wasting, in the deltoids. The abductor indicis had slowly wasted with merely diminution of both faradaic and voltaic irritability, and it remained in nearly the same state four years later, although the paralysis of the forearm muscles recovered in twelve months. An instance of extensive atrophic paralysis was afforded by a painter, aged thirty-two, with a well marked line on the gums, and a history of three attacks of colic. First the right wrist "dropped" and then the whole arm became weak from the shoulder downward. Four months later the left arm became paralyzed in a similar manner, and soon afterward both legs became weak. He came under treatment six months later; there was general wasting of the limbs, including in the arms the flexor muscles and supinator, the hand muscles being almost entirely gone. The muscles of the legs below the knees were much wasted. Everywhere faradaic and voltaic irritability was lessened in proportion to the wasting, and even in the most wasted muscles faradaic irritability was not entirely extinct. The patient was treated with electricity, iodide, etc., for six months, but there was no discoverable improvement in the state of the limbs.

As the cases just mentioned show, the course of the primary atrophy is extremely chronic and it may never completely recover. On the other hand the degenerative paralysis, of which the wrist-drop is the type, usually recovers perfectly, although its duration is prolonged in proportion to the amount of wasting that has occurred. When this is great, the paralysis continues for six or eight, or even twelve months. Voluntary power may return before faradaic irritability. If the wasting is moderate, and faradaic irritability is not entirely lost, recovery may occur in three or four months. Simple weakness without loss of faradaic irritability usually passes away in the course of six or eight weeks. Wrist-drop readily recurs if the patient is again exposed to the influence of lead, and a very slight exposure is sufficient to bring it back. A painter had wrist-drop, and on his recovery became a college porter, spending his time in a small "porter's box." Some time afterward a relapse occurred, and the only cause that could be ascertained was that the box had been repainted.* Occasionally, a severe relapse is permanent.

Sensory Symptoms.—There is usually little or no pain in the affected limbs, even at the time of the onset of the palsy. Rarely some sharp, darting pains occur before the palsy develops, more often in the legs than in the arms. Sensation on the limbs is also, as a rule, normal. Occasionally there is a little impairment of tactile sensibility, but when this occurs it is irregular and does not correspond to the distribution of the nerves; in one case there were irregular areas of anæsthesia on the front and back of the hand and wrist, and on the tips of the fingers.†

Laryngeal palsy is a very rare effect of lead poisoning. One or both vocal cords may be paralyzed, and they may undergo atrophy (Morell Mackenzie). Three cases have also been described by Seifert.‡ In one the muscle affected was the arytenoideus, in another all the intrinsic muscles of one side, in a third the posterior crico-arytenoids, which were found pale and atrophied after death. Horses that work in lead mines are said to get paralysis of the laryngeal muscles.

Local Spasm.—Cramps in the legs are not uncommon in the early stage of the affection, but more definite local spasm is very rare. I have once known flexor spasm in the forearms to precede the onset of wrist-drop. Another patient, with lead poisoning and colic, but no paralysis, suffered from a brief but well-marked attack of tetany, for which no other cause than the lead poisoning could be discovered; the paroxysms were confined to the arms, and recurred several times daily for about a week.

Tremor is less common in chronic poisoning by lead than in that by some other metals, but it is occasionally present in the hands in cases of long duration. It may be fine tremor, like that of old age; occasionally it resembles closely paralysis agitans; more frequently it is increased by movement, more than that of paralysis agitans, and is wider in range and more irregular in its distribution. I have known it to affect chiefly the flexors of the elbow and wrist and the supinator longus—the muscles which escape in paralysis. The lips and tongue may also be involved, and if there is no palsy the case then closely resembles one of mercurial poisoning. An occasional but rare symptom is difficulty of micturition, depending, it is said, on spasm of the sphincter.§

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* It has been suggested by Putman (*Boston Med. and Surg. Journ.*, Aug., 1887) that slight weakness in the legs, with excessive knee jerk, and also other symptoms of impaired nutrition of the nervous system, may be due to slight chronic lead poisoning. He has found minute traces of lead in the urine of many patients thus affected. It is at present doubtful whether the detection of a trace of lead in the urine justifies us in ascribing any symptoms to lead poisoning, when there is no line on the gums. Of course, the condition of the gums must be taken into consideration in estimating the significance of the absence of a lead line (see remarks in "Diagnosis").

† Oliver, *Brit. Med. Journ.*, *1885*, vol. ii, p. 732.

‡ *Berl. kl. Wochensch.*, *1884*, p. 535.

§ Lejeune, *Thèse de Paris*, *1881*.

Local Saturnism.—Under this name, Manouvriez* described supposed local effects of lead on the parts to which it was applied. Such a local influence, except as regards the cutaneous nerves, is very difficult to understand, and the facts that have been supposed to demonstrate it have received very little confirmation. Manouvriez collected thirty cases in which local symptoms coincided with the local application of lead to the skin. In some there was a lead line, in others there was not. In most cases there was loss of motor power; nine employed their feet to stamp on lead and had weakness of the muscles of the legs. He ascribed the fact that wrist-drop occurs first in the right hand (or in the left in left-handed persons) to the chief use of the hand affected, and described two right-handed men, who manipulated lead with their left hand, in whom this hand suffered first. Among a large number of sufferers it would not be difficult to find some examples of such coincidence, even if there were no causal relation between the phenomena. Nevertheless, some analogous facts have been recently published by others. Monnereau † observed in himself distinct loss of sensibility at the place where lead had been rubbed in. Capelle relates ‡ the case of a smith with colic and wrist-drop, who had worked with his left hand in lead, and there was extensive anæsthesia in the left arm and none in the right. Again, a man, whose boots were strongly impregnated with white lead, had anæsthesia of both feet, and weakness of some muscles of the legs. These facts suggest that lead may cause some local anæsthesia, but it is probable that the coincidence of muscular weakness was accidental.§

Cerebral Disturbance; Encephalopathia saturnina (Tanquerel). Symptoms of disturbance of the function of the brain occasionally occur in severe lead poisoning, and sometimes in cases of moderate severity. They vary much in character, and may be acute, subacute or chronic. The disturbance is usually general, but in rare cases hemiplegic symptoms have been observed. The loss of motor power is slight, but that of sensation is considerable; there may be partial or complete hemianæsthesia, involving general and special sensation. These symptoms are usually transient, and are apparently due to functional disturbance, such as occurs in hysteria. Such hemiplegia must not be confounded with that which results from cerebral hemorrhage, also common in the subjects of prolonged lead poisoning.

Much more frequent is general cerebral disturbance, manifested by convulsions, delirium and coma. Its onset is often acute, but may be preceded by certain slighter symptoms, giddiness, noises in the ears, tremor, restlessness and insomnia. The convulsions may occur at the onset or during the course of the delirium ; they are epileptiform in character, consisting of tonic and clonic spasm. They are usually general, but occasionally one side is affected before, or more than, the other. Very rarely they may be hystero-epileptic (Bramwell) or cataleptic (Bartens). There may be much muscular agitation, and general hyperæsthesia has been observed (Gabbett). The acute delirium often sets in suddenly, and is usually active, with considerable excitement, and sometimes attended with hallucinations of sight and hearing. When it is accompanied by muscular tremor, the condition may closely resemble delirium tremens. Somnolence and coma usually succeed delirium and convulsions. The coma is not often absolute; the patient can be roused for a few moments, but quickly relapses into unconsciousness. This acute cerebral disturbance is often accompanied by optic neuritis, and is frequently fatal, especially when there are repeated convulsions or deep coma. Slight fever (100°–101°) sometimes attends these cerebral symptoms. Death may occur in the coma, from respiratory failure, the heart continuing to act after the respiration has stopped.

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* *Gaz. des Hôp.*, *1874*, p. 290.

† Monnereau, *Thèse de Paris*, *1883*.

‡ Capelle, *Thèse de Paris*, *1883*.

§ Frémont (*La France Méd.*, *1882*, p. 892) has recorded a case of a man who one day worked with his left hand only in contact with lead, and did not wash it for three hours. Next morning there was tingling in the hand and weakness of the extensor of the fingers. The possibility of a slight injury to the musculo-spiral nerve renders this case of little significance.

Chronic general cerebral disturbance also occurs. It may succeed the acute stage, or may commence gradually. I have seen several cases in which convulsions, similar, in character and course, to those of ordinary epilepsy, occurred in the subjects of lead poisoning, without other symptoms, and continued for years after the toxic influence had ceased.

More frequent, however, is chronic mental disturbance, often necessitating confinement. The most common form is melancholia with delusions. Sometimes there are failure of mental power and muscular weakness, resembling those of general paralysis of the insane, but coming on more rapidly. True general paralysis of the insane, with exalted delusions, typical course and post-mortem appearances, has occurred in the subjects of lead poisoning and may have been due to it (Monakow, Ullrich).

The eye symptoms that occur are of considerable importance and deserve special mention. Inequality of the pupils is occasionally observed. Vision may be affected without any ophthalmoscopic changes; there is complete bilateral loss of sight, resembling uræmic amaurosis, but occurring when there is no renal affection. When unilateral, or greater on one side, it may be associated with hemianæsthesia and affection of the other special senses on that side. The incomplete amblyopia is attended with concentric limitation of the field and loss of color vision. These symptoms have been chiefly described by French observers. Annular scotomata have been observed (Landolt). I have once met with transient green vision.

In cases of acute lead poisoning, especially with cerebral symptoms, optic neuritis is frequent. The swelling of the papilla is considerable in degree, and accompanied by hemorrhages ("Medical Ophthalmoscopy," plate vii, fig. 6). In less severe cases I have occasionally seen slight chronic optic neuritis. The inflammation, both slight and severe, may pass off under treatment, without impairment of sight, but severe neuritis may cause "consecutive atrophy." Atrophy occurs, also, without preceding neuritis, or with only transient congestion of the discs at the onset (Horner, Hutchinson).

It is usually double, but one eye may be affected before, and more than, the other. The ultimate condition is usually a grayish atrophy, often with white lines along the vessels. It may slowly progress to complete loss of sight.

The acute and chronic symptoms of lead poisoning are often variously combined, and thus different cases may vary in their aspect. Moreover, the symptoms present in chronic cases differ much. Colic, although the most frequent, is not invariable; palsy may come on without any preceding colic, and tremor or pains sometimes exist for a long time without paralysis.

Pathological Anatomy.—Lead may be found in various organs by chemical analysis, but its presence only causes visible signs where it has been exposed to the influence of sulphur in a form that can enter into combination with the lead. The intestine may be dotted with minute black specks, as Fagge has pointed out. The chief lesions in lead poisoning have been found in cases of local paralysis and wasting.

The wasted muscles are small and pale, sometimes even yellowish in tint, and are brittle. In cases of short duration, many of the muscular fibres are narrower than normal, but the striæ are preserved, although a tendency to longitudinal or transverse fissuring is sometimes seen. The nuclei of the sheath are increased in number. When the wasting has lasted for a considerable time, the fibres present still greater narrowing, colloid or vitreous degeneration, and there is often granular or fatty degeneration, although in some cases this is absent. The nuclei are increased in number, and there is also an increase of the connective tissue, in which fat may be found. The process of destruction goes on, until ultimately many of the sarcolemma sheaths become empty, and in the end almost all trace of muscular tissue may disappear, connective tissue, nuclei and masses of fat taking its place. The walls of the vessels often become thickened.

In the nerves, morbid changes are constantly found. They are always intense in the intramuscular twigs, and in the branches from which these twigs proceed. They are usually considerable in the larger trunks, but commonly become slighter the further from the periphery the nerve is examined. Sometimes a few degenerated fibres can be traced throughout the nerves. The affection of the nerve seems to begin by a change in the medullary sheath, which becomes narrower, cloudy and granular, the axis cylinder being intact and the outer sheath and nuclei presenting little alteration. With this, however, is combined a more considerable change resembling the "Wallerian" degeneration that is secondary to a lesion of a nerve (see p. 60). The myelin undergoes segmentation, the axis cylinder also breaks up, the nuclei and protoplasm of the sheath become increased, and the products of this degeneration accumulate in masses of myelin and granule corpuscles. The slighter change (which Gombault,* who first described it, termed "peri-axial neuritis") is sometimes segmental, affecting short tracts of the nerve, that are separated by normal portions, or the upper part of the degenerated region may present only the change in the medullary sheath, while in the lower part there are the more extensive alterations. These alterations are always most intense in the radial nerve and its branches; but it is only in extreme cases that all the fibres of the nerve are involved; generally, normal fibres are mingled with those that are degenerated, and become more numerous the higher up the nerve is examined. In some cases of long duration, appearances have been seen suggestive of a process of regeneration; these are very narrow but otherwise perfect nerve fibres, or an axis cylinder ends suddenly, and is continued by two smaller ones (Westphal, Gombault).

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* *Arch. de Phys.*, vol. v, p. 592.

In most cases the degeneration ceases long before the anterior roots are reached; and these are normal. In a few cases, however, marked changes have been found in the anterior roots, similar to those in the peripheral nerves. Normal fibres were mingled with those that were affected. In one case of unilateral palsy, only the roots on the side corresponding to the paralysis were affected. The posterior roots have always been found unchanged.

In the spinal cord, in most cases, no morbid appearances have been found even when wrist-drop has been of long duration. In cases with advanced kidney disease the walls of the vessel have been thickened, and sometimes there has been a general increase in the connective tissue throughout the cord. In a few cases, however, distinct alterations have been seen in the cord, especially in cases with the slower form of muscular atrophy. In one case the consistence of the cervical enlargement was reduced. Atrophy of the ganglion cells of the anterior cornua has been several times noted,* slight in some instances, in others considerable, especially in the inner anterior group of cells. This was especially conspicuous in a case described by Monakow, in which there was slow wasting of the deltoid, thenar, and hypothenar muscles, in addition to the common wrist-drop. Spots of softening in the anterior cornua and intermediate gray matter were found in one case by Oeller, in addition to wasting of some of the nerve cells.

The brain has presented no marked changes even in cases in which the cerebral symptoms have been well marked. Slight traces of inflammation have been found in the membranes in a few instances, and in the case in which there were the characteristic symptoms of general paralysis of the insane, there was evidence of considerable inflammation both of the pia mater and of the external surface of the dura mater. In the optic papilla the usual signs of inflammation have been found, and in Oeller's case there was also "hyaline degeneration" of the walls of the vessels, extending back into the trunk of the optic nerve.

Pathology.—The changes that have been found in lead poisoning clearly point to a primary influence on the peripheral nerves, and the knowledge we now possess of the occurrence of a primary degenerative neuritis as an effect of many toxic agents renders the pathology of lead poisoning less mysterious than it was a few years ago. The wrist-drop is the palsy that we can refer most surely to this mechanism. The peculiar limitation of the paralysis to the muscles supplied by a single nerve has always suggested its neuritic origin, although the escape of the supinator longus constituted a difficulty in accepting this explanation, and the significance of the limitation is lessened by the fact, on which Remak has insisted, that palsy of a precisely similar distribution may result from a primary affection of the spinal cord. But it is in the cases of simple wrist-drop that the integrity of the cord has most frequently been demonstrated; the escape of the supinator longus is explained by the tendency of the degeneration to affect the peripheral part of the nerve (and is, moreover, not invariable), while the correspondence of saturnine wrist-drop with that which is due to alcoholic neuritis completes a very strong chain of evidence regarding its nature. It is probable that the same conclusion holds good regarding the palsy of the muscles of the leg that correspond to the extensors in the arm and are supplied by the peroneal nerve. The neuritis appears to be essentially of the degenerative variety, and it must be ascribed to a primary influence of the toxic agent on the nerve elements. It appears, moreover, that the motor fibres suffer far more than the sensory fibres, and probably they suffer alone in cases of slight or moderate severity. This feature is more conspicuous than in the case of alcoholic neuritis. The localization of the influence, the special tendency of the radial nerve to suffer, is at present as unexplained as are other analogous facts regarding the "elective" influence of other poisons. It is probably to be ascribed at least as much to a proclivity of the nerve to suffer as to any characteristic of the poison, since this nerve is first affected by many toxic agents. Other nerves suffer also in severe cases, although less readily than the radial.

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* By Vulpian, Monckton, Zunker, Monakow, Carriew, Oeller, and others. Pigmentation of the cells has often been seen, but its significance is doubtful. I have seen it in one young subject.

It is doubtful whether lead has any primary action on the muscles. The changes in their nutrition, and in their reactions to electricity, are those that are met with after primary lesions of nerves, and are sufficiently explained by the invariable neuritis. Its assumption is not needed to explain the symptoms.

But the action of lead is certainly not confined to the peripheral nerves. In severe cases the motor cells of the cord and the anterior roots may also undergo degeneration. Probably most poisons that act first and chiefly on the peripheral parts of the motor nerve fibres—on the periphery of the lower segment of the motor path—have also a tendency to influence the cells from which the fibres spring, and of which they are really part, *i. e.* they have an influence on the whole of the lower segment. Many recorded facts suggest, moreover, that the observed difference in the character of the muscular affection corresponds to a difference in the seat, as well as in the character of the degeneration; that the acute "atrophic paralysis," with the rapid wasting succeeding loss of power and attended by the degenerative reaction, is due to the acute degeneration of the peripheral nerve fibres, and that the slower wasting, with slow failure of power and irritability, is due to changes in the nerve cells of the cord, of more chronic course.*

The cerebral symptoms of lead poisoning seem to be independent of any visible change. Although they have been ascribed to some vascular disturbance, it seems more probable that they are due to the direct action of the poison on the nerve elements. We have conclusive evidence of an action on the nerve elements themselves in the nature of the peripheral neuritis. At the same time, acute disturbance, even when it begins in the nerve elements, is apt to involve also the interstitial tissue, and even the vessels, and may assume the character of general inflammation in tissues disposed to this process. The optic neuritis may be such a direct effect of the poison, but it is possible that it is excited by some slight irritative process descending from the brain.

DIAGNOSIS.—The recognition of lead poisoning depends, first, on the character of the symptoms of nerve disturbance; secondly, on the existence of other indications of the presence of lead in the system; thirdly, on the discovery of the fact that lead is entering the system; fourthly, on the fact that lead is leaving the system by the urine. The diagnosis can only reach a high degree of probability when two or more of these indications are combined. It is especially important that too much weight should not be placed on the character of the symptoms alone; many errors in diagnosis are due to this.

When the symptoms are such as to suggest that lead may be their cause, the next step in diagnosis is to ascertain if there is any other evidence of the presence of this poison in the system. The variations in the lead line have been already mentioned. If no line is conspicuous, and the gums are, in general, good, a search should be made for any place where the gum is not closely attached to the tooth, and the projections between the teeth must be carefully examined, since the line may be found only at these places. A fragment of distinct line is quite conclusive, and the absence of any line does not exclude lead poisoning if the gums are everywhere in perfect contact with the teeth. On the other hand, if the gums are separated from the teeth in many places, and there is tartar on the teeth, the absence of a line, according to present facts, makes it in the highest degree improbable that the symptoms are due to lead. The only condition in which there is a line like that of lead is silver poisoning, but this is sufficiently distinguished by the tint of the skin.

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* Whether lead has also an action on the upper segment of the motor path (see p. 130) so as to cause weakness without wasting and with an increase in myotatic irritability, we do not know. The facts collected by Putnam (p. 1263, note), suggest that it may be so, but no distinct sclerosis of the pyramidal tracts has yet been observed, even in cases of long duration. It is, however, probable, from analogy, that the extremities of these fibres would be the first to suffer, and the fibres themselves might undergo as little change as do those of the upper parts of the peripheral nerves. It is evident that the excess of myotatic irritability could only be revealed where permitted by the integrity of the lower segment. (Compare remarks on lateral sclerosis in "Progressive Muscular Atrophy," p. 365.)

The presence of a lead-line cannot be taken as absolute proof that the symptoms are due to lead. The line may last long after the poison has ceased to be active. Iodide of potassium removes lead from its organic combination in the tissues, but has no action on the deposit of sulphide of lead in the gums. Indeed, a solution of iodide of potassium is absolutely incapable of dissolving sulphide of lead, even if the two are boiled together. The lead-line very slowly disappears in the course of years, but I have known it to present little change in aspect two years after the entrance of lead into the system had been stopped, although the patient had been taking iodide of potassium during most of that time. Hence, the presence of a lead-line does not necessarily show that lead is active in the system.

Other effects of lead poisoning, colic, gout, anæmia, seldom do more than corroborate the diagnosis, although in the absence of a lead-line owing to the intact state of the gums, frequent attacks of colic are of some significance, and even the fact that gout has developed, without its usual causes, may be allowed some weight. In these cases, however, the diagnosis depends on the discovery of a source of lead poisoning, or of the fact of the excretion of lead. If there is no obvious source of poisoning, the water and its receptacles should be examined. Most cases of lead poisoning that are not due to occupation are due to the contamination of water, although the other occasional sources should always be borne in mind. The examination of the urine is the last resource of the investigator; it should be analyzed after iodide of potassium has been taken for a week. It is doubtful whether the presence of a very faint trace of lead is of significance. On the other hand, if iodide of potassium has been taken for several weeks, the absence of lead probably does not exclude lead poisoning.*

It is on the above points that the differential diagnosis from maladies that resemble those produced by lead chiefly turns. The wrist-drop has been confounded with paralysis of the musculo-spiral nerve, but this is always unilateral and usually comes on suddenly. In alcoholic neuritis the palsy may closely resemble that from lead, but the diagnosis is not usually difficult; the other signs of lead poisoning are absent; pains are generally obtrusive; the legs suffer earlier, and there is almost always a clear history of alcoholic excess. Wrist-drop may be produced, also, by arsenical poisoning, and apparently also by silver; the distinction will be described in the account of the effects of these metals. The greatest difficulty is presented by some cases of central disease. I have twice known progressive muscular atrophy (ultimately extensive) to commence by a subacute palsy of the extensors exactly like that produced by lead. If the gums are perfect, an examination of the drinking water or urine may be indispensable for the diagnosis. The cases of general muscular atrophy of saturnine origin scarcely ever present any difficulty, because they usually supervene on lead poisoning that has been intense and characteristic.

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* See note on next page. On the danger of mistaking bismuth for lead in the urine, see Putnam, *Bost. Med. and Surg. Journ., 1883*, Oct. 14th, p. 315.

Most errors in diagnosis occur in cases in which the nervous symptoms are anomalous and slight, and are not such as to suggest their cause. In the case of a lady with saturnine neuralgia, the cause of the symptoms might never have been discovered (for the perfect gums told no tale) had not her husband suffered from wrist-drop. A gentleman had some obscure cerebral symptoms with inequality of pupil; a fragment of lead-line opposite one tooth suggested lead poisoning, and it was found that his water was stored in a leaden cistern. It is not possible to avoid error in such cases except by the habit of remembering this as a possible cause in all obscure cases. The severe cerebral disturbance is practically confined to workers in lead, and usually results from a dose so large as to cause characteristic symptoms.

PROGNOSIS.—It may be said, generally, that the more acute the onset of paralysis the better is the prospect of complete recovery. The acute cerebral symptoms are alone attended with immediate danger to life, and the prognosis is worse when there are convulsions, and especially when there is coma, than in simple delirium. Convulsions are of graver significance when they succeed delirium than when they occur first. If death does not take place, the patient usually recovers completely. The prognosis in the chronic cerebral symptoms is unfavorable to recovery when these take the form of pronounced mental derangement or of epilepsy. Of the paralytic affections, those with the wasting and the degenerative reaction in the muscles, of which the wrist-drop is the type, almost always ultimately get well, but their course is very slow, and the chief prognostic indications have been mentioned in the account of the course of the disease. In relapses, if these are due to a considerable dose of lead, the prognosis is less favorable, although two or three successive attacks may be recovered from. In all cases the ultimate prognosis must be influenced by the extent to which the patient can be withdrawn from the deleterious influence.

TREATMENT.—The first step is to arrest the ingress of lead into the system, and the second is to promote the elimination of the lead that has been already taken in. The chief agent for securing the latter is iodide of potassium, given in doses of three or five grains. Although iodide of lead is an insoluble salt, it has been abundantly demonstrated that the administration of iodide of potassium increases the excretion of lead by the urine, apparently in some complex combination (Pouchet). The amount of lead in the urine increases for a few weeks and then gradually subsides.* The quantity of lead excreted is much less than might be expected, but this may be due to the fact that the liver, not the kidney, is the chief channel by which it passes away. When there are acute symptoms, iodide should not be given at once, since the toxic effects may be increased by the sudden liberation of lead that has been fixed in the tissues and rapidly passes into the blood. Such a result has been noted by many observers. By saline aperients (especially the sulphates of soda and magnesia with some free sulphuric acid) the alimentary canal should be cleared of any lead it may contain, or which may be excreted from its mucous membrane. If colic exists, it may be necessary to give first a full dose of opium, to relax the spasm, and aperients, inoperative before, may be at once effective. The bowels sometimes act spontaneously after a few hypodermic injections of morphia. Subcutaneous injections of atropine have been recommended for the same purpose. In two or three days the administration of the iodide may be commenced. Sulphur baths have also been extensively employed to promote the elimination of lead, but their utility is doubtful. Even in the copious diaphoresis produced by pilocarpine, no lead passes off by the skin. If there is anæmia, iodide of iron may be advantageously combined with the iodide of potassium.

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* Thus, in one case in which no lead could be found in the urine before the treatment, five grains of iodide were given four times daily, and on the first day five milligrammes of lead were found in the urine, on the fourth twelve, on the fourteenth twenty-two, and then the quantity gradually lessened, and after two weeks more only traces were found. (Swete, *Brit. Med. Journ., 1882,* Nov. 25th, p. 1034.)

For the local paralysis, strychine has been given internally. Its mode of action suggests that it may do good, although its actual effect is difficult to appraise. There is no evidence that its local injection has special advantages. The most important agent in the treatment of the paralysis is electricity. The nerves have lost irritability, but the muscles still respond to the voltaic current, slowly interrupted, which certainly keeps up their irritability, and must therefore influence the nutrition of the muscles while the nerves are recovering. Thus the amount of muscular change is lessened, and the ultimate recovery is rendered more perfect. In a severe case, if the muscles are left alone for a few months, or are merely treated with faradaism, there may be only the feeblest response to the voltaic current but (as I have seen) this increases notably after two or three applications, and the paralysis, before stationary, may in a fortnight be distinctly less and the improvement may steadily go on to complete recovery. In such a case the value of electricity scarcely admits of question. Only the voltaic current, to which the muscles respond, is of service. Faradaism should only be used if it excites contraction, as, for instance, in the primarily atrophic form. But in this it is difficult to get evidence of the value of the application. Massage of the limbs is also of service.

ARSENICAL POISONING.

Arsenical poisoning differs from lead poisoning in being seldom the result of occupation. It has generally been due, in some way, to the use of pigments containing arsenic, and has been produced through the agency of paints, wall papers, book covers, etc. The poison differs from lead in being more readily diffused through the air, and taken in by the lungs. Some interesting facts regarding these accidental causes have been collected by the Medical Society of London.* Extensively as arsenic is used in medicine, toxic effects (beyond a little conjunctivitis and gastric irritation) are almost unknown when it is administered in ordinary doses. One case, indeed, is on record in which severe symptoms were produced by the medicinal administration of arsenious acid,[*] and another in which they resulted from Fowler's solution, but this was given in increasing doses up to ʒss three times a day.[†] There is, however, no doubt that those who are taking arsenic often suffer from herpes zoster, especially on the trunk. This was first pointed out by Hutchinson, and I have seen at least a dozen instances of it.

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* *Proc. Med. Soc. Lon., 1880.*

The nervous symptoms produced by arsenic resemble very closely those that are caused by alcohol.[‡] They are of two classes: first, a palsy of the muscles of the limbs, especially of the extensors of the hands and feet; and secondly, a "pseudo-tabes"—ataxy, with defective sensibility, especially in the muscles. These symptoms occur as a result of chronic poisoning by small doses, or as an after-effect of acute poisoning, and they may also be a sequel to acute symptoms produced by the cumulative influence of small doses. Thus in one case of attempted suicide by arsenious acid, the acute symptoms passed away and a little time after their cessation the nerve symptoms commenced.[§] In one case the interval after the cessation of the acute symptoms was a fortnight, in another three weeks,[||] in another four weeks.[¶] In one of Dana's cases [‡] the ataxy commenced about the sixth day from the acute poisoning.

The paralysis in the arms has the same distribution as that produced by lead poisoning, but sensory symptoms are much more pronounced. There are usually severe darting pains in the arms and legs, and diminution of tactile sensibility, especially on the back of the forearm and front of the leg. It may be accompanied by some increased sensitiveness to pain.[**] Atrophy of the muscles occurs very rapidly. The electrical irritability presents the same degenerative changes as in other toxic palsies, but in very severe cases the voltaic irritability may quickly fall below the normal; the knee jerk is usually lost. Sometimes other muscles are involved, those of the upper arm and thigh may lose power, but not to the same degree as those of the extremity.

Arsenical ataxy seems to be less frequent than the muscular paralysis. It was present in one of the cases described by Seeligmüller, and two well-marked instances are described by Dana.[‡] It resembles closely the incoördination of tabes, but develops in a much more acute manner, and is accompanied by considerable muscular weakness. Severe pains attend the onset, and there are also subjective sensations, formication, tingling, etc. The sensibility of the muscles is lessened or lost, and so, as a rule, is the knee jerk;

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[*] Da Costa, *Phil. Med. Times*, *1881*, pp. 381 and 614.

[†] Dana, *Brain*, vol. ix, p. 456.

[‡] See p. 1288 and p. 110.

[§] Gubler, "Cours. de Thérap.," 1880, p. 13.

[||] Gerhardt, *Virchow's Jahresb*, *1882*, i, 396.

[¶] Seeligmüller, *Deut. med. Wochenschr.*, *1881*, p. 1.

[**] Mills, *Boston Med. and Surg. Journ.*, March 15th, 1883.

in Seeligmüller's case it was not lost. Cutaneous sensibility may be diminished in places, or may be preserved, and the sensitiveness to pain may be greatly increased. In one of Dana's cases there was optic neuritis.

This condition of the nerves has not hitherto been examined after death, but the close analogy to other forms of toxic paralysis leaves no doubt that the symptoms are due to a peripheral neuritis. The probability of this was urged by Jaeschke in 1882.

In some cases there have been other indications of the chronic effects of the poison—cachexia, falling off of the nails, loss of hair, and sometimes skin eruptions, bullous or erythematous.

The distinction from lead poisoning rests on the common onset after acute symptoms, on the more pronounced disturbance of sensibility, and on the absence of other indications of the effect of lead on the system. The absence of a history of intemperance is the most important distinction from alcoholic palsy, which that produced by arsenic closely resembles. The detection of a cause of arsenical poisoning is, of course, of great importance ; it is not usually difficult if the observer is led to suspect it from the character of the symptoms. In a doubtful case the discovery of arsenic in the urine may decide the diagnosis.

The course of arsenical palsy is similar to that which is due to lead, and the treatment necessary is essentially the same. Iodide of potassium appears to have a similar influence in removing the arsenic from the system.*

SILVER POISONING; ARGYRIA.

Chronic silver poisoning is due almost exclusively to the medicinal use of the metal. It is less frequent now than when a long course of silver was the fashionable treatment for epilepsy and spinal-cord disease. Most cases are due to internal administration, but argyria has been known to result from application to the palate or tongue, continued during many years. Working in silver involves no liability. Of the cases that have come under my own notice, two were epileptics (in whom the fits continued in spite of the influence of the silver), a third had been treated with silver for a syphilitic gumma pressing on the spinal cord, and a fourth had taken a salt of silver in a "dinner pill." The discoloration of the skin that is the characteristic of 'argyria' depends on a deposit of silver beneath the Malpighian layer,† and commences when about an ounce of salt of silver has been taken, in any period of time. It is accompanied by a black line on the gums, close to the tooth, closely resembling the lead-line. This appears before the darkening of the skin, and is therefore important as a warning. Among other internal organs,‡ silver is deposited§ in the kidneys—in the Malpighian bodies, and around the tubules; albuminuria has been observed both in animals and in man. In these two points the effect of silver resembles that of lead. In animals, moreover, paralysis is produced. I have been unable to find any recorded instance of paralysis in man, but one remarkable case has come under my own observation, which shows that the effect of silver may be almost the same as that of lead. The case was that of a gentleman, aged forty-four, who had been ordered, twelve years before, a "dinner pill," without any caution or other direction than that one pill was to be taken three times a day. He did not follow the directions literally, but took sometimes one, sometimes three, a day, for a few days at a time. He estimated that he had taken about six pills a month during the twelve years. After he had taken the pills for about eleven years a dusky tint of the face attracted notice, but its nature was not suspected until the tint had become well marked, when it was found that the pills contained silver (the exact form could not be ascertained.) Two months before I saw him the extensors of the right hand became weak; shortly afterward those of the left. When he came under observation the tint of the skin was very characteristic; his gums presented a well-marked black line, which resembled that of lead in being present where there was tartar, and absent where the gum was in perfect apposition to the teeth. In both arms there was paralysis of the long extensor of the fingers and of the extensors of the phalanges of the thumb, not of the extensor of the metacarpal bone of the thumb. On the right side there was also paralysis of the radial extensors of the wrist. The affected muscles were wasted, and their irritability to faradaism was lost; that to voltaism was preserved and increased. There was no affection of the legs. The urine contained some albumen and casts, and the patient had had several attacks of gout. During three months in which he remained under treatment (electricity, iodide of potassium, etc.) there was slight improvement in the arms, but no considerable change. He soon afterward showed symptoms of cancer of the liver, of which he subsequently died, but no post-mortem examination was obtained.

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* A full bibliography is given by Dana, loc. cit.

† See Neumann, *Wien. Med. Zeitung*, *1878*, No. 10.

‡ The distribution and precise seat in the internal organs was carefully investigated in one case by Weichselbaum, *Allg. Wien. Med. Zeit.*, *1878*.

§ It is said, as an organic compound (Krysinski, *Gaz. lekarska*, *1886*, and *Virchow's Jahresb.*, ib. I, 372).

MERCURIAL POISONING: HYDRARGYRIA.

Chronic poisoning by mercury causes symptoms of which some are due to an action of the poison on the nervous system. These symptoms rarely follow the medicinal use of mercury, but occur chiefly in those who work in the metal. Mercury will volatilize at all ordinary temperatures, and it is probable that it enters the system mainly through the lungs, but it may sometimes pass in through the skin when certain forms of mercury are much handled, and want of cleanliness may increase the amount of mercury that gets into the system, by causing contamination of the food. Sufferers are met with especially among those who work in quicksilver mines, at silvering mirrors, and making barometers and thermometers, and also occasionally among bronzers, furriers and hat makers—mercurial salts being used for preparing the skins. The weakly suffer more readily than the strong, and the individual differences in susceptibility are very great. The symptoms sometimes develop after the exposure to the cause has ceased.

The nervous symptoms may exist alone, but sometimes they are preceded by others outside the nervous system that resemble those of acute mercurial poisoning. These are stomatitis and ulceration of the mucous membrane of the mouth, with extreme fetor, necrosis of bone, gastric and intestinal catarrh. The remarkable removal of lime from the bones and its deposit in the kidneys, discovered as a consequence of acute mercurial poisoning by Prévost and Fruchtiger, does not seem to attend the chronic form, in which the kidneys do not suffer.

A peculiar tremor, known among workmen as "the trembles," and medically as "mercurial tremor," is the most common and characteristic symptom. It is at first occasional, occurring only when the patient is excited, and it is always increased by emotion. It usually begins in the face and tongue and then invades the arms, and afterward the legs. At first the tremor occurs only on movement, but ultimately it may become constant. In the former case the condition of the patient resembles that of one suffering from disseminated sclerosis; except that the tremor is less wide and less irregular than in characteristic cases of the latter disease. When constant the tremor resembles that of paralysis agitans. During sleep, the tremor usually ceases, but in extreme cases may only lessen. It interferes much with articulation, rendering the speech stammering and hesitating. When considerable it may render the movement of the arms so unsteady that the patient cannot feed himself, and his gait becomes affected. At first the limbs are strong; but after a time muscular power is impaired, sometimes more in one limb than in another, but it rarely progresses to complete loss. Reflex action and power over the sphincters are always unimpaired, and electric irritability of the muscles is normal throughout.

Psychical symptoms are also common, and may precede the tremor. They have been very carefully studied by Kussmaul. Irritability and a difficulty in giving attention to a subject, are often the first symptom, and may be accompanied by considerable mental distress and sleeplessness. Hallucinations sometimes occur, and there may even be outbreaks of maniacal excitement, but the insanity rarely corresponds to any distinct variety. This condition has been termed "mercurial erythism." It is sometimes accompanied by headache and palpitation of the heart.

Sensory symptoms are present in many cases; pains, especially in the region of the fifth nerve and in the joints; formication in the limbs, and even local loss of sensibility to pain; paroxysms of distressing sensations in the thorax, resembling those of asthma. The sensory disturbance always augments the mental instability. In rare cases more grave cerebral symptoms occur—considerable hemiplegic weakness, aphasia and deafness.

Even when the tremor is extreme, no alterations have been discovered in either brain or cord, even with the aid of the microscope. The character of the symptoms makes it probable that they are due to disturbance of the brain rather than of the spinal cord.

The diagnosis is rarely difficult. From paralysis agitans the tremor is distinguished by its immediate increase on movement, and by the absence of the rigidity of the limbs and fixation of the features. In disseminated sclerosis there is never the peculiar tremulous stammering that is met with in mercurial poisoning, and the movements are wider in range and wilder in their irregularity. The symptoms often resemble those of general paralysis of the insane more than any other disease, but such preponderant tremor is rarely met with in general paralysis, and the inequality of pupil, optimism and indications of spinal degeneration, present in the latter, are absent in mercurial poisoning. The presence of stomatitis should always direct attention to the probable nature of the disease, and when this is once suspected, the nature of the patient's occupation is usually decisive. The greatest diagnostic difficulty is presented by some cases of lead poisoning in which tremor is the chief symptom. These are distinguished by the presence of the lead-line in the gums, and if there is a suspicion that the patient is suffering from both poisons, the question can readily be set at rest by an investigation into the materials with which he works. If these are free from mercury, the symptoms are certainly due to lead only.

The prognosis is, in most cases, favorable as to the ultimate issue, but all cases are prolonged, and in some the tremor never passes away. For recovery, it is essential for the patient to cease to work in mercury. No precautions avail to prevent sufficient absorption to maintain symptoms that have once developed. Nutritious food, fresh air and iron, are important aids to recovery. To promote the elimination of the mercury, iodide of potassium may be given, but in small doses—two or three grains very gradually increased. There seems no doubt that it does facilitate elimination, and the sudden administration of large doses has been followed by a considerable increase in the symptoms; apparently mercury released from the tissues passes into the blood and to the nervous system. Neither sweating nor sulphur baths seem to cause an elimination from the skin, although it is said that the latter have been found in some way to increase the amount excreted by the kidneys. The stomatitis is relieved best by chlorate of potash.

ALCOHOLISM.

The nervous system is especially sensitive to the influence of alcohol, and suffers in many cases, more than other tissues, from the effects of habitual alcoholic excess. The acute poisoning by alcohol to which, *par excellence*, the term "intoxication" is applied, is chiefly of physiological and toxicological interest. Only the morbid states that are distinct from definite intoxication come into the province of medicine. These are exceedingly numerous; many of the organic and functional diseases already described may be produced, or predisposed to, by intemperance; some other derangements are due chiefly to this cause, and are seldom or never produced by any other. To these the term "alcoholism" is sometimes applied. These disorders and symptoms may be acute, subacute or chronic. The acute disturbance generally takes the form of an attack of delirium, called, from the tremor that accompanies it, "delirium tremens."* The chronic derangements are exceedingly varied.

All forms of alcoholism have certain common causes. The tendency to drink may run in families in its pure form, or it may be distinctly related to a family disposition to suffer from definite diseases of the nervous system, especially from insanity. It is probably facilitated by peculiarities of nervous constitution, in consequence of which the sensations of intoxication are more pleasurable in some persons than in others. Parental intemperance, moreover, often involves facilities for habitual excess. Occupation is notoriously influential, partly on account of the facilities for drinking afforded to those who are engaged in the distribution or sale of alcoholic liquors, partly on account of the social temptations to drink involved in other callings. The temporary relief to physical and mental depression afforded by alcohol is another potent cause of intemperance.

ACUTE ALCOHOLISM, ALCOHOLIC DELIRIUM, DELIRIUM TREMENS.

Alcoholic delirium occurs under two forms; as a primary affection, and in association with some local inflammation or injury. The latter may be termed "associated delirium;" it has a double causation, the influence of alcohol, and the influence of the local disease. The latter may be sufficient, by itself, to cause some delirium, although far less in degree, and without the peculiar characteristics of the alcoholic form; or it may be wholly insufficient to derange the mind. The simple primary delirium varies much in the intensity of its symptoms; it has been divided into two classes according to the presence or absence of considerable fever, but the difference depends only on the degree of severity, and there is no real justification for the division.

CAUSES.—Delirium tremens is almost confined to drunkards, in the popular sense of the word .In this respect it differs from the "associated delirium," which is not uncommon among those who have habitually taken an excessive quantity of alcohol to keep up their energy against the depressing influence of debility or the prostration of some chronic disease. Thus a man who had never been known to be intoxicated and was believed to be temperate, died with symptoms of acute delirium tremens, but it was found that his lungs were extensively diseased, and that he had been keeping up his working power by large doses of alcohol during the acute development of the pulmonary disease.

Delirium tremens is far more common in men than in women, the proportion being about six to one.† There are no facts to show whether there is any sexual tendency to the disease, such as certainly exists in the case of another effect of alcohol, multiple neuritis, to which women possess a special liability. As far as the figures given below suggest any conclusion, it is that drinkers in the two sexes are equally prone to suffer from delirium tremens. The disease is most frequent in the middle period of life; and in both sexes the maximum mortality is at the same age; in each one-third of the total number of deaths occur between thirty-five and forty-five years of age, and about five-sixths of the deaths occur during the thirty years—twenty-five to fifty-five. It rapidly lessens in later life, no doubt because the worst drinkers acquire the habit early, and die off from some effect of intemperance, but cases of delirium tremens occur even up to advanced senility.* Moreover, females bear a proportion to males that steadily increases as life goes on, no doubt because drinking habits are acquired relatively later, under the influence of individual causes rather than of the influences of occupation or social life.† It is doubtful whether an hereditary tendency to insanity influences the occurrence of this disease, although such a tendency unquestionably modifies its course, and Magnian has observed that this influence sometimes determines the occurrence of relapses in which the physical disturbance is slight in proportion to the amount of mental derangement. The disease is less frequent in beer drinkers than in those who drink wine, and most frequent in spirit drinkers, but it is probable that the amount of alcohol taken has much greater influence than its form.

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* By Thomas Sutton ("On Delirium Tremens," a tract published in 1813).

† During the twenty-five years ending 1872, there died from this cause in England and Wales 10,448 males and 1398 females. It is probable that this disease is one on which the Registrar-General's returns are fairly accurate, since this cause of death is unlikely to

In most cases the malady follows a severe "bout" of drinking, often without any interval. Occasionally, there has been a cessation of drinking for a few days before the onset of the delirium. These cases have led to the idea that the discontinuance of the stimulant has been the cause of the attack, but it is more probable, as Anstie and others have suggested, that a sudden distaste for liquor was really the first effect of the commencing attack. The onset occasionally follows some slight injury or trifling illness, which apparently disturbs the nervous system, and serves to excite the disturbance that is ready to break out. Such cases present a gradation to the "associated delirium tremens," in which there is some real cause of general indisposition, a severe accident or a local inflammation, such as pneumonia. The pyrexia resulting might or might not be attended with delirium in the

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be given unless there is a history of intemperance. The figures give the proportion of deaths as 7½ males to 1 female. But the disease is not only more common, it is also more often severe, and therefore it is more often fatal, in males. Hence the disproportion between the sexes is probably rather less in cases than in deaths. Of 54 consecutive cases in University College Hospital, 8 were in females, giving the proportion of 6 to 1, which is probably very near the truth.

* If, moreover, from the number of deaths, the number per 100,000 living is ascertained, the cases in advanced life are seen to be proportionally more numerous than the actual number of deaths suggest.

† The proportion of deaths of males to each female is about 12½ in the ten years 15–25, and steadily falls, the proportion in each successive decade being 10–1, 7¾–1, 7⅓–1, 6–1, 4¼–1, and only 3–1, in those over 75.

healthy; in the alcoholic the delirium is severe, and presents the same characteristics as in the pure form of delirium tremens.

SYMPTOMS.—The symptoms of delirium tremens usually develop gradually but rapidly, attaining considerable intensity in the course of two or three days. Disturbed sleep, loss of appetite, and restless irritability, are commonly the first indications of the impending disorder. Distressing or horrible dreams of peculiar vividness attend sleep, and the patient cannot shake off their effect when he wakes. He is depressed, uneasy, restless during the day, often annoyed with floating specks, or flashes of light before his eyes, and the next night brings the same distressing dreams. As soon as he closes his eyes, unpleasant scenes present themselves before him, and he tosses about all night, half awake, or perhaps is unable to obtain any real sleep. Next day, from time to time, more definite hallucinations of sight or hearing occur; the patient can shake them off and knows that they are unreal, but they return again and again. When night comes they take possession of him, prevent all sleep, and in the morning they are no longer to be shaken off. This insomnia, first partial and then absolute, with the distressing hallucinations which disturb the imperfect sleep that is obtained, characterize the early period of the disease. The transition to more considerable disturbance is usually attended with unnatural loquacity, and by indications of suspicion and uneasiness. The period of definite delirium begins with the persistence of the hallucinations during the day. They are chiefly visual and almost always unpleasant. At first the objects that the patient actually sees are transformed into other things than they really are, often into living creatures in active motion; the pattern of the wall paper becomes beetles, spiders, snakes; and then these appear spontaneously, and are crawling about the bedclothes; the sufferer tries to brush them away or to escape from them. At first he can be recalled from his aberration for a few moments, and will answer questions correctly, but soon relapses into delirium. Faces appear before him, grinning at him, or he fancies that persons are standing by his bedside, upbraiding or abusing him. The attendants are thought to be the subjects of his delusions, and he may strike at them under the impression that they are attempting to injure him. Often, the modified incidents of his daily life seem to occur before his eyes. He usually talks incessantly, but wanders incoherently from one subject to another in the course of the same sentence. Auditory hallucinations may become grafted on those of vision; the patient thinks he hears reproaches or insults from the fancied bystanders. Less commonly, an unpleasant smell or a disagreeable taste annoys him. The delusions often excite intense emotions of horror or dread. A characteristic of the delirium is the versatility of the false ideas; they change continually. Gradually, he ceases to be able to shake them off, even for a moment, and no longer recognizes his friends or doctor.

The characteristic tremor is usually present from the first. It occurs only on movement, and is irregular, and considerable in range; it is most conspicuous in the arms, the face, and tongue, but is to be seen also in the legs when these are put into voluntary movement, especially if the patient attempts to stand. It is the more conspicuous, because the patient is usually in constant movement, picking at the bedclothes, searching for imaginary objects, attempting to get out of bed. This extreme restlessness is seldom absent. Often, in addition to the tremor on movement there are spontaneous, slight, partial muscular twitchings, and in severe cases these may amount to considerable shock-like contractions, and may occur in the muscles of the trunk as well as in the limbs.

The countenance of the patient is often flushed, and the conjunctiva injected, but occasionally the face is pale, and sometimes initial flushing is replaced by pallor as the stage of depression comes on. The pulse is frequent and soft; the frequency is proportioned to the severity of the attack and to its duration. At first the pulse is full and large, but as the disease goes on, it usually becomes smaller as it gets more frequent. In severe attacks the frequency may be 140 or 150, sometimes even more, and it is then always feeble. The patient usually perspires freely. The temperature is raised in all except the slightest cases. When the attack is of moderate severity the elevation is slight, and does not exceed two or three degrees, but in severe attacks it may attain 103°, 104°, or even 105°. Occasionally hyperpyrexia is attained, 108° or 109°, a symptom of fatal significance. The tongue is thickly coated, and there is usually complete anorexia; the patient may or may not be thirsty; thirst is often a consequence of the copious perspiration. The urine is scanty if there is much sweating, and is usually deep colored, of a high specific gravity, and very often contains a small quantity of albumen apart from organic kidney disease. There is occasionally slight blurring of the edges of the optic disc, with, according to Uhthoff, some opacity of the retina.*

This state usually continues for two, three or four days without interruption. The patient gets no sleep, or only dozes for an hour or so at a time, and wakes up unrefreshed. The pulse becomes softer and more frequent, and the muscular strength becomes lessened by the constant exertion. In favorable cases the patient at last falls into a sound sleep, which continues for eight, ten, or twelve hours, and he wakes up free from all mental disturbance, or with only a trifling amount of delirium, which another sleep entirely removes. The tremor lessens, but often continues in slight degree, sometimes for some days or weeks. The pulse falls in frequency and improves in strength after the cessation of the delirium, and in the course of a few days the slight tremor and muscular weakness are the only relics of the attack through which the patient has passed. In other cases, however, sleep, when it does come, is briefer, and is not followed by the same signs of improvement; the delirium continues, but is less active than before, and the signs of general prostration are greater. In such cases there is considerable danger to life. A second or third period of sleep may bring improvement, and slow convalescence may ensue at the end of six or seven days, but in other cases the prostration increases, the pulse becomes still feebler and more frequent, and a comatose state comes on, in which the patient dies. This unfavorable condition is sometimes reached without any sleep having occurred, in spite, it may be, of strong narcotics. Convulsions or hyperpyrexia may attend the increased prostration of the later stage, and are generally a fatal omen. Convulsions may also occur at the onset, and are not then of much significance; at the later period of an attack, by the disturbance of the nervous system which they reveal, as well as by their effect on the patient, they add greatly to his peril. The convulsions resemble those of ordinary epilepsy.

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* Uhthoff, quoted by Moeli, *Charité Annalen*, ix, p. 524.

Complications.—The most important of these are other diseases due to alcohol, or local acute inflammations, especially pneumonia. Cirrhosis and fatty degeneration of the liver, and disease of the kidney, are especially serious complications. Uræmic coma occasionally comes on in the course of delirium tremens. Pneumonia may be a subordinate complication of the disease, developing after the onset of the toxic disorder, when the patient is prostrate. Its development is often insidious, and may be unaccompanied by pain or cough, while the effect on the breathing may be masked by the restlessness of the patient. Hence, examination of the chest is of extreme importance in every case. On the other hand, pneumonia may be the primary affection, and the toxic delirium is then to be regarded as a complication of the pneumonia and an instance of "associated delirium tremens." A rare complication is meningeal hemorrhage, which has been met with in a few cases.

Course and Sequelæ.—Delirium tremens lasts from three to seven days, and commonly terminates in recovery. Death occurs chiefly in those who have had several previous attacks, or whose tissues are gravely damaged, either by the influence of long-continued intemperance or by senile degeneration. Its common causes are exhaustion and gradual failure of the heart's action. Sometimes death occurs suddenly, from unexpected syncope. Death may also result from associated acute diseases, especially from pneumonia. The mortality has been variously stated; v. Franque's estimate of 18 per cent. is probably near the truth.*

Most patients who recover do so perfectly. Sometimes, however, the mental state continues abnormal. The sleep that brings physical quietude may only partially restore the mental balance. The distressing hallucinations cease, but some more stable delusion remains, and may last for weeks or months. If the patient has had a previous attack with this sequel, the duration of the mental change is longer, and the state may even be permanent. In that event there is usually progressive failure of memory and of mental power, so that the patient passes into a condition of chronic insanity or of chronic dementia. This persistence of mental change is met with chiefly in those who inherit a tendency to insanity, and the stronger this is, the greater is the tendency to this sequel, and the more readily does it occur.

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* This estimate was calculated from 2117 cases (v. Franque, "Ueber Delirium Tremens," Munich, 1859).

Pathology.—In a case of pure alcoholic delirium, the pathological changes are slight, and consist only in signs of congestion of the cortex of the brain and sometimes of other parts, especially of the medulla and spinal cord. There is often some opacity of the arachnoid, but it is probable that this is a chronic change, the result of long previous intemperance. Signs of actual acute meningitis are rare, but have been occasionally found in both the pia-arachnoid and the dura mater. Congestion of the bases of the lungs is common, and sometimes there are signs of congestion in the kidneys, but the alterations met with outside the nervous system are chiefly the chronic changes that result from intemperance.

Alcohol may be found by chemical analysis in various organs of the body up to the fifth day after its ingestion; by the seventh day it has disappeared. But the presence of alcohol may be demonstrated; in like manner, in persons who have been drinking, but have not suffered from delirium tremens. We cannot, therefore, regard it as affording a full explanation of the disease. This evidently depends on an acute disturbance of the nerve centres, probably involving all parts of them, but especially the cerebral cortex—a disturbance of function that runs a definite course and has a tendency to subside. This statement, however, still leaves the actual pathology of delirium tremens a mystery.

Diagnosis.—The disease is easily recognized. The preponderance of visual hallucinations, and the associated tremor, distinguish it from acute mania on the one hand and from symptomatic delirium on the other, while the absence of headache and of paralysis is a distinction from ordinary acute meningitis. Cases of general paralysis of the insane, in which muscular tremor is conspicuous, have been mistaken for delirium tremens; but if there is much mental alteration, its character is altogether different in the two diseases; the exaggerated delusions of general paralysis, and the mental complacency that commonly attends them, are very different from the anxiety and horror of the alcoholic. When depression occurs in general paralysis, the mental state has generally developed gradually, in a manner very unlike the acute onset of delirium tremens.

Prognosis.—The prognosis is good in first attacks, free from complications, and in those whose tissues are not seriously damaged. The pulse and temperature afford the most important prognostic indications. The danger is great in proportion to the frequency, softness, and small size of the pulse, and, as Anstie has shown, the sphygmographic tracing presents very clearly the serious characters, although the instrument is not actually needed to detect them. As a rule, the occurrence of sound sleep for six or eight hours greatly improves the prognosis, but if an exceedingly unfavorable pulse presents no improvement after sleep, the danger is great, the patient is very likely to die in a few hours. A temperature above 103° is always of serious import, and the danger is greater in proportion to the degree of pyrexia; a rise to 105° and over is seldom survived. Moreover, a sudden considerable rise in the later stages of the disease is also of very grave significance, even although the actual height attained is not extreme. Advanced years add greatly to the gravity of delirium tremens, especially when there is considerable pyrexia; in a man of sixty a rise to 103° is as serious as would be a temperature of 105° in a young person. Any complication also lessens the chance of recovery, the most serious being pneumonia and kidney disease. The prognosis is also grave when delirium tremens is associated with traumatic lesion, both because the tendency to depression and collapse is increased by the shock of the original injury or operation, and, also, because wounds run a very unfavorable course in this condition.

Treatment.—The mental state of the sufferer from delirium tremens necessitates very careful management. It is essential that all undue excitement should be avoided, and only the necessary attendants should be in the room. It is well to have the light dim, unless this distinctly increases the patient's suspicion and uneasiness. When there is much disposition to act according to the false ideas, male attendants are necessary, and physical restraint is often indispensable, or the patient may injure himself or others. He may even attempt suicide by jumping from a window or swallowing some accessible poison, sometimes deliberately, sometimes under the influence of a false idea. Hence windows should be securely fastened, and no lotions or poisonous liquid should be within the patient's reach. In many cases the patient can be kept in bed by judicious management and persevering persuasion on the part of the attendants. In other cases, however, force is needed. The mode in which it should be applied is a matter on which authorities differ. If there is great violence, continued exertion on the part of the attendant is necessary to keep the patient in bed; two attendants are needed to cope with a strong, delirious man, and the constant struggles are exhausting to the patient. The "strait-waistcoat" offers, in such cases, an effectual mode of restraint; the interference with the movement of the chest produced by it has been urged as an objection to its use, although this drawback has certainly been exaggerated. Leather gauntlets on the wrists and ankles may be secured to the sides and foot of the bed, and restrain the patient effectually without any interference with respiration. Bandages are sometimes employed for this purpose, but may cut into the skin if the struggles are violent. Another effectual means of restraint is a sheet across the trunk and arms, firmly secured at the sides of the bed. In most cases of delirium tremens this answers as well as the strait-waistcoat, and has the advantage of being always available, but it may slightly hamper the respiratory movements.

Food must be given in liquid form; beef tea, milk, and beaten-up eggs should form its chief part. The anorexia often renders it difficult to give nourishment, but the difficulty can generally be overcome by a little perseverance. It is well, in this as in other acute diseases, to commence the process of digestion of food before it is taken. Benger's Liq. Pancreaticus and Liq. Pepticus offer the readiest means for doing this, the former being used for milk, the latter for beef tea. Half a drachm of either may be added, and the food kept warm (not hot) for twenty minutes or half an hour, and then given. The process of digestion thus commenced goes on within the body.[*] If there is vomiting, peptonized enemata, well digested, must be given. The patient should not be without food for more than three hours; a due supply of nourishment is by far the most important element in treatment. Whether alcoholic stimulants should be given must depend on the state of the pulse. They are better withheld unless the pulse calls for them in language that is not only unequivocal, but is rather more decided than would be held a sufficient indication in an acute disease of other nature. Some stimulation may be obtained by ammonia and ether. Coffee has been recommended for the same purpose, but it is not likely to increase the influence of sedatives. It is probable that more help would be given by the administration of a grain of cocain twice a day. In all cases an aperient should be given at the outset, enough to open the bowels freely, but not so strong as to cause prostrating purgation.

It was formerly customary to administer sedatives freely from the first in the endeavor to procure the sleep that usually ends the acute disturbance. Bromides are commonly inadequate to compel sleep, although often useful as tranquillizing agents; opium or morphia and chloral are the hypnotics usually given. In most severe cases efforts to produce sleep fail until near the time when the terminal sleep naturally comes. The insusceptibility to sedatives in the early stage is extraordinary; dose after dose may be given until a poisonous quantity is in the system, and still the patient may be sleepless, with increasing and perhaps alarming exhaustion. If the hypnotics are boldly pushed, they at last may act, and the patient may die from their influence. Anstie believed that when opiates fail to influence the brain and to induce sleep, they sometimes paralyze the heart. He mentioned a case in which, almost immediately after the administration of a second large dose of opium, the patient became ghastly pale, the pulse fluttered and then stopped, and in a few minutes the patient was dead.[†] Without doubt many cases have ended fatally in consequence of the idea that it is absolutely necessary to procure sleep at all risks. It is only in a very slight case that it is right to try to force sleep in the early stage. In all severe attacks it is wiser to postpone the attempt until the duration of the disease has increased the prospect of success, that is, until the third or fourth day of the attack. If a narcotic dose is given earlier (such as a quarter of a grain of morphia beneath the skin, or sixty grains of chloral by the mouth) it should not be repeated until sufficient time has elapsed to permit its action to pass off. It is better, however, in the early stage to be content with moderate doses, merely sufficient to favor tranquillity. A scruple or half a drachm of bromide, with fifteen or twenty grains of chloral, may be given every six or eight hours, and then, after about forty-eight hours, a stronger hypnotic may be tried. Chloral generally acts better and is attended by less danger than opium or morphia. Of the latter, not more than a third of a grain should be injected, and it is wiser to give first a smaller dose, a quarter or a sixth. The practice of repeating moderate doses at short intervals has been recommended, but it is better to wait for six hours and then to give a second full dose. Opium has been thought by some to be safer than chloral if there is weakness of the heart, but the fact is doubtful. In mild cases, half a grain of the extract of Indian hemp sometimes promotes sleep.

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[*] This is a much better method than that commonly employed, of carrying the process of artificial digestion further, and then stopping it by heat.

[†] Art. "Alcoholism," "Reynolds' Syst. of Med.," vol. ii, 2d ed., p. 169.

The treatment by large doses of digitalis, introduced by Jones, of Jersey, has been universally relinquished. The good thereby effected is doubtful, and certainly altogether disproportionate to the danger of the treatment. The use of pilocarpine to increase the sweating and thus help to eliminate the alcohol has been recommended,* but the agent is unneeded in slight cases, and in all others the use of a drug that depresses should be carefully avoided. The same remark applies also to the employment of tartar emetic for the same purpose, which was at one time in vogue. A drug that has been strongly recommended, and is at least not open to the same objection of lowering the strength of the patient, is capsicum. Three or four doses given every three hours are said frequently to produce calm, refreshing sleep. Ten or even twenty grains of cayenne pepper, in the form of an infusion, or half-drachm doses of the tincture, may be given.†

If alcohol is necessary during the acute stage of the disease, it should be discontinued as soon as possible, and altogether withheld during convalescence. An attack of delirium tremens is sometimes a turning-point in a drunkard's life, and every means should be taken to enforce upon him the lesson of the illness, a lesson which he is proverbially prone to forget with returning health.

ACUTE ALCOHOLIC INSANITY.—Delirium tremens, although the patient is for the time insane, is not classed as a form of insanity. Acute attacks of mental derangement sometimes result from this cause, and are scarcely to be distinguished, except in their origin, from the similar condition that comes on from other causes. Indeed, in almost all cases of the kind there is an hereditary tendency to mental derangement, and the alcoholic excess is merely the exciting cause. *Acute mania* may thus come on, distinguished from delirium tremens by more persistent delusions and by the absence of the tremor and of the restlessness of mind and body. Some of these cases run a shorter course than acute mania commonly does; sleep has a more pronounced ameliorating influence, and some tremor may be present. Thus there are forms that seem to be intermediate between simple acute mania and delirium tremens. Some mental change is apt to be left behind for a time, but this, as we have seen, is not uncommon after delirium tremens in those who inherit a tendency to insanity. *Acute melancholia* also sometimes occurs from alcoholic excess, but does not present any special peculiarities. A peculiar form of recurring insanity, associated with, and excited by, a drinking tendency, has been termed *oinomania*. There is sometimes permanent moral deficiency, and there are periods in which the subject loses all moral sense, falls into various excesses, and may wander about and act in an absurd and often indecent manner. In some cases, during the intervals between the outbreaks, the patient exhibits no moral or intellectual defect.

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* Isham, *Am. Med. News*, *1885*, p. 312.

† Cayenne is an old negro remedy in Jamaica for many acute ailments. (See Kinnear, *Lancet*, *1882*, i, p. 291, and an anonymous letter, *ib.*, p. 390.) It was at first given in a glass of spirit, a vehicle open to objection in delirium tremens. In many of the cases in which its influence has been supposed to be most conspicuous by contrast with other drugs, it was given after these had been tried in vain, and at the period when a spontaneous subsidence of the disease might be looked for.

CHRONIC ALCOHOLISM.

Various morbid states of the nervous system are slowly induced by habitual alcoholic excess. These differ from the acute disturbance in that they may be induced by habitual excess which never attains the degree necessary to cause actual drunkenness. The most common symptom is muscular tremor, seen chiefly in the hands, lips and tongue. It is inconspicuous in the legs, although it may often be found there if looked for. It is a fine irregular tremor, and occurs only when the muscles are put into action by the will. It results from all kinds of excess, and may be as conspicuous in those who drink only beer as in spirit drinkers. It is generally greatest in the morning, and is less after some alcohol is taken. Sometimes there are also sudden starts of the limbs, especially during sleep. Insomnia is often troublesome; sleep, when it occurs, may be disturbed by distressing dreams, and visual hallucinations may distress the patient in the state between sleeping and waking, and may prevent sound sleep. To these symptoms some persistent mental changes are often added, irritability, restlessness, failure of memory. Other symptoms of disease outside the nervous system often coexist, congestion of the face and conjunctiva, fullness of the eyelids, a tendency to skin eruptions on the face, a furred tongue, anorexia, and morning vomiting.

Besides these chronic disorders certain definite maladies may occur, subacute or chronic in character. The most important of these is multliple neuritis, a description of which is given on page 108, et seq. Alcoholism is by far the most common cause of this disorder,* and it is, strange to say, a disease to which women are certainly more prone than men. Instead of the mixed motor and sensory symptoms that usually result from this disorder—palsy of the extremities, loss of sensation, muscular tenderness and acute pains—it may have a tendency to affect the nerve fibres, according to their function, so that motor palsy sometimes occurs with but little sensory disturbance, or there may be incoördination closely resembling that of locomotor ataxy.†

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* It has been pointed out by Dr. Dreschfeld (in a valuable article in *Brain*, Jan., 1886, p. 434) that an accurate sketch of the symptoms of alcoholic neuritis was given by Dr. James Jackson, of Boston, U. S., in 1822.

† A peculiar kind of unsteadiness in walking is caused, as Charcot has shown, by the localization of the palsy in the legs. But in other cases there is true ataxy, probably the consequence of an affection of the sensory muscle nerves.

Chronic and subacute myelitis certainly sometimes results from alcoholism, as is shown by a case figured in Fig. 97. In this, as in most cases of the kind, the inflammation of the cord accompanied peripheral neuritis, by which its symptoms were masked to a considerable extent. Disease of the spinal cord was formerly thought to be the common cause of alcoholic palsy in the form that we now know to be the result of neuritis. The cases of "alcoholic paraplegia" described by Dr. Wilks* are certainly examples of neuritis. I have not met with any case in which there was satisfactory evidence of an isolated affection of the cord due to alcohol, but since chronic myelitis certainly results from this cause, in association with neuritis but anatomically separate, it is probable that it does occasionally occur alone. It would, seem, however, that the susceptibility of the peripheral nerves is greater than that of the cord.

In France, hemianæsthesia is said to be not uncommon in alcoholics, and some examples of it have been described by Magnan,† similar apparently to the unilateral loss that is met with in hysteria. But the condition is rarely met with in this country. Epilepsy is occasionally an effect of alcoholic excess, but the attacks seldom occur periodically, as they do in the ordinary form of the disease. Usually a series of attacks are excited by a bout of drinking, or even by a single intoxication, and the patient is free from them until he again gives way to excess. In such cases the fits are apt to accompany an attack of delirium tremens.

Neuralgic pains in the limbs are sometimes troublesome; these may sometimes be the result of an influence of alcohol on the nerves, such as, in greater degree, causes actual neuritis. But some of these nerve pains are produced through the agency of a gouty diathesis, to which alcoholic excess unquestionably contributes.

It is an undecided question whether alcohol causes amblyopia and optic nerve atrophy, such as result from the use of tobacco. In many cases of tobacco amblyopia there is a history of alcoholic excess, which has been thought to favor the occurrence of the affection, but its production by alcohol alone is not well established.

I have occasionally seen distinct slight optic neuritis in the subjects of chronic alcoholism, generally in association with headache, and slight mental changes. It is probable that in many of these cases there is slight chronic meningitis. Opacity and slight thickening of the arachnoid and dura mater are met with after death in some cases, chiefly marked over the convexity of the brain, and are probably a direct effect of the habitual excess. This, with some slight shrinking of the cortex, constitutes the most common postmortem change in cases of long duration. Fatty degeneration of the walls of the small arteries is probably more common in alcoholic subjects than in others. Minute foci of softening due to vascular disease, and called "encephalitis," have been met with in the cortex in rare cases, usually in association with considerable and long-standing mental changes.

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* "Lectures on Diseases of the Nervous System," 1878, p. 224.

† "On Alcoholism," Greenfield's Trans.

Chronic alcoholism may aid in the production of many forms of definite insanity, but the only variety that can be certainly ascribed to this cause, acting alone, is chronic dementia—failure of memory, commonly progressive for a time, accompanied by defective power of judgment, and often by want of cleanliness and other indications of defective moral sense. Mental weakness may be associated with defective articulation, and with recurring slight paralytic attacks, so as to constitute a group of symptoms bearing some resemblance to general paralysis of the insane, but differing in the non-progressive character of the disorder if alcohol is given up. Actual recovery is, however, rare, although the sufferers may live for many years. It is probable that, in these cases, there is chronic meningitis. The influence of alcohol in causing true general paralysis has been much discussed ; the disease very seldom results from this cause acting alone, although intemperance may aid other causes in producing it.

The diagnosis of the effects of chronic alcoholism from the maladies with which they may be confounded has been sufficiently considered in the description of those diseases. The most common question is the distinction of alcoholic tremor from other forms, especially from the very similar tremor that is sometimes congenital, or if not congenital, has existed from early life, and is often inherited. The history of the tremor sufficiently distinguishes the two affections.

The chief element in the treatment of chronic alcoholism is the change in the habits of the patient. Bitter tonics, such as bark, have been sometimes found of service, as in some measure satisfying the craving for drink. Tincture of capsicum has also been praised ; ten-minim doses may be given before meals or when the craving for drink comes on.* The special derangement will generally subside when the cause ceases to act. The special treatment that may be needed has been described in the account of these diseases. Strychnine and nux vomica are useful for the tremor. For the insomnia and nervous restlessness, Marcet has recommended oxide of zinc, in two-grain doses, two or three times a day.

HYSTERIA.

The term "hysteria" is applied to a morbid state of the nervous system which is far more common in women than in men. The primary derangement is in the higher cerebral centres, but the functions of the lower centres in the brain, of the spinal cord, and of the sympathetic system may be secondarily disordered. Hence the symptoms may be varied in their range, and in some cases the secondary manifestations of the disease may so preponderate as to obscure the primary disturbance. The name is derived from an erroneous idea that there is a special connection between the disease and disorders of the womb (ὑστέρα). It was even once thought the womb moved about to various parts of the body, and so caused the local symptoms. The opinion that the disease is primarily and chiefly one of the cerebral functions is now all but universally held, and the use of the word "hysteria" does not now imply the theory involved in its derivation.

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* See Ringer's "Therapeutics," 5th ed., 1876, p. 362.

It should, however, be remembered that there is a difference between the popular and the medical use of the word. To the public, the name is chiefly associated with the simulation of symptoms, or at least with the idea that the patient could prevent the symptoms if she liked. But in medicine it is now generally recognized that the malady is a real one, occasionally of great severity, and to a large extent beyond the direct influence of the patient's will. It should also be remarked that the word is applied more widely to women than to men; the same conditions are often termed "hysteria" in the former, and "hypochondriasis" in the latter.

ETIOLOGY.—Both race and civilization have probably more influence on the occurrence of hysteria than has been recognized or studied. It is almost unknown among barbarous races, and seems to be a product of the cerebral development that accompanies the process of civilization. In certain stages of civilization peculiar forms of the disease have attained great development, as in Europe during the Middle Ages. Among races that have attained, apparently, an equal degree of civilization, hysteria reaches a higher degree in some than in others, in the French, for instance, than in the English; no doubt in consequence of the underlying differences in nervous constitution that are recognized in the expression "national temperament."*

Hysteria has been estimated to be twenty times as frequent in women as in men (Briquet), but the varying use of the word already mentioned renders the facts difficult to ascertain. Every form does occur in the male sex, and certain varieties are not infrequent. The liability of females is probably determined by the character of their nervous system, and not by the possession of certain sexual organs. Some disposition to hysteria is inherent, if not in all women, at least in the vast majority. The affection is far more common in boys than in adult men; the differences in the nervous constitution of the two sexes are far less before than after puberty. Adult men who are the subjects of the disease often present mental characteristics resembling those of the female sex. The exact influence of age is also difficult to ascertain, because the special manifestations of the disease are the result of an underlying and antecedent morbid state, which develops so gradually that its commencement can seldom be determined. The frequency with which distinct symptoms commence in the several decades of life is shown in the following table, calculated from the figures given by Briquet and Landouzy:—

Age	—10	—20	—30	—40	—50	—60
Per cent	8	50	28	10	3	1

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* The following account of the disease is based chiefly on its manifestations in the English race. Some features, that are merely alluded to here, have attracted great attention

Thus, in one-half the cases the disease is first manifested in the second decade of life. Of these, a larger number begin between fifteen and twenty than between ten and fifteen. In nearly a third of the whole, the first manifestations occur between twenty and thirty, and a much smaller number in the first and fourth decades. The cases met with under ten generally commence after six, although symptoms of similar character have occasionally been met with at three, four, or five years of age (Barlow). These figures are true chiefly of females. Regarding the age at onset in males, we can only say that the commencement is before puberty in a larger proportion of males than of females.

Heredity unquestionably influences the occurrence of the disease. There may be direct inheritance, or a family history of allied neuroses. One of the most severe cases of juvenile hysteria I have seen was in a girl of nine, both of whose parents had insane relatives. In another case, a girl had peculiar hysteroid fits, and both her mother and grandmother had had similar fits at the same age. Briquet found a history of hysteria in the parents of hysterical patients twelve times as frequently as in the parents of those who were not hysterical, but such a proportion is probably much larger than would be found to obtain in this country. It must be remembered that a tendency to hysteria finds favorable conditions for development in the injudicious moral training that is received by the children of an hysterical mother.*

The causes that directly determine the development of hysteria in an individual may be either physical or mental influences. Both are often conjoined. Of the two, the mental and moral influences are the more potent and the more frequent. They may both increase the inherent predisposition and excite pronounced manifestations of the disease. The inherent predisposition, indeed, probably depends on the physiological preponderance of emotion in females, which is essential for the part they play in the continuance of the race, but which may be perverted either by congenital tendency or injudicious training. License and indulgence in childhood beget self-indulgence in later life. When the disease has once developed it is often greatly increased by injudicious management. The near relatives of the hysterical are often conspicuously deficient in judgment, and the little common sense they may possess is often rendered useless by their affection for the sufferers. In some cases the symptoms develop without more exciting cause than the trifling annoyances of home life, or the change that the nervous system undergoes at the time of puberty. More frequently, however, the decided manifestations of the disease are due to some distinct exciting cause. This is rarely intellectual exertion ; in most cases it is some emotional disturbance. It may be a sudden alarm, which is an especially frequent excitant of hysterical disturbance in children. It may be merely the

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abroad. These have been, for the most part, fully described in the lectures of Charcot, accessible to English readers in the translation of the New Sydenham Society.

* See on this subject Dr. Russell Reynolds' address to the Sanitary Congress, 1887.

depressing emotions from which no life is exempt, trifling in themselves, but potent because unresisted. It may be some sudden and deep emotion, an unhappy love affair, a fall from luxury to the need for unaccustomed labor. The effect of the real causes may be merely seen in depression or irritability, often with failing health, and the outbreak may be immediately excited by some trivial disturbance, adequate only to turn the scale too delicately poised. Defective general health, though by no means essential for the development of the disease, is yet a common antecedent. Hysteria may occur in women who are in good health, but more frequently there is obvious deterioration. The patients are anæmic, easily fatigued, and often with functional disturbance of various organs.

Of the physical conditions that may influence the development of hysteria, disorders of the generative organs have always attracted most attention. The share which they take has been variously estimated. It is certain that some morbid state of these organs is present in many cases, but the estimate that at least one-half of the sufferers from hysteria are free from such disease (Jolly) is probably near the truth. Further, such disease, in a considerable proportion of the cases in which it exists, is certainly not the cause of the hysteria. The morbid states of these organs that are met with are rarely grave; they are commonly trifling in themselves, but such as cause frequent or continuous suffering, and thus may depress the nervous system. The rectification of such disturbance has, in rare instances, been followed by a marked improvement in the symptoms, but such an effect is rare. Commonly, the symptoms of hysteria continue unmodified by the removal of the local disturbance, and the indirect influence of the treatment is often anything but beneficial. The influence of uterine disease or displacement which causes no obtrusive symptoms is more than doubtful.

The observations of Charcot have lately directed attention to the ovaries rather than to the uterus. Tenderness in the ovarian region is unquestionably frequent, but is not invariable, nor is it confined to the subjects of hysteria. It is usually a neuralgic affection, and not an actual local disease. It is very rare for there to be any adequate evidence even of congestion. When the generative organs are healthy, the menstrual period is attended, in most women, by some discomfort and depression, and it is natural that the symptoms of hysteria, if they exist, should be aggravated at these times. In such patients, moreover, menstruation is often irregular, in consequence of the defective general health, and its disturbing influence is thereby increased. Severe cases of hysteria with much ovarian pain have been cured by the removal of both ovaries; but too wide an influence may readily be drawn from the fact. The operation not only removes a source of irritation, but involves a profound moral influence and prolonged physical rest.

Sexual excess is an occasional cause of hysteria in men, and, as masturbation, still more frequently in boys, but its influence in women is probably not large. Continence has been supposed to be a cause of hysteria, but probably is so only when it suddenly succeeds habitual indulgence, and its influence is chiefly confined to the female sex.

Hysterical symptoms, especially convulsions, sometimes spread from one individual to another by sympathetic imitation, "moral contagion," as it has been termed. Many singular outbreaks of this character have been recorded. The sufferers, living together in a hospital, or workhouse, or school, have often been exposed to similar predisposing influences; and the materials in all are ready for explosion.

In a predisposed person the symptoms of hysteria may be excited or intensified by other diseases; nervous, general and local maladies often determine the direction of more enduring hysterical disturbance. The symptoms which result from this union of disorders may be most perplexing. In typhoid fever in young girls, hysterical tenderness of the skin and spine may be present, and anæsthesia, pharyngeal constriction, rapid breathing and even contracture may occur (Huchard). Tuberculosis is often attended with hysterical phenomena, which may simulate, or more often mask, the symptoms of tubercular meningitis. The pains of rheumatism may persist as hysterical neuralgia. The secondary stage of syphilis, especially among prostitutes, is often attended by symptoms of hysteria, sometimes intense. Fournier believes that they result from a specific action of the virus on the nervous system, but the depressing effect of the disease, coupled with the pyschical influence of the mode of life, is probably sufficient to account for the condition. Almost all forms of local inflammation may determine the occurrence and locality of hysterical symptoms. Thus, arthritis may set up an "hysterical joint," a laryngeal catarrh may excite persistent hysterical aphonia, a slight attack of bronchitis may lead to hysterical dypsnœa and rapid breathing. Even more potent is the influence of local injury; spinal tenderness may be excited by a blow or fall, and injuries to the limbs may be followed by pains, anæsthesia and contractures, beginning in the affected member, and often tasking all the skill of the diagnostician to separate the direct and indirect effects of the injury. The latter, however, often become more extensive in distribution, and are then more readily distinguished.

If hysterical symptoms may thus complicate general diseases, it is not surprising that they should still more frequently accompany various diseases of the nervous system, functional and organic. The effect of disease of one part is often to disturb the functions of other parts, and of this disturbance hysterical symptoms are a frequent result. As Weir Mitchell has well expressed it, "the symptoms of real disease are painted on an hysterical background." There is hardly a single disease of the nervous system by which such symptoms may not be produced. Cerebral tumors in young women often cause conspicuous hysterical phenomena in addition to their direct results. The subjects of infantile hemiplegia, when they reach puberty, may present a high degree of hysteria. In one case of old hemiplegia in which true epileptiform convulsions occurred in the stunted limbs, the patient suffered also from the most severe hysteroid convulsions I have ever witnessed, and also from aphonia, rapid breathing, and phantom tumor. Slight or old organic diseases of the spinal cord may determine hysterical spinal symptoms, and thus an hysterical paraplegia may be grafted on slight real weakness of the legs. So, too, with acute diseases, as tubercular meningitis. A real diphtheritic paralysis may pass into hysterical palsy and anæsthesia. I have known hysterical convulsions to attend the onset of embolic hemiplegia, proved to be such by post-mortem examination. Hysteria sometimes acccompanies chorea, and this disease may also be succeeded by hysteroid fits. Similar convulsions occur with great frequency after true epileptic fits during the ages at which hysteria is common (see p. 1093). The most enduring case of hysterical aphonia I have ever known was in an epileptic woman. Lastly, hysterical symptoms and convulsions may form part of the phenomena of hydrophobia, apparently as the direct effect of the poison on the nervous system.

Symptoms.—The manifestations of hysteria may be divided into two classes, the continuous and the paroxysmal. Of the former, the most important is the mental state, which, in the majority of hysterical patients, presents marked characteristics. These differ in their details according to the ever-varying peculiarities of individual character. Most prominent among them, and rarely absent in severe cases, is a defective power of will, imperfect self-control, inability to resist the impulses of inclination. With this is often associated irritability of temper, and an undue sensitiveness to annoyance, under which the trifling cares and vexations of life become grave troubles. Occasionally this deficiency of will power is inconspicuous. Some patients with originally well-balanced minds, under the depressing influence of ill health or care, may have their mental strength undermined, and the symptoms of hysteria may develop insidiously as disorders of subordinate functions. In other cases, some violent shock may shatter a strong will, and emotion may become dominant in a mind in which it was previously held in strict subordination. But these cases are far less common than those in which the will has been gradually allowed to fall into servitude. Self-consciousness dominates, more or less completely, the patient's thoughts and even actions, and finds its expression in manner, glance and tone. There may be obvious exaggeration in the description of sufferings or an implied consciousness of much more than is expressed. The sympathy that is excited is a source of gratification to a patient whose sufferings often secure a relief from other annoyances which to her are greater, and the attention she receives is a new stimulus to her self-consciousness. The motives become stronger to yield to, than to resist, morbid tendencies, which are thus unconsciously cultivated. This defective will power is sometimes associated with a low moral tone, and the cultivation of symptoms, which is at first unconscious and involuntary, may then become conscious and intentional. Those that are at first merely unresisted may be afterward welcomed, then invited, and at last actually induced or consciously stimulated. Every stage in this gradation of development may be met with by itself, and sometimes, from the first, the symptoms are assumed. It must not, however, be inferred that all mimicry of disease is intentional simulation. The nervous system is dominated by ideas, as well as by emotions; the definite conception of a symptom may lead to its occurrence; and when idea and emotion are conjoined, and a symptom is not only conceived, but either dreaded or desired, its occurrence is still more easy. The idea of a loss of power may render it impossible for the patient to will the movement; the conception of a muscular spasm may induce the contraction, and if a definite pain is thought of, before long it may be felt, without the symptom being in any case intentionally induced. Medical inquiries and examinations often suggest to patients the definite ideas of symptoms, and the physician's knowledge of the natural association of symptoms may thus lead to their consistent grouping in a mimetic malady, even when there is not, and still more when there is, deliberate simulation. The pathogenic influence of idea is seen in all varieties of hysteria, and sometimes in singular isolation in children, in forms which Russell Reynolds has designated "ideal paralysis." The idea is most definite, and therefore most effective, when such symptoms have been actually observed in another person. Hence the spread of hysterical convulsions and other symptoms by imitative contagion. I have known two children in a family to suffer, one from a cerebral tumor, the other from startlingly similar symptoms of pure mimetic origin, evoked by the witnessed sufferings of the first.

The excess of emotion in the subjects of hysteria commonly finds free expression. Laughter and tears come readily, and these manifestations of emotion may occur in paroxysms on the most trivial excitant, and attend or constitute the slighter form of hysterical "fits." Certain other common symptoms of the disease are also natural results of profound emotion, and are morbid because spontaneous or too readily produced. One of these is the well-known "globus hystericus," a feeling of something suddenly closing the throat and stopping the breath. It is often described as a ball rising from the stomach to the throat; in other cases it is a mere sense of constriction or swelling referred to the fauces or pharynx. An identical sensation may, from sudden alarm, occur in those who are not hysterical. It is a frequent precursor of an hysterical fit, but is also excessively common apart from any other paroxysmal symptom. Some have supposed that the sensation is the result of an actual spasmodic contraction of the œsophagus and pharynx, but there seems no sufficient evidence of this. The sensation in its most typical form appears to be identical with that which immediately precedes many epileptic fits, and I have even known the same sensation to precede unilateral convulsive seizures in a case of tumor of the pons Varolii. It is often accompanied by a sudden sense of suffocation, for which there is no reason in the interference with breathing, and it appears to be the expression of a disturbance in the respiratory portion of the vagus centre, which is normally specially sensitive to emotion.

The same relation to emotion may be traced in many other symptoms of hysteria, which will be described in detail; such, for instance, are the disturbance of the heart's action, the limpid urine that may result from fear, and the muscular tremor of alarm, while many of the phenomena of hysteroid convulsions are but the frenzied manifestations of horror or rage.

Sensory Symptoms.—In hysteria all sensations, general and special, may be felt with abnormal acuteness, and those that are in health unnoticed may give rise to distress. It is often difficult to learn how far there is an actual increase of sensation and how far the exaggeration is in the description of the feeling. That an actual increase sometimes exists is proved by rare cases in which there is an extraordinary acuteness of the special senses. Sounds may be heard which are inaudible to others, a print may be read in a light so dim that it is quite illegible to ordinary sight, and the sense of smell may attain an acuity comparable only to that which it possesses in animals, so that, it is said, persons may be distinguished by its means.

In these cases of increased sensitiveness painful sensations are readily produced and are often spontaneous, giving rise to the varied forms of hysterical tenderness and neuralgic pain, the locality of the two being often the same. The tenderness is frequently superficial, more pain being produced by a touch on the skin than by pressure on deeper structures; such superficial tenderness is common on the skin of the abdomen, thorax and scalp, and sometimes affects one-half of the body. There is often, in certain localities, deep-seated tenderness, and when considerable, the pain occasioned by pressure is peculiarly distressing, radiates to the chest or throat or head, and tends to cause the dyspnœa, sense of faintness, globus, and even in highly-developed forms of hysteria, convulsive attacks. Hence, these tender spots have been termed "hysterogenic," by Richer.

One of the most frequent seats for this tenderness is the ovarian region, and here it is commonly deep-seated. Its position is at the intersection of a line joining the anterior superior iliac spines with that which limits externally the hypogastric region, and Charcot believes that a tender body, which may, in a thin patient, be felt between the finger and the brim of the pelvis, is really the ovary, although in an opened body the ovary is usually found within the pelvis. It is doubtful whether the ovary is really felt, but it is in many cases so tender that its disturbance by pressure on the neighborhood probably causes pain, and the adjacent parts may also be the seat of neuralgic tenderness, for Weir Mitchell has observed extreme deep tenderness in this situation when the ovary could be felt, by vaginal examination, to be displaced downward out of the usual position. Either ovarian region may be tender, the left more frequently than the right, and if both are sensitive the tenderness is usually greater on the left side. Occasionally, as Todd pointed out, there is extreme superficial tenderness in a circumscribed spot over the ovary. The deeper sensitiveness may be accompanied by spontaneous pains. In estimating the significance of this symptom it should be remembered that tenderness in this region is not uncommon in women who are not hysterical.

Another frequent seat of tenderness is the spine, especially the upper, middle, or lower dorsal region. The tenderness may be superficial, but more frequently it is deep seated. Spontaneous pain in these situations is often complained of, sometimes severe and burning in character, sometimes a dull aching that is compared to toothache. It is usually increased by exertion. Occasionally the whole of the vertebral column is tender and painful, and the pain seems to shoot up from the coccyx to the occiput. Sacral pain may be complained of, but sacral tenderness is rare.

Deep-seated tenderness may be present in the left hypochondriac region, and may be greater there than in the ovarian region. Other occasional seats of tenderness, usually superficial, are the infra-mammary regions, and spots on the front of the abdomen and thorax, or on each side of the dorsal spine (Richer), which may be symmetrical on the two sides. Occasionally there is superficial tenderness (of the skin and sometimes of the muscles also) over the whole abdomen; this simulating the tenderness of peritoneal inflammation has received the absurd name of "false peritonitis." When there is general hyperæsthesia the whole surface is "hysterogenic;" a prick on the forearm, for instance, may cause sharp pain, darting to the throat and causing globus.

The spontaneous pain common in the left infra-mammary region is apparently identical in character with that which is so common in the same situation in anæmia. Still more frequent, and very distressing, are the pains which occur in the head. The pain is sometimes frontal, temporal or occipital, but much more frequently vertical, and because it is severe and occasionally described by patients as like a nail being driven in, it has received the name of the "clavus hystericus."

The hyperæsthesia of the special senses often occasions distress, which may be described as actual pain. Intolerance of light is very common, and is fostered by the dark blinds provided by sympathetic friends. Subjective sensations of various kinds may be complained of; noises in the ears, flashes of light or color, or, much less frequently, sensations of taste or smell. Pricking or tingling sensations in the limbs, or vague feelings of numbness, are very common, and are often unilateral, affecting, for instance, one-half of the tongue. Another sensation which is frequently described is that of cold water trickling down the spine. It is sometimes associated with a nervous shivering, analogous to that which is produced in healthy persons by a slight degree of cold combined with nervous excitement. Some joint is often the seat of spontaneous pain, attended, it may be, with slight swelling such as may also occur in other parts, apparently from vasomotor disturbance.

Lessened sensibility is very common, although often overlooked because the patient seldom complains of it, and she may, indeed, be unaware of its existence. Sometimes it occurs on the legs in association with motor weakness, but as an isolated symptom it usually affects part or the whole of one-half of the body, constituting "hysterical hemianæsthesia," and the unilateral loss may be complete or incomplete. The special senses are involved in a way that will be presently described. When complete, a touch cannot be felt; a needle may be run into the skin without causing any sensation; neither cold nor heat can be perceived, and the skin may even be burned without pain. The loss extends up to the middle line, and may involve the mucous membrane of the conjunctiva, nose, mouth, vagina, and also the deeper structures, muscles and bones. There are, however, certain anomalous features. Ovarian and other deep-seated tenderness continues on the affected side; reflex action is unchanged, the pupil still dilates when the skin is stimulated, and the fingers can still be used (*e. g.*, for needlework) without the guidance of the eye. When partial, sensation may be lost to either pain or touch, rarely to temperature only. Often the sensation of touch is lost, and of pain only lessened; a prick may not be perceived, but faradaism with a wire brush may be felt acutely. When partial in distribution, the arm suffers more than the leg, and the loss may be limited to the arm and cease abruptly at the shoulder or on the chest; the arm and leg may be affected, and not the trunk or face. The loss may reach the middle line in front, and stop far short of it behind. Loss of tactile sensibility may be more extensive than loss to pain. Occasionally only small areas are anæsthetic. When complete, the limbs may be pale and colder than those of the other side, and a prick may not bleed; but this is, on the whole, rare. Usually there is no difference in aspect or vascularity; and the mucous membranes are never paler. Normally, pin-pricks in many places do not bleed. Hemianæsthesia is more common on the left side, and usually there is considerable ovarian tenderness on the affected side. Exceptionally I have found no ovarian tenderness, or tenderness on the opposite side.

The loss may come on spontaneously, or may follow a hysteroid fit. It may be present one day and gone the next; in one such case a prick on the previously anæsthetic arm caused so much pain as to induce a hysteroid convulsion, and when this was over the hemianæsthesia had returned, in typical and complete form. An increase may follow the testing of sensibility, and it is possible that an examination sometimes induces the anæsthesia. The area affected may vary from day to day, and may increase at menstruation.

The anæsthesia may also change from one side to the other without apparent cause, or such a "transfer" may be induced; a phenomenon discovered by Charcot, and much studied in France. The agents that cause the transfer may be (1) such as stimulate the skin and dilate the vessels, *e. g.*, blisters, sinapisms or faradaism; (2) the application for half an hour of certain metals, especially gold, or of a large magnet or electro-magnet, which need not be actually in contact with the skin. Painting with collodion sometimes succeeds. The transfer is seldom lasting; after a few hours or a day the loss reverts to the original side. In the elaborate hysterics of France, sensitiveness to certain metals has been described (Burq, Dumontpallier, etc.), and it has even been asserted that patients may be cured by the internal administration of the metal to which they are externally sensitive! Mysterious and unknown forces have been invoked to explain the phenomena of "metallo-therapy," but they have received little confirmation elsewhere. Wood has been found as effective as gold, and so has a mental shock or the inhalation of nitrite of amyl.* Metallic idiosyncrasies have been practically unconfirmed, and the balance of probability is strongly in favor of the opinion that most agents act through the mind of the patient, a theory in harmony with what we know of the pathology of the disease, and with many facts in its history which show how much care is needed in drawing conclusions from the mysterious blending of psychical and nervous disturbance in the disease. If there is one lesson more clearly written in its history than another, it is that the more complex symptoms are best interpreted by the light of those that are more simple.

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* See a case recorded by Urbantschitsch, *Arch. f. Ohrenheilk.*, Bd. xvi, p. 171.

As part of the hemianæsthesia, and capable of transfer with it, there is a remarkable affection of the special senses. These may all be lessened on the affected side. The impairment of vision (carefully studied by Charcot and Landolt) is in the form of "crossed amblyopia" such as sometimes results from organic disease (pp. 463, 585). Acuity of vision and the field are both greatly reduced on the anæsthetic side; the color fields are reduced in the order of their normal extent, and they may be lost in the same order, violet first, then green, red, yellow and blue. The loss may be demonstrated by the "confusion test" (see p. 573) as well as by the method of naming colors. The effect of electrical stimulation on the retina is lessened, but the action of the pupil is normal. A similar but much slighter affection of vision may be found in the other eye.

Vision may, as I have seen, be sometimes impaired alone, almost always on one side. There may be absolute loss of sight, or only great amblyopia and reduction of the field to a small area around the fixing point. The iris still acts perfectly to light, and the fundus oculi is normal. The loss is usually sudden; sometimes it follows a hysteroid fit. It is usually transient, passing away in a few days or weeks. An instance of more permanent loss of sight of this character is mentioned on p. 590; in this, such unilateral amblyopia has now lasted for six years,[*] sometimes improving a little, and then relapsing, still without change in the action of the pupil or in the fundus. Very rarely hysterical patients have transient bilateral loss of sight.

Hearing is involved in the hemianæsthesia, but scarcely ever alone. The loss is usually greater for sounds conducted through the bone than for those conducted through the air.[†] The nerve also loses its normal sensitiveness to electrical stimulation.

Motor Symptoms.—Paralysis is very common, and may involve almost any part of the motor apparatus. The onset may be sudden or gradual; it may follow a convulsive seizure, or may be excited by emotion. Some transient palsy may follow each fit, paraplegia after one, hemiplegia after another. When the onset is sudden, the palsy is usually at first incomplete, and increases under the influence of the idea and fear of loss of power. It must never be forgotten that the palsy of an hysterical patient may not be wholly functional; real paralysis of organic origin may be increased by the mental state.

The most common form of hysterical paralysis is that of the larynx, causing the well-known hysterical aphonia. There is loss of voice, *i. e.*, of phonation, so that the patient always speaks in a whisper. Sometimes this s merely voluntary; in a state of general hyperæsthesia the sound of the voice distresses the patient, who habitually whispers, although perfectly able o phonate. More often there is an actual inability to utter vocal sounds. In the common form the laryngoscope shows that the vocal cords are far apart, and are not approximated during phonation, as in health; there is defective phonic adduction (see p. 693). The glottis can be closed efficiently n coughing, except in very rare and extreme cases. Sometimes patients can sing well, although speech is whispered, and they have been known to speak n a loud voice during sleep. A scream may often be obtained by strong faradaism applied either outside or inside the larynx. Hysterical aphonia may come on spontaneously, but is frequently excited by emotion; a circumstance which is not surprising when it is remembered that the larynx s the channel through which certain emotions are most readily manifested. It is also occasionally excited by laryngeal catarrh—the real loss of voice from cold persisting after the catarrh has ceased. The aphonia is not usually attended by any unpleasant sensation, but in one severe case the loss of voice was referred to a feeling "as if a bar of iron were laid across the chest." Speech often returns suddenly. The duration of the aphonia is extremely variable, and it is prone to relapse. One patient had been aphonic for ten years, with occasional intervals during which the slightest right would at once remove her voice.

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[*] At p. 590 the loss is said to have existed two years, but I have since ascertained that the condition is still essentially unchanged.

[†] Walton, *Brain*, Jan. 1883, p. 458.

Paralysis of the abductors of the vocal cords is an exceedingly rare consequence of hysteria. The symptoms are those described in the account of aryngeal paralysis. A few cases are on record, and I have seen one very striking instance, mentioned on p. 693. It is probable that this paralysis in hysteria has been sometimes mistaken for laryngeal spasm.

In rare cases of hysterical aphonia, the tongue shares the laryngeal inaction, and loss of articulation is added to that of phonation, so that even whispered speech is lost, and the patient can express herself only by signs. In one girl, any sudden emotion would induce this state; it was accompanied by a sensation "as if the tongue were being twisted up." Simple aphonia may deepen into such absolute speechlessness. Sometimes there is a form of respiratory stammering. In a girl with hysteroid fits, one or two deep inspirations always preceded every attempt to speak, and sometimes nterrupted a sentence or a word. During her fits she talked with perfect fluency. Rarely, actual stammering may be a part of severe hysterical disturbance.

Paralysis of the limbs is also common, and may assume the form of hemiplegia, of paraplegia, or of general loss of power. Certain forms of hysterical ataxy of movement may also be ranked in the same category. In his country paraplegia is certainly more common than hemiplegia. As a rule, the onset in all forms is sudden, and often follows some emotion. The oss of power is usually moderate in degree at first, and gradually increases. The power which remains is put in action irregularly. Resistance to passive movement, for instance, is not sustained, but varies, at one moment being slight, at another considerable. If an attempt is made to execute a give movement, opponents of the acting muscles may be felt to contract. If t patient, for instance, attempts to extend the knee, the flexors of the kn may contract so as to prevent any movement. When there is some pow of movement, this is accompanied by tremor, presently to be describe There is no muscular atrophy, or only very slight wasting from long disus Electric irritability, as a rule, remains perfectly normal. So constantly this true, that the nature of the rare cases in which a marked change in ele tric irritability has been found, is open to grave doubt. Loss of sensatio may or may not be conjoined with the loss of power.

Paraplegia is excited by emotion with especial frequency. Even in healt a sensation of weakness in the legs may be caused by sudden alarm, an this, in hysteria, may be followed by a progressive loss of power. It is con mon for the onset of persistent weakness to be preceded by occasion momentary "giving way of the legs," at once recovered from—a very cha acteristic feature.

In other cases, some unpleasant sensation or pain in the legs seems excite the palsy. The pain may be that of a real disease, or it also may of hysterical origin, and to its influence that of emotion is frequently adde Spinal pain is very common in these cases, and, being increased by standin may distinctly excite the paralysis.

There is rarely absolute loss of power; the legs can commonly be move about in bed, although slowly and jerkily, but on an attempt to stand, the give way at once, and the patient sinks to the ground. If the loss of pow is slighter in degree, the patient may be able to walk a little, but with slo short, shuffling steps, rarely, however, catching the toes against the groun Retention of urine is uncommon, and there is never incontinence of eith urine or fæces. Myotatic irritability may be perfectly normal; it is so i more than half the cases. In many, however, especially in those with pe sistent spinal tenderness, there is slight increase of this irritability; the kn jerk is excessive, and the patient is peculiarly sensitive to the tap on t patellar tendon or on the top of the depressed patella, which causes a refle start or jerk of the trunk and often a sharp pain in the back. On the oth hand, when there is no excess, the knee jerk may seem to be absent, but th is always due to the movement of the leg being prevented by involunta tonic contraction of the flexors of the knee, detected without difficulty by finger placed on the hamstring tendons. As a rule there is no foot clonu A uniform persistent clonus, such as is so common in organic disease, extremely rare, but occasionally a spurious clonus, due to a half-voluntar contraction in the calf muscles, may be found. When the foot is first presse back there is no clonus, but presently a contraction in the calf causes slight extension movement of the foot, and with it a clonus occurs, whic varies from moment to moment, sometimes almost ceasing, and again renewe by a fresh contraction of the muscle. This form is characteristic of hysteri When there is a true characteristic clonus it is probable that there is a co siderable alteration in the nutrition of the motor elements, although this may have arisen from a primary functional disturbance.

Hemiplegia sometimes comes on suddenly, but more often slowly. It is at least three times as frequent on the left side as on the right. The loss o power is never complete; the leg is often more affected than the arm, and the face always escapes entirely—important distinctions from hemiplegia o organic source. Loss of sensation usually accompanies the loss of power but is more extensive in its distribution, affecting often the face and specia senses. Reflex action from the skin is not lessened on the paralyzed side, a it so often is in ordinary hemiplegia. The knee jerk may be normal or i may be increased, sometimes on both sides. The spurious foot clonus jus described may sometimes be obtained on the affected side and not on the other. The jerky character of the muscular contraction in the arm may render its movement unsteady, when the leg is simply weak. Not rarely some persistent contracture of arm or leg accompanies the palsy.

Bilateral paralysis, a sort of double hysterical hemiplegia, is sometime observed, although loss of sensation may be one-sided. In one case, a month after a whitlow on the finger, the patient, a girl, had some general pain, followed by inability to move the legs or arms, to swallow, or to speak. Th fingers became strongly flexed, with the thumb thrust between the two middl fingers. In this state she lay for a month. Then the fingers became relaxed the arms were moved a little, and at times a word or two was jerked out with difficulty. The influence of faradaism restored her speech in a few days, and in a fortnight she had regained full power of limb.

Disorder or ataxy of movement occurs in the hysterical in many forms and may exist alone or accompany loss of power. One form, well described by Briquet, in which movements are steady under the guidance of the ey but become irregular as soon as this guidance is withdrawn, is apparentl due to muscular anæsthesia. But this pathological condition has bee invoked without sufficient reason to explain other forms of incoördination The varying force of muscular contraction, which may be often recognize in the resistance to passive movement, imparts a jerky unsteadiness to voluntary movements. Sometimes these are steady enough while the patient is i bed, but on standing she at once sways, first to one side and then to th other. Or, without any loss of cutaneous or muscular sensibility, the patien may be unable to stand with the eyes closed, although perfectly steady whe the eyes are open, the effect of closure being greater than is ever seen i true ataxy apart from impairment of sensation. In other cases there is tendency to fall backward.

In the functions of the cranial nerves, the only other paralytic affectio of hysterical origin is the peculiar form of simulated ptosis described a p. 621.

Inaction of the diaphragm in hysterical patients may closely simulate a tru paralysis. The diaphragm naturally acts less in women than in men, and ma take little or no share in deep voluntary breathing, which is chiefly effecte by the superior thoracic muscles. In proportion as respiration is influence by the will the diaphragm is inactive. The effect of observation on an hysterical patient may be to render the breathing almost wholly volitional an to throw the diaphragm out of action. Repeated examination, and the diversion of the patient's attention, will generally reveal the absence of rea paralysis.

Spasmodic Affections.—The Protean character of hysterical disorders i nowhere more conspicuous than in the varied forms of spasmodic affectio to which the disease may give rise. They may be persistent or paroxysmal the former class includes the form of tonic spasm, or contracture, and th persistent varieties of clonic spasm; the latter comprehends the variou degrees and forms of hysterical convulsion.

The term "contracture" is applied to the condition in which the muscle become rigid in tonic spasm, fixing a limb or limbs in a certain posture, fo a few minutes or for a longer time. The contracture commonly succeed a hysteroid fit. Occasionally it is excited by some local injury or local pain as that of neuralgia or rheumatism; less commonly it comes on spontaneously In most cases it is uniform while it lasts, but occasionally the muscular spas may be felt to vary in intensity from time to time. It is usually greates when attempts are made to overcome it; the varying resistance of th muscles, then felt, is characteristic, and of much diagnostic importance. Th contracture usually persists during ordinary sleep, and is only relaxed by th deepest chloroform narcosis. In extremely rare cases, in which it ha existed unchanged for years, structural alterations appear to take place in th muscles; the contracture leads to organic shortening, and can no longer b removed even by chloroform (Charcot).

One form of contracture is that of the muscles of mastication, causing hysterical trismus, in which the teeth cannot be separated for more than a quarter of an inch. Usually succeeding a fit, it often continues until anothe convulsion, and then is gone. Sometimes it comes on spontaneously. I seldom lasts more than a few days, but it is prone to recur. One patient for instance, had an attack, lasting a quarter of an hour, three or four time a day for several weeks. Very rarely there is transient contracture in th depressors of the jaw, keeping the mouth wide open.

Contracture in the limbs may involve the arm, alone or with the leg on th same side, or the two legs may be affected, or it may be general. The ar is the most frequent seat. It is always rigid in flexion; the elbow is bent a right angle, or even still more flexed; the wrist is flexed; the fingers ar sometimes flexed at all joints, as in the "late rigidity" of hemiplegia, wit the thumb within them or thrust between the first and second fingers. Th contracture in the fingers is not lessened, when the wrist is passively flexe as it is in the late rigidity of hemiplegia. Sometimes the digits are flexe at the metacarpo-phalangeal, and extended at the distal joints, as in athetosis, from the preponderant contracture of the interossei; the posture the resembles tetany. The forearm may be pronated or supinated. Contracture in the arm may follow a fit, and may be transient or continue ti another fit. Injury is a very common cause of contracture; from this caus it usually comes on gradually and slowly; while local, it is often thought to be due to a neuritis, but if, as is sometimes the case, it spreads beyond the arm, involving the leg on the same side, or becoming universal, its nature is clear. As an instance of this cause may be mentioned the case of a girl of sixteen, who let a hot iron fall on her left wrist, burning it slightly. Immediately she felt a pain in the thumb and lost feeling in it. A week later, and in the course of a few days, first the thumb and then the fingers became flexed and rigid, one after another. Then the elbow became partially flexed, and soon afterward the foot was strongly extended and the toes bent downward. In the hand and foot there was fine tremor during observation. Sensibility was lost in the whole arm but nowhere else. Ovarian tenderness existed on the same side, and the contracture could be partially removed by firm pressure there, or by faradization of the limbs, but quickly returned. Under moral treatment, however, it gradually passed away, and at the end of two months no trace of it remained.

Hemiplegic contracture, such as the above case illustrates, is not very rare. Some loss of power is usually associated with it, and, as in simple paralysis, the face is always free from rigidity. Anæsthesia is rarely absent on the hemispastic side, and the loss of sensibility may be complete and involve the face and trunk as well as the limbs. Sometimes, as in the above case, the loss of sensibility is partial and is confined to the most contractured limb, or there may be absolute loss in it and a slighter loss over the whole of that side.

The contrast, in the case just described, between the form of spasm in the arm and leg, is common to almost all cases of hysterical contracture; the arm is fixed in flexion, the leg in extension, and the latter may be transformed into a rigid bar, fixed to the pelvis by unyielding spasm, with the heel so drawn up that the dorsum of the foot is in a line with the front of the leg. The foot is commonly inverted and the toes flexed. Partial contracture affects most the extremity of the limb. In these slighter cases the toes may be over-extended, in spite of the heel being raised. In hysterical contracture with extension of the ankle, the foot clonus can often be obtained, as Charcot has shown. It is analogous to the physiological clonus, which occurs in most persons after standing for some time on tiptoe.

Paraplegic contracture, affecting both legs, is decidedly less common. The position of the legs is that already described, rigid extension; the flexor spasm, occasionally seen in organic disease, is scarcely ever met with in hysteria. In very rare cases the contracture affects all four limbs.

Contracture, in any form, often disappears suddenly under the influence of emotion. Charcot has related several cases in which severe rigidity, of very long duration, was removed by strong emotion, not induced with any therapeutic aim.

Faradization of the limb may also remove it in some cases, and, when the current is not so strong as to exhaust the irritability of the nerves, the agency by which it acts is probably psychical. Usually, repeated applications are required, but sometimes the effect is instantaneous. One patient was thus cured in a moment, but months later relapsed, and then all treatment faile until she fell into the hands of a "miracle worker," at whose touch th spasm vanished amid the plaudits of a public audience.

Severe forms of contracture may last for years, and, as already stated, th changes in nutrition which follow, at a distance, the disturbances of fun tion, may, in time, attain such structural degree that the contracted muscle are permanently shortened. There is reason to believe that changes in th spinal cord may also result from the enduring functional disturbance, and after long years of spasm, sclerosis may develop. Charcot has recorde such a case, in which the contracture was certainly at first functional, disa pearing from time to time, but ultimately it became unchanging, and afte death the lateral columns of the cord were found sclerosed. It is to b noted that this change, as a primary lesion, is expressed by the same extensor spasm as constitutes hysterical contracture, though slighter in degree "Plastic contracture" may be present in the condition of induced sleep o hypnotism, to be mentioned later.

The contractures described above involve groups of muscles according t their function. A part of a muscle may also pass into a state of spasmodi contraction, evidenced, not by the distortion of the parts to which it i attached, but by the swelling of the muscular substance. A local "tumor is thus produced, which may simulate a morbid growth. Several instance of this have been recorded by Weir Mitchell; the swelling was in th calf in two, in the pectoralis major in a third, the contracted part rising hal an inch above the adjacent muscle. Allied in nature, although less simpl in mechanism, are the "phantom tumors" of the abdomen, in which th middle part of the belly, usually below the umbilicus, becomes prominen and appears as if bulged forward by a mass within. The mechanism b which it is produced appears to be a relaxation of the rectus and a spasmodi contraction of the diaphragm; the intestines, often distended with flatus are pushed forward in the region where the wall is lax. An arching of th vertebral column may sometimes contribute to the effect. Occasionally th enlargement of the abdomen is general, the wall being everywhere lax an the diaphragm contracted. The swelling disappears under chloroform, and as it goes, the liver dullness may be observed to rise in consequence of th relaxation of the diaphragm. The peculiar affection termed "tetany" (p 1050) sometimes occurs in the subjects of hysteria, but it is usually trifling i degree, and is limited to the hands. In some cases of hysterical tetanoi contracture the fingers are extended and separated by the spasm.

Clonic Spasm.—*Tremor* is very common in the hysterical, especially as a accompaniment of paralysis and contracture. It is rarely constant, but i usually evoked by movement and excitement. Frequently it does not atten the commencement of movement, but if the muscular action in the arms o legs is maintained for a few moments, the limb is agitated by fine quic tremor, varying in degree and in time, always increased, and sometimes distinctly induced, by attention, and when the mind is diverted the tremo may cease. When the legs are the seat of contracture the shaking is ofte caused by attempts to move them, or even by the manipulation of a medical examination. In all these respects it differs from the tremor of paralysis agitans, while the fineness of the tremor, and the absence of actual incoordination, distinguish it from that of disseminated sclerosis.

Such tremor, widening in range into what deserves the name clonic spasm, is occasionally paroxysmal, affecting the head or limbs, produced by emotion, and often to some extent under voluntary control. A young unmarried man, of highly nervous temperament, was liable to outbursts of hysteria, of which such clonic spasm was an invariable part. Under the excitement of a medical examination, for instance, his head began to shake violently. He said that the shaking in the head distressed him peculiarly, and that he could by an effort send the shaking into his leg, where it was much more bearable. Presently the right leg quivered in quick spasm, approaching in rapidity the foot clonus, and the head was still. Similar spasm, general in distribution, is a conspicuous feature of many of the hysteroid convulsions presently to be described.

Local and persistent clonic spasm is a most troublesome but happily a rare symptom of hysteria. An example of it was presented by a girl who had two attacks of such spasms in the left pectoralis muscle, jerking the shoulder forward with great force. The spasm was regular in time, but irregular in force, and during the waking hours it never ceased. Each attack lasted for several months. A circular blister around the arm arrested the first, but had no effect upon the second, which resisted all treatment until a journey to the South of Europe cured it. A similar spasm may sometimes be seen in the muscles of the neck, causing a sort of spurious torticollis. It is usually bilateral, jerking the head backward or forward; the movement is slight in range and quick in time, and thus differs from the more extensive and deliberate movements presently to be described.

Certain forms of widely-distributed clonic and irregular spasmodic movements have been called "hysterical chorea," but they differ much in their characters, and to most of them the term chorea can only be applied when used as a generic designation for all persistent involuntary movements. It is not uncommon, however, for witnessed chorea to be reproduced by the imitative tendency of hysteria, and the mimetic malady may closely resemble its original, the movements being varied and irregular; but much more often the muscular contractions are more sudden and shock-like than in true chorea, resembling in this the "electric" form. The imitative form is usually transient, and quickly vanishes when the patient is withdrawn from the influence of example. Occasionally, similar forms of choreoid affection come on in these patients apart from imitation, and these are frequently most obstinate affections, lasting sometimes for years. Often the movements, in the hands especially, have a rhythmical character. There may be, for instance, quick, regular, flexor movements of the fingers of each hand, all joints being bent. The movement is always increased by notice, and as it lessens in degree it may occur only when the attention is directed to it.

Rhythmical movements of more complex, and, therefore, more deliberate character, and of wider range, have also been included under the ter "hysterical chorea" by the French (after Germain Sée, etc.,) and consi tute the disease long known as "chorea major" by the Germans. Tl movements are wide in range and regular in sequence, and consist of alte nating contractions in opposing muscles, especially the flexors and extenso of limbs and trunks; they cause an oscillatory motion as regular as tl movement of a pendulum, ceasing only during sleep, and lasting for day weeks or months. The head may be thus moved from side to side, bacl ward or forward, the jaws up and down; or, less commonly, the tongu may be alternately protruded and withdrawn, the eyelids rapidly closed an opened. Much more frequently one limb is thus moved, or the arm an leg on one side, with or without the trunk, very rarely all four limb: Briquet has related a case in which the leg was flexed until the foot touche the forehead and again extended, in regular sequence, for more than a yeaı in spite of treatment, until the movements were arrested by a violer emotion.*

Rhythmical, coördinated spasm of these forms is analogous, as Charcc has pointed out, to that which occurs, in paroxysmal form, in many hysteı oid convulsions. In all cases it is probably pathognomonic of hysteria and may occur at the onset of other forms of hysteroid affection. A girl c seventeen, while at supper, became suddenly faint, and immediately the righ arm became the seat of rhythmical flexion and extension movements; iı half an hour the leg was affected in the same way, and then the other limbs and for five or six hours these movements went on without any spasm in th trunk. They ceased when sleep was obtained by a hypodermic injection o morphia, and there was no return, but the patient woke to pass into a stat of contracture and paresis which lasted for several months.

Convulsive Attacks.—Among the paroxysmal symptoms must be include the most frequent of all hysterical phenomena, the globus hystericus, and th paroxysmal manifestations of emotion, which have been already described Just as emotion is naturally expressed by muscular actions, in the "jumping for joy" of a happy child, the stamping of rage, or the wrung hands o distress, so the violent emotional discharge of hysteria may be associated in more severe cases, with violent movements of the limbs, purposive iı aspect, but purposeless in aim. The arms and legs are dashed about witl violence, the head is thrown from side to side, the back may be arched bj contractions of the spinal muscles. This is the mildest form of hysterica convulsion, unaccompanied by loss of consciousness—the "hysteria minor' of the French.

In still further degrees of intensity the convulsive phenomena become much more complex and severe. The attacks occur with less dependence on exciting emotion, and present more distinct alteration in the mental state. These are the attacks of "hysteria major" of the French, and from a frequent resemblance to epileptic attacks, they have long been known by some compound name, and especially, through the influence of Charcot, as "hystero-epilepsy." The term, however, applied as it is to phenomena of pure hysteria, is open to the objection of definite inaccuracy, and it is better to call these seizures "hysteroid" (a term suggested by Sir W. Roberts), which, although not perfect, is at any rate intelligible and not materially inexact.

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* A striking example of unilateral spasm of this character is related by Charcot, *Brit. Med. Journ., 1878*, i, p. 221.

In the severe attacks, rigid fixation of the trunk and limbs, often opisthotonic, alternates with wild movements, in which the limbs are thrown about with great force and rapidity, the arms strike out, the legs kick, the head is dashed from side to side. These phenomena may be varied by quiet intervals, often attended by hallucinations or delirium. Consciousness may be apparently lost, or manifestly perverted, and, as a rule, the patient retains no recollection of the fit. In the most severe and elaborate forms of attacks, such as occur especially among the French, certain stages may be distinguished, of which Richer has given careful descriptions and striking illustrations.* The attack is often preceded by a period of mental disturbance, with hallucinations. The onset is attended with sudden loss of consciousness, and general tonic spasm, followed by clonic spasm. These constitute the first or "epileptic" stage. Then, sometimes after a brief interval of coma, the second stage of coördinated spasm, or "*grands mouvements*," comes on, opisthotonos, bounding movements of extreme violence, succeeded by the third stage of mental and emotional disturbance, in which the patient talks deliriously with manifestations of joy, anger, or erotism. Tenderness of the ovarian region is almost always present, and by pressure there a fit of the above description may be at any time induced, or may at any period of the attack be at once arrested.

In this country the attacks rarely correspond closely to this description; they present similar phenomena, but in less regular sequence, and often in isolated form. The initial stage seldom presents any close resemblance to an epileptic fit. There is often initial tonic rigidity, and this is sometimes followed by a form of clonic spasm, succeeded by the coördinated movements, but the clonic spasm differs from that which occurs in epilepsy, and not unfrequently the patient passes at once into the violent coördinated movements, in which tonic and clonic spasm occurs from time to time.

It may be well to consider separately the various phenomena of these attacks as they are met with in this country. Their occurrence is often distinctly aided by, or due to, emotional disturbance—prolonged annoyance or sudden alarm. The premonitory mental disturbance with hallucination is never met with, but the attacks are often preceded for some hours by headache or some general dysæsthesia. Immediate warnings are not uncommon, especially a sense of illness, the throat *globus*, giddiness, palpitation of the heart, or some sensation commencing in *both* feet and ascending to the head. These warnings are common in pure hysteroid fits, but it is important to remember that these attacks sometimes occur as sequelæ to epileptic fits of slight or moderate severity (see pp. 1089, 1093), the warning of which must not be mistaken for the warning of the hysteroid attacl Usually, at the moment of the onset, there is no change in the color of tl face, but sometimes there is pallor for some minutes before the attack com on. The patients fall to the ground, sometimes with considerable violenc but they never suffer the injuries so common in epilepsy from falls upon dar gerous objects or on the fire. Very often the fall is gradual, a sliding to th ground rather than a fall. When there is initial tonic spasm, the limbs ar usually rigid in extension, the toes pointed downward. The arms may li by the side of the body, or be extended at right angles to the trunk in th attitude designated "cruciform" by Charcot. The fingers are usuall flexed at all joints, the fists being clenched. There is never, in this stag the "interosseal position"—the flexion of the metacarpo-phalangeal an extension of the other phalangeal joints which results from preponderan spasm in the interossei, and is so common in epilepsy, and in some hysteri cal contractures. During the existence of the tonic stage, the foot clonu can often be obtained, as Charcot has pointed out.

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* Études cliniques sur l'Hystero-epilepsie," Paris, 1881.

The opisthotonic spasm is one of the most characteristic features of hy teroid convulsion. It is the *arc en cercle* of French writers. It occur especially during the stage of coördinated movements, rarely at the com mencement of the fit, although the initial tonic spasm may pass into it There may be only slight arching of the spine, or the contraction of th extensor muscles may be so severe that the patient rests on the back of th head and the heels, and, in extreme cases, the trunk is pushed up by th feet, and the neck so much bent backward that the vertex, or even the fore head, is the anterior point of support, and it seems as if the neck would b broken. It rarely continues for long, but the patient, from time to time may sit up, and then bound backward into the rigid arch. Opisthotono may also occur as the patient lies on the side, the *arc en cercle latérale.*

The clonic spasm varies very much in its character. It never resemble precisely that of epilepsy, in which the movements are violent and shock like in their character, and cease by gradually becoming less frequent, no less strong. In hysteria the spasm is rarely shock-like, and always maintain the same frequency until it suddenly ceases. It is usually very quick in time Occasionally the conception of a universal clonus, like that of the foot, bu affecting whole limbs, conveys the best idea of its character. The fit ma commence with this, without any initial tonic spasm. In the rare cases ii which the spasm is shock-like, the shocks are infrequent and delib erate, and go on for a long time. Much more frequently the spasm i rather coarse tremor or quivering. The movements may affect the hand and feet only, so that these are struck against the ground with great rapidity The orbicularis palpebrarum is often alone affected, causing quivering o the eyelids, or quick opening or shutting movements. The tongue is neve bitten in the clonic spasm as it is in epilepsy, although, very rarely, it may b bitten accidentally in the fall. Hysterical patients, however, often bite thei own lips.

The coördinated movements which constitute so large a part of the attacl are, for the most part, wild, irregular "fighting" or struggling movements, in which the legs, feet and head are thrown about with great violence. The movements are usually without serial order, but occasionally certain movements are repeated in a rhythmical manner. The head may be moved from side to side; there may be regular flexion or extension movements of the legs (sometimes propelling the patient head first along the floor), and still more frequently a similar rhythmical movement in the arms. These coördinated movements are conspicuously increased in force by attempts to restrain them; the more force is used the more is needed; and their violence is often extreme. Accompanying them there is often delirious mental disturbance, most conspicuous during intervals of comparative rest or of alternating tonic spasm. Sometimes the patient talks in an unnatural manner, manifesting some hallucination. One girl, for instance, thought her long black hair, which had been flying about her head, was seaweed. Usually there is more or less emotional disturbance, especially manifestations of terror, which may increase to maniacal frenzy, and culminate in a paroxysm of convulsion. There is often a strange tendency to bite in a curiously animal manner, and occasionally noises of animals are imitated (therio-mimicry).* The patient will make a sudden gnash at the hand of an attendant, and if not prevented may inflict serious bites on her own fingers. In a prolonged seizure there are often intervals of tranquillity, in which the patient may seem well, but, by slight peculiarities in manner, those who know her are aware that "she is not yet herself," and presently the spasm suddenly recommences.

The eyelids in hysterical attacks are usually closed, rarely open. The eyeballs often converge strongly from time to time. Reflex action from the conjunctiva is usually lessened. General sensation is distinctly diminished; a pin may be run into the skin without causing any evidence of pain. In rare cases the attacks are characterized by violent laryngeal spasm, causing intense dyspnœa.

Pressure on the tender ovarian region, or other tender hysterogenic spots, as already stated, sometimes induces an attack, and prolonged pressure will often arrest the seizure. Sometimes the ovarian compression simply arrests the coördinate movements, and causes tonic spasm. The fits may be usually cut short more readily by strong faradization of the skin; by Dr. Hare's expedient of closing the mouth and nose for twenty or thirty seconds; by cold water, thrown on the face; or, if the mouth be open, by pouring into it a little water so that this gets to the larynx and causes a choking cough. These methods clearly act by a strong stimulation of the sensory nerves, or of the respiratory centre. The duration of the attacks, if left alone, varies extremely. Some last only for a few minutes; more frequently they continue for a quarter to half an hour, or even for several hours. Occasionally transient contracture of limbs, or trismus, or local paralysis, or anæsthesia succeeds an attack.

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* See "Epilepsy and other Chronic Convulsive Disorders," p. 140.

The severe hysteroid seizures are very common in young women and gir at the time of puberty, and sometimes earlier, even at seven or eight yea of age. They are by no means rare in boys, and may occur in young adu men.* They are not confined to the waking state, but may occur also i sleep. The frequency with which they are sequelæ to slight epilepti seizures, as already mentioned, is an extremely important fact.

Cerebral Symptoms.—The conditions of hemianæsthesia, paralysis, an contracture must be regarded as the expression of a condition of restraine function (inhibition) or unrestrained activity, of certain cerebral centre sensory and motor. In rare cases a similar perversion of function ma involve higher centres; the patient may pass into the peculiar sleep-lik state of "trance" or "lethargy," with or without the condition of plasti rigidity of limb denominated "catalepsy" (see p. 1331). Such conditior are met with chiefly in the subjects of hysteria; they may come on in paroxysmal attacks, as at a certain time each day, sometimes in the evening Although spontaneous trance is rare, the condition, as Charcot and hi pupils have shown, can be readily induced in hysterical patients, and in th hypnotic state there may be an extraordinary increase of excitability in brain nerve, and muscle (p. 1330).

Other disturbances of the psychical functions occasionally occur in hysteria and are very important. The ordinary mental characteristics of thes patients have been already described. In rare cases there may be such a amount of mental disturbance as to raise the question whether the patient i actually sane, and sometimes the limits of sanity are distinctly passed. Th relation of insanity to hysteria is a subject, however, on which generalization is difficult, and various opinions are held by competent authorities Mental derangement, like other nervous diseases in females, may be accompanied with symptoms of hysteria, without the association proving any essential relation. Sometimes, however, the pronounced mental aberration seem distinctly to grow out of the slighter psychical disturbance of hysteria, an the process may be apparently similar to that which leads to the more grav corporeal symptoms. Just as a mind unfixed by emotion may become se in its unbalanced state by a fixed idea of motor inability, and the latter ma progress to extensive paralysis or contracture, so the intellect may becom the prey of such an idea as must be regarded as an insane delusion, unde the influence of which the mental powers may fail further. An emotiona girl, for instance, with frequent globus, became possessed with the idea tha all persons whom she met were making faces at her, and then she manifeste timidity, infantile in its degree, so that she dared not go out of the hous alone, and became irritable and depressed, with a vacant look, and suc loss of memory as to constitute partial dementia. Such cases may be mos various in their form, and the ultimate symptoms may resemble those o simple insanity, from which they are to be separated, if at all, only by th conditions of their origin.†

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* See "Epilepsy and other Chronic Convulsive Disorders," p. 140.

† I have related examples of these forms in "Epilepsy," p. 165, etc.

There is another important relation of hysteria to insanity. The later stage of a developed hysterical fit is a paroxysm of mental disturbance of maniacal violence, in which chaotic storms of emotional frenzy alternate with hallucinations and delirium. The patient is, for a few minutes, wildly insane. Just as some of the motor spasm of the convulsion may continue when it is over, or may occur alone as persistent contractures, so some elements in the paroxysmal mental disturbance, hallucination or delirium, may occur alone. In an hysterical child, whose case I have described at length elsewhere,* apart from the delusions that accompanied the convulsive seizures, there were occasional periods of spiteful mischievousness, wholly foreign to her habitual character, and also periods of a semi-demented state, lasting for days or weeks. The subjects of such mental disturbance may threaten or attempt suicide, apparently with every intention of success. In another patient,† a girl of twenty-six, violent delirium accompanied each hysteroid convulsion, and after a time paroxysms of mental disturbance occurred without any preceding fit. After an interval of improvement, a condition came on which rendered her confinement in an asylum necessary. She recovered, but relapsed, in consequence of a mental shock, and passed into a very unpromising state, with most degraded habits, but ultimately became well.

Visceral and Vasomotor Symptoms.—The digestive, respiratory, and circulatory systems are liable to be deranged in hysteria. Some of the resulting disorders are very remarkable in character, and indicate how profound may be the secondary influence of the disease on subordinate functions.

The sensation of the globus hystericus may be accompanied by actual pharyngeal spasm, and occasionally this spasm occurs in paroxysmal form, accompanied by an intense feeling of suffocation. Swallowing may be impossible for hours, sometimes for days. The œsophagus also may be the seat of spasm, and food is then regurgitated before it reaches the stomach. Vomiting is much more commonly from the stomach itself, and constitutes one of the most frequent and most enduring of the visceral disturbances. Food is rejected, usually in less than a quarter of an hour after it is taken. Sometimes gastralgia exists, and the presence of food in the stomach excites pain, which seems to cause the vomiting, but more commonly the vomiting is painless, and unaccompanied by nausea. The symptom, like many others in hysteria, may be set up in the first instance by an adequate attack of real gastric disturbance, accompanied by nausea, but persists when this is over, as a morbid habit, involuntary in character, but often yielded to by the patient, and sometimes actually induced by the will. The stomach is not, it is true, under direct voluntary control, but vomiting may be produced by an emotion of disgust, and the needed emotion may be called up by an idea without sensorial agency, as is evinced by the strange cases in which the vomiting of pregnancy makes the husband retch, and at last the latter becomes sick as soon as he knows his wife is pregnant.‡ The connection may become so strong that the idea will produce the act without t agency of adequate emotion, and food is rejected on a thought of sickne which may arise unbidden, and is easily called up. It is not probable th the stomach returns all the food taken, for patients kept at rest may not lo weight, although they constantly vomit their food. A very small quanti seems sufficient for the needs of the system at rest. Conjoined with vomi ing, or existing alone, there may be absolute anorexia, and these togeth may render feeding by the mouth impossible.

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* "Epilepsy," etc., p. 157.

† Loc. cit., p. 172.

‡ Weir Mitchell, "Nervous Diseases in Women." A similar case has been related to me by my colleague, Dr. John Williams.

Such hysterical anorexia has given rise to the extraordinary cases of "fas ing girls" which, in all ages, have excited popular wonder. In many these, however, there has been unquestionable fraud. When abstinence fro food is complete, the body invariably loses weight, and the absence of th progressive loss is certain proof of deceit, which is stimulated by the intere excited, and aided by the friends to secure the contributions of amaze benevolence. Even in slight cases of hysteria, deliberate deceit is mo common in this than in any other symptom. Some patients, who would n deliberately assume a positive symptom, fall easily into the habit of fastin at meals and eating on the sly. But in some cases of severe hysteria ther is absolute abstinence from food for many days, and even during weeks th quantity taken may be so small as to be insufficient to prevent the sel starvation from producing conspicuous effects. In a case related by Wei Mitchell, no food was taken for ten days, and, during five weeks, onl twenty-four ounces of milk were swallowed. Another patient swallowe neither solid nor liquid for twenty-seven days. In some cases, forced feed ing by the mouth or rectum may give rise to such severe convulsions tha the attempt has to be relinquished. The patients lose weight, and becom emaciated and feeble in extreme degree; the temperature may be raised the tongue become dry and brown; they may pass into a condition of stupor and not only appear in danger of immediate death, but may actually sink from exhaustion. In most cases, however, when this alarming state i reached, a little food is again taken, and the patient slowly revives. Th danger is greater in cases of prostration from long-continued vomiting tha from simple refusal of food, since forced feeding is less effectual in the former. One patient died on the eighty-second day of the vomiting, in spite of the use of the stomach tube.*

Dyspepsia, in every form and degree, is common in the hysterical, and the nerve disturbance thereby excited may profoundly intensify other symptoms. Sensations of "sinking," palpitation, breathlessness, flushing of the face, pains in the back, which often attend dyspepsia, may reach an extreme degree in the subjects of hysteria. Flatulence, gastric and intestinal, is very common, and gives rise to much discomfort and to varied sounds, some of which are apparently the effect of an involuntary contraction in the muscles of the abdominal wall. Complicating and intensifying these troubles, there is often constipation, which, so common in women, is peculiarly obstinate in the hysterical. The bowels, left alone, may act only once a week, or even once a month. Whether this is from a nervous inhibition of the intestina wall, or from mere indisposition to aid the bowels by timely attention, is difficult to say. It is certain that the muscular action of the intestines may be gravely deranged in severe hysteria. A strange case is recorded by Briquet, in which liquids injected into the rectum were vomited by the mouth. Even tincture of litmus was vomited twelve minutes after its injection into the rectum, the patient being watched by doctors and nurses the whole time. That there is pathological inactivity of the bowel is probable from the large amount of purgative which is needed. Occasionally there is a peculiar sensitiveness to the action of the bowels, so that the passage of a stool, especially if relaxed, may cause sensations of faintness, and even actual syncope.

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* Guyot, *Gaz. Méd. de Paris, 1882*, p. 206.

Retention of urine is common in the hysterical, but incontinence is almost, if not quite, unknown. Calls to micturate are often annoyingly frequent, but this usually depends on the character of the urine, which becomes abundant, pale, and of low specific gravity, and seems to irritate the bladder much more than that which is less dilute. Such urine is probably due to dilatation of the renal vessels. It is secreted in most persons under the influence of emotion, and may be almost constant in the persistent emotional state of the hysterical. In rare cases the secretion is changed in the opposite way. Instead of being increased it is lessened, and the diminution may go on to absolute arrest, "hysterical ischuria" and "anuria," which have been carefully studied by Charcot. Complete suppression may last for ten days without any symptoms of uræmic intoxication such as, in anuria from calculous obliteration of the ureters, usually supervenes before the end of a week, and causes death in two or three weeks Considerable diminution of the urine is always attended with vomiting. The quantity of liquid vomited varies inversely with the amount of urine secreted and the vomit contains some urea. In a case recorded by Weir Mitchell severe vomiting alternated with profuse perspiration, which left a thin film of urea on the skin. To the supplementary excretion, and to the facts tha little food is retained and that the tissue changes, the patient being at rest are extremely slight, Charcot believes that the tolerance is to be ascribed The symptom itself is probably due to spasm of the renal vessels, and i comparable to the arrest of the secretion which occurs in an animal whe the abdomen is opened. Although in considerable degree and duratio ischuria is extremely rare, in slight and transient form it is not infrequen (as Laycock pointed out), being often ascribed to retention in the bladde

The disturbance of the functions of the sexual organs that exists in hysteria has been already sufficiently described in the section on causation.

Respiratory Organs.—Intensely rapid breathing, fifty, sixty or eight respirations per minute, is not an unfrequent symptom, and is often terme "hysterical dyspnœa." There is no real difficulty of breathing, and th patient's pulse may not be more frequent than is habitual. Such extrem rapidity, without other signs of dyspnœa, is almost confined to this disease When conjoined with hysterical pains in the chest, the resemblance to intra thoracic mischief may be perplexing. Actual dyspnœa, most intense i degree, may, however, attend another hysterical disorder, laryngeal spasm which may occur in violent paroxysms. Stridor and cyanosis attest th severity of the manifest struggle for breath, the muscles of the neck stan out in violent action, blood may be hawked up in the straining, and th patient may even appear to be on the point of death by suffocation. Th attack may sometimes be arrested by closing the mouth and nose and causing actual apnœa, by tickling the pharynx and thus inducing nausea, an still more effectually by an injection of apomorphia. Persistent cough i another occasional paroxysmal symptom—often hoarse and croaking. Hiccough is also sometimes a troublesome and enduring symptom.

Vasomotor Symptoms.—Neurotic disturbances of the vascular system are in slight degree, extremely frequent, and often give rise to great discomfort Occasionally they are severe and even violent. The heart's action may b habitually too frequent, and may be readily accelerated or rendered irregular by any trivial emotion, or by the common gastric derangement, an intermission or tumultuous action may occur, apparently without cause There is undue consciousness of the action of the heart, normal or irregular which is described as "palpitation." It is often a source of much distress and may be accompanied by cardiac pain and giddiness, by sensations o sinking, of dyspnœa, and of faintness, by extreme pallor, and even by actua syncope. Sometimes the deranged innervation is shown, not only by a habitual frequency of 120 or 130 beats per minute, but by a reversal of th usual effect of posture on the pulse. The attacks of pain, with pallor, faintness and dyspnœa, may closely counterfeit genuine anginal seizures, especiall when the pain, as is sometimes the case, radiates to the left arm; I hav seen repeated attacks of this character in a nervous, overworked man, whos mother had died in an attack of true angina pectoris, of which he had alway lived in dread. Much less frequently the heart's action becomes less frequent especially in some of the cases of spontaneous trance.

Disturbances in the peripheral vasomotor may be associated with the perturbed action of the heart, or occur independently. Flushing of the face sometimes local in distribution, is exceedingly common. It occurs spontaneously or on slight emotion, and from the misconstruction to which i gives rise is a source of great annoyance to the patient. Flushing and pallo may alternate, or the face be red and burning while the feet and hands ar cold. Flushing of the feet, with a sensation of burning heat, often alternates with cold, and much more rarely there is persistent fullness of th vessels of both legs. Weir Mitchell has recorded an extraordinary case i which at times, through emotion or spontaneously, a paralysis of the abdominal vessels seemed to concur with vascular spasm elsewhere, so that the fla abdomen of a thin, spare widow would attain, in the course of a few hours a size corresponding to the sixth month of pregnancy, and be turgid an pulsating, while the rest of the body was pale and bloodless, the pulse at th wrist being a mere thread, and faintness ensued if the patient sat up. Th state would subside in the course of a few days, and was the source of infinit annoyance to the patient.

Local perspiration is a rare symptom. In one case the hands and fee were the seat of profuse sweating, which alternated with attacks of lethargy double amaurosis, paraplegia and anæsthesia (Siredey).

Vasomotor spasm seems sometimes to accompany hemianæsthesia, so tha pricks do not bleed, but, as we have seen, it is no necessary concomitant in complete hemianæsthesia the vascularity of the skin may be normal an pricks may bleed readily.

Occasionally local swellings occur, especially about the hands or feet, o about joints, sometimes in the seats of neuralgic pain. They are due apparently to the effusion of serum into the cellular tissue in consequence o vasomotor disturbance, and often occur near the time of the monthly period

It is probable that, in rare cases, the vasomotor disturbance may lead t the occurrence of small hemorrhages into the skin, but extravasations i definite spots, as in the "stigmata" of crucifixion, are always of artificia origin. Hemorrhage from the stomach has been thought to be vicariou with the menstrual flow, but in at least some of these cases actual ulceratio has existed. Hemorrhage from the lungs is unknown except in actua disease, although the straining of laryngeal spasm may rupture small vessel in the throat and windpipe, and small clots may be coughed up. A form o spurious hæmoptysis is, however, not uncommon; the patient spits a brownish-red liquid consisting of saliva uniformly mixed with blood, the source o which is probably the gums.

When an hysterical patient emaciates from self-starvation, the skin ma become dry and the epidermis scale off. Artificial skin eruptions are ofte produced by means of irritants, such as cantharides, by the lowest class o hysterical patients, who may exhibit much cunning in baffling detection.

The temperature in hysteria is, as a rule, normal, but in severe cases slight rise occurs at some period of the day, a circumstance which is no surprising when the severe vasomotor symptoms are considered. The registering thermometer, however, furnishes some hysterical patients with a irresistible temptation to fraud. By friction or pressure on the bulb, or b the dexterous manipulation of hot bottles or poultices, the index may b driven up even to the top of a clinical thermometer, and the patients enjo the wonder which has been excited when temperatures of 120° or 130° hav been registered by special instruments.

Course and Terminations.—The severe nerve disturbances of hysteria ma persist in continuous or recurrent form for a short or long period, usually fo months and sometimes for years. Recurring convulsive attacks and persistent contractures are perhaps the most enduring. But the transitory manifestations of the disease are merely the symptoms of an underlying stat which is from its nature persistent, and is always to be measured by years often by a lifetime. In its slighter forms it is as much a temperament as a disease. If it is moderate in degree, and the patient can be placed i favorable circumstances, it may, after a time, cease to be recognizable, espe cially when the emotional disturbance of youth has passed into the steadie career of the woman; when its degree is greater it may survive even th latter, and the influences that should steady the mind may only cause add tional perturbation.

Over the slighter forms of hysteria, the moral influences involved in mar riage exert a distinctly beneficial influence, especially in the social class an conditions in which it involves activity of mind and body for definite an adequate objects. But the severe forms of the disease, especially with long continued convulsions, may be unrelieved by marriage and even by mater nity, and may be distinctly aggravated by the anxieties which are neve altogether absent from the married state, and which not rarely constitute th greatest trials life can bring. Hence, severe hysteria often persists to middl life and even beyond it; in some cases to cease, in others to be intensified at the climacteric period, and occasionally to continue until old age.

The course of the disease is always varied. The infinite diversity o symptoms which different patients present, may be almost paralleled in th history of individual cases; palsies, motor and sensory, contractions an convulsions, dyspnœa and dysphagia, anorexia and aphonia, faintings an vomitings, may succeed one another in apparently inexhaustible variety.

In what proportion of cases a practical recovery takes place cannot b determined. As a rule, however long the symptoms have lasted, the remain disorders of function only, and their complete disappearance is stil possible. To this an exception must apparently be made in the case of per sistent contractures, which, as already stated, after lasting for many years may be attended with structural changes in the spinal cord; such cases are however, excessively rare.

Recovery from hysteria is probably always gradual, although it is ver common for the individual symptoms to cease suddenly, especially unde some profound emotion. The same agent which disturbed, in a give direction, the unstable balance of the nervous system, may readjust the equilibrium, but without rendering it more stable than before, and such influ ences are never permanent in their effects. Nor are they always efficien even in their limited degree. It has been said that an hysterical paralytic will always run out of a burning house, but Weir Mitchell has mentioned an instance in which under such circumstances the patient only fell down helpless, and another instance of this has come under my own notice. A sufficient motive for sustained, unselfish exertion is much more effectual, and many an hysterical girl has recovered health under the necessity of rising from her couch to exchange the part of invalid for that of nurse.

Pathology.—Many points of the pathology of hysteria have been alluded to in the account of the characters and causes of the disease, and our knowledge of the subject is so scanty that little more remains to be said. The teaching of anatomy is purely negative. The changes which, in rare cases, have been found on post-mortem examination, have clearly been either accidentally associated, or have merely served to evoke the condition which in other cases exists as an independent malady. The only symptoms common to all forms of the affection are those of certain cerebral functions, and we are not justified in looking beyond these for the primary derangement. It is clear that whatever influence disorders of other organs (as those of the uterus) may exert, they merely excite the manifestation of a disease already existing.

Hysteria is probably the most perfect type of a functional malady. It not only consists in, but arises by, a functional disturbance, a loss of the due balance between certain of the higher functions of the brain. But many, probably most, of the definite groups of symptoms depend on the secondary derangement of lower centres. Thus hemianæsthesia must be ascribed to an inhibition of a sensory centre relatively low, while hysterical anuria shows that the secondary disturbance may reach nerve functions furthest removed from those in which it commences.

It is important that this definite affection of lower centres should be clearly recognized, because there is still too strong a disposition to consider hysteria not only primarily but altogether as an affair of brain and will That it is so originally there can be no doubt, and it is also certain that the disease can only be really cured by the restoration of the normal balance of cerebral function, but the secondary disturbance of lower centres (*e. g.* the vasomotor) may at times preponderate over that which can be recognized in the highest, and may be independent in their degree, and in some case apparently (though not really) in their occurrence.

This disturbance of lower centres has disclosed facts, hitherto scarcely suspected, and of much physiological interest, regarding their capacity fo limited functional derangement. We may take, for instance, the remarkable phenomena just mentioned, of hemianæsthesia and of transfer. This unilateral loss of sensibility not only shows that the sensory centres on one side of the brain may become inhibited in a peculiar way, partially or wholly but that by certain agents a partial arrest of the inhibition may be effected so that a given area in the anæsthetic region again becomes sensitive. The phenomena of transfer (of the genuineness of which, in spite of its rarity out of France, there can be no doubt) show that there must exist an intimate connection between the sensory centres of the two hemispheres, so that the restoration of functional action in a part of the inhibited centre i accompanied by an arrest of action in the corresponding part of the centr on the opposite side. The validity of this inference is independent of th mode by which the phenomena are effected, or of the exact functiona change in which they consist.

Our knowledge of the nature of the primary disturbance that constitute the essential element in hysteria is too visionary to render its discussion o practical value. Modern physiology teaches that there is a complex interaction between all parts of the nerve centres, and suggests that functiona activity and inactivity are determined, not only by the power of generatin nerve force, but by varying degrees of restraint or inhibition. Lower centre are controlled by higher ones, and controlling centres are themselves subjec to restraint. It is easy thus to conceive that some defect in the highe centres may lead to a disorder involving either part or the whole of th lower centres, although we may be quite unable to trace the process b which the disorder arises, or the conditions that determine its limitation.

In speaking of hysteria as a functional disease, it is not denied tha changes in the finer nutrition of the nerve elements may underlie and resul from it; but they are not, even in extreme degree, recognizable by ou present methods of observation. To this, one exception must be made spinal sclerosis may develop from contracture—an almost unique example c structural change from functional disturbance.

It is scarcely worth while to discuss the pathology of the individual symptoms of hysteria. The little that is known has been mentioned in thei description, and the application to them of the general principles just enunciated would be merely to repeat these principles in more special terms.

Diagnosis.—The general condition of hysteria is usually recognize without difficulty. The varied and varying discomforts of which th patients complain, and for which no organic cause can be discovered, th frequent globus and the mental state are at once readily recognized. Th special symptoms, when severe, present greater difficulties, and occasionall give rise to perplexing diagnostic problems. Not less difficulty is presente by the cases in which symptoms of hysteria accompany and complicat other diseases. Slight organic disease may be overlooked in the presenc of hysteria, while the opposite error, that of mistaking hysteria for organi disease, is more frequent in the severer special manifestations of the functional malady.

A very important element in the diagnosis of the special forms of diseas is furnished by their conditions of origin. The patient is of the sex and ag in which the tendency to hysteria is strongest, or, if a man, presents thos nervous and somewhat effeminate characteristics which usually accompan the disposition to the disease in the male. These considerations receiv additional weight from the presence of the slighter and more constant indications of hysteria which have been already alluded to. But it cannot b too strongly insisted upon that this element in diagnosis, although of grea value, is second in absolute importance. The first and most important consideration is the absence of any unequivocal symptom of organic disease Women who suffer from other diseases of the nervous system are frequentl also the subjects of hysteria, and it is clear that, in the presence of distinc symptoms of organic disease, the evidence of hysteria is of no significanc as regards the nature of the malady, and only becomes significant when th absence of evidence of such other disease has been ascertained. The firs step in diagnosis is, therefore, to search for any symptoms which indicat organic affections, and this involves a knowledge of the diagnostic symptom of almost every other disease of the nervous system, since there is scarcel one which may not be simulated by this Protean malady.

In the absence of such unequivocal evidence of organic disease, the presence of hysteria, or of the conditions favorable to its existence, becomes c important significance. It does not, however, in itself constitute evidenc that the symptoms are due to this cause; the manifestations of organic disease are sometimes in themselves equivocal, and are such as may be also produced by hysteria. But, as a rule, the grouping of the symptoms differs in the two, and from their definite arrangement and sequence a confident diagnosis can often be made. Cases are occasionally met with, however, in which even the largest experience and the utmost diagnostic skill are needed, and may even for a time be baffled.

Certain general characteristics of hysterical maladies deserve special mention. Concerning all of them, it is to be remembered that their presence is more significant than their absence. One is the relation of the symptoms to emotional disturbance, both in their commencement, course and manifestation. They frequently follow a severe mental shock, or are gradually evolved under the influence of more persistent emotional disturbance, and may be intensified from time to time under the same influence. They increase when the patient's attention is directed to them, and lessen when this is diverted. Symptoms often become greater during the course of a medical examination. Speech, that is at first distinct, becomes stuttering and hesitating, or voiceless; limbs that are at first relaxed and still, become rigid and tremulous; a patient who at first can stand, suddenly sinks helpless to the ground. Symptoms which simulate those of organic disease rarely correspond closely to the ordinary type of that which they counterfeit; some symptom exists in excessive degree or is absent.

Another important indication is the mutability of the symptoms in hysteria. Grave troubles of one character may suddenly cease, and give place to other symptoms such as could not result from the same organic cause as the first. Of equal importance is the fact that some symptoms of undoubted hysterical character concur with and form part of a group of disturbances otherwise equivocal, and give a clue to their character. For instance, a girl, in the course of a few hours, became paraplegic, with altered speech and attacks of spasm and tremor in the limbs; the case was regarded, by some physicians who saw her, as one of acute spinal mischief. But the onset of the symptoms was preceded, for some hours, by slow rhythmical spasm—alternate flexion and extension of the limbs; the movement commenced in one arm and then gradually became universal. This is a form of spasm characteristic of hysteria, and alone indicated the true nature of the disease.

At the same time it must not be forgotten that the converse of this holds good. Just as, on the one hand, the occurrence of a characteristic hysterical symptom may explain the nature of other symptoms, in themselves equivocal, so, on the other hand, a single symptom of organic disease may show that equivocal symptoms are not hysterical. For instance, a single epileptiform convulsion, beginning locally, may prove that other symptoms (headache, etc.) ascribed to hysteria were really due to organic brain disease. I was once shown an emotional young woman who was supposed to be suffering from hysterical aphonia and hysterical hemiplegia. A laryngoscopic examination showed that one vocal cord was completely paralyzed, the other moving freely. Laryngeal paralysis due to hysteria is double; unilateral paralysis is always of organic origin, and the case proved to be one of syph litic disease at the surface of the medulla oblongata.

It is unnecessary to consider in detail the diagnosis of each of the forn of hysterical disorder, since their leading and characteristic symptoms hav been already described, and the diagnosis rests, in each case, on the dete tion of these characters, and on the general principles just enunciated. Th distinction of the severe convulsive affections from epilepsy is described i the section on the diagnosis of the latter disease.

PROGNOSIS.—In no disease does the prognosis, as regards life an recovery, present greater disparity. The danger to life from hysteria, eve in its most severe forms, is extremely small. Very rarely exhaustion cons quent on vomiting, etc., terminates in death, and still more rarely sever laryngeal spasm (or more probably paralysis) has led to a fatal resul Other affections, however severe, are practically devoid of danger.

The prognosis as regards recovery varies with the circumstances of th individual case, according to the severity of the disease, and to the efficienc with which appropriate treatment can be adopted. As a rule, the specia symptoms of hysteria can be removed, but the morbid state of the nervou system, of which they are the manifestations, persists in greater or les degree, so that it has been said that complete recovery is as rare as is deat from the disease (Jolly). This is an exaggeration, but it is certainly tru that medical skill can never by itself secure recovery, although, if th patient's conditions of existence are favorable, if a life of active work an satisfying aims can be substituted for the purposeless years which commonl succeed puberty, it is by no means rare for all traces of the hysterical tem perament to disappear, and a stability of character to be developed whic can endure even severe trials without giving way.

Of individual groups of symptoms, some are more readily influenced b treatment than others. Motor paralysis and the slighter forms of convul sion are those most easily removed. Hysterical anæsthesia is more obstinate and usually persists long after any accompanying loss of motor power ha passed away. The local forms of spasm, both the common contracture an the rare clonic spasm, are much more enduring, and so also are hysterica vomiting, local pains and tenderness, especially of the spine, and the sever forms of hysteroid convulsion which are sometimes even more difficult t influence than epilepsy itself. It should be noted that the hysterical fits i which delirium is the most prominent feature involve some danger o insanity. I have more than once known such patients to become insane In all conditions the general law is true, that the prognosis is favorable i proportion as definite causes can be traced and removed, especially deterioration in the general health, of remediable character.

TREATMENT.—The predisposing causes of hysteria are to some degree avoidable. The psychical condition from which it springs, and in which i largely consists, may be in part prevented by careful training, especiall during the transition from childhood to youth and womanhood. The characters of the training needed are suggested by the description of the cause of the disease already given.

The treatment of the developed malady is partly that of the special symptoms, partly that of the underlying condition which causes them. The latte is infinitely the more important; the removal of special symptoms may d nothing for the real cure of the patient, and at best is but a small step i that direction. Nevertheless, their treatment, useless alone, is a valuabl adjunct to the removal of their cause.

The causes of hysteria are partly moral, partly physical; and treatment to be effectual, must correspond. The success of the measures adopted wil largely depend on a careful study of each case, and an accurate recognition of the relative amount of physical and mental disorder. The firs element in treatment is the removal of whatever defect in the genera health or local disorder of function can be discovered. Appropriate measures for this purpose should always accompany and often precede mor special treatment. In very severe cases, and especially after tonics an change have been tried without success, a method devised by Weir Mitchell and extensively carried out in this country by Playfair, will often succeed It consists in keeping the patient absolutely at rest in bed, and obtainin the tonic influence of exercise by daily massage and electricity—skilled rubbing and kneading the muscles, and putting them in action by faradaism At the same time abundant food is given in an easily digested form. B this method the wearying effects of fatigue are avoided, and patients ofte gain flesh and color rapidly. It must be remembered, however, that th system is only needed in severe cases, and that the skilled rubbing and electricity are essential. Without these rest in bed will probably convert th patient into a helpless invalid. This method has, however, another use it affords an effective vehicle for moral treatment. For this purpose th patient is isolated during the course, except from the necessary attendant The isolation is not only of great influence in itself, but affords an opportunity for influencing the mind, and to this the unquestionable success o the treatment is largely due.*

Of organic derangements requiring special treatment, those of the digestive organs are the most frequent. They need special adaptation of the toni treatment and careful diet. Constipation is often troublesome, and ma task to the utmost the resources of the physician and the pharmacopœia. I is of the greatest importance to secure daily action of the bowels, for whic moderate doses of aperients will often suffice, when very large doses ar ineffectual to overcome the accumulations of a longer period. In obstinat cases, in which enemata have to be employed, it is best to combine thes with aperients, and for the injection to be given at the time at which th medicine ought to act; the two influences, thus united, will often be e fectual when each, separately, is powerless.

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* The details of the treatment are described in Weir Mitchell's books, "Fat and Blood, and "Nervous Diseases in Women," and by Playfair, "The Treatment of Nerve Prostration and Hysteria," 1882. Twenty years ago, Russell Reynolds ("On Paralysis and othe Disorders of Motion dependent on Idea," *Brit. Med. Journ.*, Nov. 6th, 1869, p. 483) pointe out the value of massage in the treatment of these palsies.

The ovarian tenderness and pain, as a rule, need no local treatment; the are neuralgic in nature, depending on the general condition of the nervou system, and pass away with the latter. The treatment of uterine derange ments needs much judgment and caution. It has been already stated tha uterine conditions which cause no obtrusive symptoms have rarely any influ ence on the disease, and they should, as a rule, be left alone. Both amenor rhœa and menorrhagia disappear when the general health is improved. I there are symptoms that suggest positive uterine disturbance, such as sacra pain, "bearing down," or great menstrual discomfort, the question o local treatment has to be entertained. If the patient is a married woman, i is well that, at the outset, the actual condition should be ascertained by vaginal examination, and any distinct derangement put right; it bein remembered, however, that mere uterine tenderness is often simply a neuroti hyperæsthesia, and is aggravated instead of being relieved by local treat ment, and that even uterine displacements are not always either morbid o remediable. If the patient is unmarried, local treatment, and even a singl vaginal examination, is often prolific of indirect evil, and it is better, eve if the local symptoms are troublesome, to see, first, the effect upon them o the improvement of the general health and nerve state, and only when thi fails should the state of the uterus be ascertained. It is extremely doubtfu whether any local condition, for the discovery or treatment of which th speculum is necessary, has, in the virgin, any influence on the origin o course of the symptoms of hysteria.

The success that has followed the removal of both ovaries in very sever and long-continued cases has been before mentioned; it may have been du much more to the direct than to the indirect effects of the operation Whether or not this is justifiable in any case is a grave and open question It must be remembered that there is no degree of the disease that preclude the hope of great amelioration by other means, and that the operation i similar in its nature to the removal of the testicles of a man.

The moral treatment of hysteria, as it is the most important, is also th most difficult; tasking to the utmost the insight, ingenuity, and persever ance of the physician, and making a demand on the patience and wisdom of the sufferer and her friends, which too often meets with so imperfect response that the best efforts are fruitless. In most cases the conditions o home life in which the disease has developed are not conducive to recovery Even when the moral atmosphere of home does not aggravate the disease its influence for good has probably been long exerted in vain, and a wis change of scene and companionship constitutes an important aid to treat ment. In severe cases, however, more than this is needed, and it is neces sary that the patient should be brought under the moral influence of strangers who can combine tact in management, firmness in control, and ingenuity i expedient. This influence may sometimes be found in a family whose hom life the patient may share. This method, when practicable, is the most rapid and effectual. The success of this element in treatment depends on the person selected, who must be cheerful and discreet, and, if a nurse, should possess at least such education and refinement as shall prevent annoyance. The Weir-Mitchell treatment offers, as already mentioned, an alternative method of securing this influence.

The first step, the separation of the patient from her friends, is usually the most difficult. Affectionate relatives cannot perceive the wisdom, much less realize the need, for a proceeding which always has the aspect of harshness, and usually involves distress to the patient, salutary it may be, but no the less painful. As a rule, the more prejudicial to the patient home influences are, the less easy is it to remove her from them. Now and then some secondary point in treatment can be made to justify the course more readily than the primary object. This is the case with the Weir-Mitchell treatment in which there is something "to be done," and an ostensible reason for the removal of the patient. In a severe case, if home influences are unfavorable, it is wiser to decline to undertake treatment that will probably end in failure than to have recourse to half measures. These will probably have no other effect than greatly to reduce the prospect of success from future thorough treatment of the same kind.

The details of the moral treatment must be varied according to the individual character. In every life the forces differ by which character i shaped, and the result is the same in no two persons. The first aim of the physician must be to learn what those forces have been, the nature of the mind they have moulded, and by what influences its balance has been disturbed. The knowledge may be derived from friends, from the person to whose immediate charge the patient is committed, or from the patient herself, by ascertaining her habitual mode of life, her likes and dislikes, her tastes and occupations, and from her account of her own symptoms.

The object of moral treatment is to restore to the will the control which it has lost. Sometimes it is motive that is deficient and has to be reinforced and the influences employed must be those to which the patient's characte shows her to be the most amenable. To obtain motive force, irksom measures are sometimes necessary, adopted, of course, ostensibly for medica and not for moral purposes, and it is in these cases that the Weir-Mitchel system is occasionally most successful. The increase or relaxation of th strict treatment affords an effective means of punishment or reward, b which the will may be stimulated and moral inactivity dispelled. In al cases each successful effort must be commended, and the patient made t realize the progress effected, and the tangible promise of ultimate recover which it supplies. Care must, however, be taken not to push the patient o too rapidly. When once the will has been won over, the patient is apt t try too much, and recovery from a discouraging relapse may be far mor difficult than was the original advance. Power has not only to be evoked bu consolidated, and the real progress is often less than that which is apparent.

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* The various points in management are admirably discussed by Weir Mitchell (loc. cit.)

In many cases, also, the art of control over mind and muscle has been los and the will has to be reëducated by slow and gradual steps. The patier must be made to realize that deliberate endeavor, fruitless at first, will b effectual at last, and that when the enemy cannot be conquered by direc assault his defences may be broken down one by one, or a flank movemer may succeed. Thus, at first a direct effort to control the onset of a hyste roid fit may be unsuccessful, but if the patient can learn by some expedien such as a sudden exertion or a slouch with cold water, to keep off the paro ysm, she will gradually need less and less the adventitious help, and be abl to control the fit by a mere effort of the will. So with paralysis; a delibe ate and well-intended effort to walk may fail, but by learning to stand, fir with support, then alone, and then to walk with help, the will slowly regair its lost power. Such a course of treatment demands, it is needless to say considerable time. A morbid state, the accumulation of years, cannot b dissipated in a day.

It might seem superfluous to caution the physician against increasing th morbid state by any proceeding of his own, were it not that there has bee of late conspicuous violation of this obvious rule. Attention is a poter intensifier of the symptoms of hysteria, and by too frequent examination an demonstration, certain phenomena, such as anæsthesia, may be kept u which would disappear if disregarded. Especially is this caution necessar regarding the induction of the hypnotic state and of convulsive phenomena The influence of such induction on the patient is distinctly prejudicial, inter sifying the morbid condition, and on no account should a proceeding whic may in any degree do the patient harm be permitted or adopted merely t gratify a scientific curiosity.

Apart from the tonics already mentioned, certain drugs have for man centuries been held in repute as powerful "anti-hysterical" remedies They are for the most part substances of nauseous taste (musk, asafœtida valerian, etc.), and their early use was associated with the theory that by thei offensive character they drove the errant uterus back to its place. Althoug by some recent writers their influence for good is discredited, they are sti largely employed. Most of them undoubtedly have a stimulant action o the nervous system, which is imperfectly recognized by their designation a "antispasmodics." Their flavor is usually (though not always) most di tasteful to the patient, and the desire to escape from the necessity of takin them may constitute some stimulus to the will. This indirect moral infl ence cannot be denied, and although it is slight, and may be entirely imp tent against adverse circumstances, yet it is sometimes distinct, and eve when trifling is not to be despised in the treatment of a disease which mak the utmost demands on the resources of the physician. Distinct improv ment sometimes follows their use, even when (as in hospital out-patients) th patient's circumstances remain otherwise unaltered. Asafœtida, valeria and turpentine have been those which have been, in my experience, the mo useful. Valerianic acid in the form of valerianate of zinc, is as useful as th vegetable product. Morphia, in stimulant doses ($\frac{1}{24}$ grain) is also sometim serviceable. Large doses of opium were formerly given, but are now little used. Bromides are occasionally of service for insomnia and restlessness and for mental derangement, but their value in hysteria is not so great as their influence in other diseases might lead us to anticipate. Narcotics must be employed only with great caution, since the tendency to their habitua use is very strong in the subjects of hysteria, who furnish most of the habitua chloral drinkers met with among women. If chloral is given at all it should be without the patient's knowledge, a pseudonym, known to the chemist being, if necessary, employed in the prescription.

Treatment of Particular Symptoms.—The general principles of treatment are applicable to all cases of hysteria; the modifications needed to suit the endless varieties of the disease must be left to a large extent to the common sense of the practitioner. Some special symptoms may, however, require special measures; others will disappear when the general disease is treated.

Mere local tenderness of ovaries, spine, etc., may usually be left alone Sometimes its disappearance is facilitated by counter-irritation, as, for instance, by repeated sinapisms and by rubbing, at first gentle and afterward more firm. Supports to the spine, poroplastic jackets and the like, are better avoided. They may give temporary relief, but the patient becomes dependent upon them, they cannot easily be given up, and they increase the difficulty of effecting a radical cure. The various spontaneous pains can rarely be left without some local attempt at their relief—local application of chloroform,. counter-irritation, and hypodermic injections of simple water o atropine or of cocaine are the most useful. Hypodermic injection of morphine always gives relief, but is a dangerous remedy; it should very rarely be employed for hysterical neuralgia, and the hypodermic syringe should never be placed in the patient's hands. Care must be taken to avoid increasing the amount of attention given to the pain by local measures Patients should be encouraged to disregard it, and to exert themselves in spite of it. It is remarkable how much is sometimes endured in the shape o counter-irritation, the actual cautery, etc., by a patient kept in bed on account of hysterical neuralgia, which is quickly forgotten if she can b induced or compelled to get up and exert herself in some active way. Th various unpleasant sensations short of pain—numbness, tingling, and th like—need no special treatment, or may usually be removed by bromide Hyperæsthesia of the special senses generally vanishes if it is not fostered by unwise attention.

Anæsthesia also will often pass away if it is disregarded and is not perpetuated by repeated medical investigation. If its degree and persistenc renders local treatment desirable, faradization with a wire brush, or spark from a friction machine, are the most effectual. Amaurosis may be treate by stimulation of the retina by a feeble voltaic current, slowly interrupted Loss of hearing and of taste need no special treatment.

Hysterical aphonia sometimes yields at once to exhortation or to th introduction of the laryngoscope. It can usually be removed by faradaism applied to the exterior of the larynx, the patient being made to utter sound during the operation. If this fails, sparks from a static machine ma succeed. In obstinate cases, faradaism to the interior of the larynx or circular blister round the throat are usually effectual, but I have neve found the former necessary. Aphonia is very prone to relapse unless i removal is combined with radical treatment. In the defective coördinatio of the respiratory and laryngeal muscles, Weir Mitchell has found th most effectual plan is to insist on all utterances being preceded by a dee inspiration.

For paralysis of the limbs, no single remedy is so effectual as faradaism The movement of the muscles, and the pain produced have often a profoun effect. In slight cases the full power of movement may be restored by single application. More often its use has to be repeated, and the effect i gradual, each application being followed by a slight increase in power. I is important that the patient should expect the gain in power which wi follow its use. In severe cases, the calls to increased effort must be ver gradual and systematic, care being taken that no attempt is allowed to fa entirely, or is prolonged to fatigue. In severe incoördination, the powe of steady movement has to be carefully and gradually trained ; if necessary first in bed and then on "all fours," the erect position being graduall assumed. Great assistance is often derived, in both ataxy and weakness from a steady frame on wheels, or from sticks or crutches which have a concave handle for the grasp and a large flat base, eight inches by two, covere with india-rubber, as suggested by Weir Mitchell.

Contracture of limbs can sometimes be removed by an unfamiliar agen which produces a strong sensory and moral impression, such as faradaism, o static electricity, or a circular blister around the limb. If these fail, mor gentle and continuous measures must be adopted. Violent physical force i seldom successful, and not rarely does harm, failing to relieve the symptoms and inducing other nerve troubles. Occasionally, contracture may b removed by chloroform, and the relaxed limb fixed in some other postur before the patient recovers consciousness. More permanently successful however, is rubbing (which rarely fails, for a time, to lessen the contracture) combined with gentle passive movements, and the position of the limb ma be slowly changed by means of splints. The voltaic current is rarely o service. When the contraction is lessened, systematic voluntary movement should be added. Many forms are extremely obstinate, and their treatmen requires great patience and perseverance. Contraction of the muscles o the jaw is an exception, and generally disappears spontaneously if disregarded.

Phantom tumors in the abdomen are best left alone. They cause littl inconvenience, and are often perpetuated by local treatment. Faradaism to the muscles which are relaxed will lessen the prominence during the application, and this, sometimes combined with rubbing, may be employed if an local treatment is thought desirable.

The tremor-like forms of clonic spasm are merely accessory to the weakness and contracture, and disappear with these. More violent local spasm are sometimes removed by a circular blister around the limb. They are occasionally extremely obstinate, and resist all symptomatic treatment, yielding only to the radical amelioration of the hysteroid state. This is true, also, of rhythmical spasm, which, however, as a rule, is more amenable to treatment, and is sometimes arrested by hypodermic injection of morphia, atropine, or arsenic. The occasional use of morphia for such a symptom entails less danger than its employment for the relief of pain.

The visceral symptoms of hysteria also give great trouble. In anorexia and refusal of food, the patient must be compelled to take liquid or pulpy food, and it is often best for the nurse to feed the patient. If even this is refused, the nasal tube may be employed, or rectal injections; these operations are often interfered with by the occurrence of hysteroid convulsions, which usually prevent the employment of the stomach pump. The nasa tube should not be deferred too long; its moral effect is very great, and may save much further trouble. By its means or the œsophageal tube the food may be given prepared as described below.*

Hysterical vomiting is often most troublesome, both on account of its obstinacy and of the impaired nutrition to which it may give rise. It is best treated by absolute rest, liquid food, and the spare feeding which res renders adequate. The rejection of food should be met by judicious disgust, and a more liberal diet may be promised on its cessation. Severe vomiting is one of the symptoms for the treatment of which seclusion i essential. When other means fail, forced feeding by the stomach pump ha been successful. The avoidance of the act of swallowing seems to have something to do with its effect in arresting the vomiting, since when injected food had for some time been retained, that taken in the ordinary way may still be vomited (Dujardin-Beaumetz).

The vasomotor symptoms of hysteria rarely yield to direct treatment Occasionally digitalis lessens the palpitation, and the flushings and cardia discomfort are relieved by belladonna; but in spite of the apparent independence of these troublesome symptoms, the radical treatment of th disease is usually the only means of affording more than temporary relief.

The treatment of the cases which present hysteroid convulsions mus vary according to the nature of the seizures. It has been mentioned tha the attacks of which such convulsions are the conspicuous feature may b really compound, consisting of a true epileptic fit and of hysteroid convu sion as a post-epileptic phenomenon. In such cases the treatment has t be that for epilepsy, and bromides do most good. Where, however, ther is no real epileptic element, and the attacks are purely hysteroid, bromide usually fail, and other agents sometimes succeed. Moral treatment has great influence on many of these cases. The attacks often cease at onc when the patient is admitted to a hospital. If they do not, she should b urged to endeavor to control them. The attempt will often be aided b the effect of drugs in rendering the attacks less severe, and the patien should be impressed with the fact that attempts at control, which ma have failed before, will, thus aided, be less difficult. When direct contr fails, various expedients may afford help, as cold water, smelling-salts, etc.

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* Debove, who has largely employed this forced feeding, recommends that the daily qua tity should be 5 pints of milk, 1¼ pounds of meat, 12 eggs, and the flour of cooked ar dried lentils. The raw meat is minced, warmed, pressed, placed in a stove and drie thoroughly, and reduced, in a mortar, to an extremely fine powder. The lentils should al be cooked and then pounded. The whole is mixed with the milk and with the beaten- eggs, and given in four or five injections into the stomach (*Gaz. Méd. de Paris, 188* p. 206).

Drugs exert more influence on the convulsions than on many of the othe symptoms of hysteria. Those that are most useful are the valerianate o zinc, iron, morphia, and turpentine. Iron appears to do good by a speci action on the nerve centres, apart from its hæmatinic effect, and is especially useful in the hysteroid attacks which occur in boys. Turpentine ma be given in ten-minim doses, gradually increased until slight symptoms o strangury are produced, when it must be omitted for a few days. It is perhaps more useful than any other single remedy.

The onset of slight hysterical seizures, emotional in character, may ofte be averted by an "antispasmodic" draught of ether, lavender, etc., an sometimes by the inhalation of nitrite of amyl. The developed attac may also be usually arrested, although the means most effectual for this are n the same in all cases. A douche of cold water on the head often succeeds but it sometimes needs to be very copious ; the first jugful may only increas the violence of the convulsion, and a second may arrest it. When s much water is needed, it is a clumsy remedy. A few teaspoonfuls poure into the mouth, or, if this is closed, into the nostrils, are often at onc effectual, apparently by stimulating the respiratory centre. The sam result may readily be effected by Dr. Hare's plan of closing the mouth an nose with a towel for fifteen or twenty seconds. Sternutatories usually fail Another method of cutting short an attack is a painful impression. I ovarian (or other hysterogenic) tenderness is known to exist, firm pressure a the spot will often succeed, but considerable force has to be employed t overcome the rigidity of the abdominal muscles, and the pressure must b maintained for several minutes, or the attack will recommence.

Cutaneous faradization is another very effectual remedy, and a curren from a small magneto-electric machine answers very well. The electrode may be applied almost anywhere, on the two hands, or from the neck to th hand ; a strong current must be used. Where all other advantages fail, have found the hypodermic injection of a twelfth or sixteenth of a grain o apomorphia invariably successful. It causes vomiting in six or eight minutes and with the nausea, which comes on in about four minutes, all spasm ceases and the patient regains perfect consciousness. In the paroxysms of laryngeal spasm, which often resist all other influences, apomorphia is alway effectual. If it is not at hand, nausea and vomiting may be induced b tickling the fauces. The arrest of attacks by apomorphia is not a pleasant process to the patient, and has a powerfully moral effect in lessening th tendency to their occurrence.

TRANCE AND CATALEPSY.

A curious group of diseases have for their characteristic the occurrence o a state of sleep-like unconsciousness, which is called "catalepsy" when it i accompanied by a peculiar plastic state of the limbs, and "trance" o "lethargy" when this condition of the limbs is absent. These condition are most common in the subjects of hysteria, but occur sometimes in state of weakness or exhaustion of the nervous system when there are no pronounced hysterical symptoms. They may often be induced by the method termed "Mesmerism," and "hypnotism." The conditions thus produced were long ago studied by Braid, Elliotson, and others, but the extraordinary susceptibility presented by the elaborate hysterics of France ha enabled them to be re-investigated in a more scientific manner, by Charcot Feré, and others. These investigations have yielded many curiou facts, and an enormous amount has been written on the subject during th last few years, but its practical importance in medicine is not considerable The phenomena may possibly throw light on some obscure points in th physiology of the nervous system, but their chief importance at present i due to their correspondence with the similar states that arise spontaneously Hence a very brief outline of the most important facts may be given.

INDUCED HYPNOTISM.

Three different conditions may be produced in the hysterical, and hav been thus summarized by Charcot.*

1. A *cataleptic* condition may arise under the influence of some loud, unexpected sound or dazzling light, or persistent fixation of an object by th eyes. It may also occur as a sequel to the second form, the state of lethargy if the eyelids of the lethargic patient are raised in a well-lighted room. Th cataleptic condition is characterized by rigid fixation of the limbs, with ope eyes, a stiff look, and slow breathing. In spite of the fixation of the limbs they can be moved passively with great readiness, and remain in whateve posture they are placed, without apparent fatigue. Very little evidenc of myotatic irritability can be elicited. The skin is completely anæsthetic but the organs of special sense retain their sensibility, at least in part, an through their means the subject can be induced to perform automatic action of a more or less complicated character, by imitation or by the suggestion of description, but when left to herself relapses into the state of statuesqu rigidity.

2. The *lethargic* condition. A person in the cataleptic state just describe may pass into the lethargic condition on the occurrence of a deep inspiration, or the transition may be induced by closing both eyes, or by darkening the room. The condition may be primarily induced by prolonged fixation of the eyes. In this state the eyes are closed or half closed, and th globes deviate upward and inward. There is complete relaxation of th muscles, and the limbs fall when raised. Myotatic irritability is greatl

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* *Progrès Méd., 1882,* p. 124.

augmented, and there is an extraordinary increase in the mechanical excitability of the nerves and muscles, so that contraction follows not only a ta on the muscles but pressure on the nerves. A pointed object pressed on th "motor point" of a muscle will put it in action, just as does faradaism. I the limbs and trunk the contractions thus produced continue; they ma even persist after the patient has emerged from the lethargy, and can onl be removed by stimulation of the antagonists. In the face the contractio ceases when the pressure is discontinued. There is complete anæsthesia, no only in the skin but also in the organs of special sense. If the eyelids ar raised in a lighted room, the lethargy passes into the first condition, that o catalepsy, and if only one eyelid is raised the change occurs only on th corresponding side, so that the two conditions coexist and can be contrasted

3. The third condition is termed *somnambulistic*. It may develop pr marily on persistent fixation of an object by the eyes, or through the action o a repeated monotonous sensory impression, and it may be produced also i the condition of catalepsy or lethargy by gently rubbing the top of the skull In this condition the eyes are closed or half closed; there is a sleepy look the relaxation of the limbs is less complete than in the lethargic condition myotatic irritability is normal, and there is not the hyper-excitability of th nerves and muscles that characterizes the state of lethargy, but rigidity o the limbs may be produced by gently stroking the skin, and there is the considerable resistance to passive movement. Sensibility is not lost, an there is an increased sensitiveness to certain cutaneous impressions. Aut matic actions of complex character can readily be produced by exampl This condition can be made to pass into the lethargic state, with neuro-mu cular hyper-excitability, by gentle pressure with the finger on the eyeball

Similar phenomena may be produced in many hysterical patients in th country. When the tendency to these conditions is strong, the patien often pass into them spontaneously.

CATALEPSY.

Spontaneous catalepsy has been met with in both sexes and at most age from six to sixty, but it is most frequent in the female sex and in early adu life. It thus occurs chiefly when hysteria is most common, and in mo cases there are other distinct symptoms of this neurosis; when there are no the cause of the disorder is generally such as might give rise to an hysteric attack. Nervous exhaustion is the common predisponent, and freque immediate causes are emotional disturbance, especially religious excitemen sudden alarm, or blows on the head or back. It occasionally occurs in th course of mental affections, especially melancholia, and has been said t occur in epilepsy, but its connection with the latter disease is very doubtfu In an imperfect form, it has been supposed to be a consequence of malari poisoning, and has been observed in some toxic conditions, such as chlor form narcosis. Very rarely a similar condition has been met with in th course of some organic diseases of the brain, especially in meningitis.

SYMPTOMS.—In some cases headache, giddiness, or hiccough, has precede the attack. The onset of the special symptoms is usually sudden, commonly with loss of consciousness. The whole or part of the muscular system passes into a state of rigidity. The limbs remain in the position they occupied at the onset, as if petrified. The muscular rigidity is at first considerable, and movement is resisted; but after a short time the limbs can be moved, and then remain in any position in which they may be placed. The resistance to passive movement is peculiar; it is as if the limbs were made of wax, and hence the condition has been termed *flexibilitas cerea.* The rigidity commonly yields slowly to gravitation. The countenance is usually expressionless. The respiratory movements and heart's action are weakened. Substances placed in the back of the mouth are swallowed, but slowly. The state of sensibility varies; in profound conditions of catalepsy it is lost to touch, pain and electricity, and no reflex movements can be induced even by touching the conjunctiva. In other cases partial sensibility remains, and reflex phenomena may be excited. In rare instances paroxysmal hyperæsthesia is present. Consciousness is frequently lost, but may remain more or less obscured, rarely intact. The temperature is commonly lowered. The attack may last a few minutes or several hours. Recovery is gradual or sudden; it is common for the patient at first to be unable to speak. Sometimes a strange periodicity may be observed in the occurrence of the paroxysms. In the intervals between the attacks, headache, giddiness, or hysterical manifestations may be present, or the patient may feel and seem perfectly well.

PATHOLOGY.—The pathology of catalepsy can only be guessed at from the character of the symptom. There is apparently a continuous stimulation of the muscles by some of the motor centres, which pass into a state of peculiar over-action, probably in consequence of the withdrawal of some restraining influence, combined with the stimulation produced by certain sensory impressions. The condition seems thus to be neither purely central nor purely reflex, but a combination of both.

The DIAGNOSIS of the condition presents no difficulty. Many cases of simple trance have been included under the term "catalepsy," but it is better to restrict the name to the condition in which the peculiar rigidity exists. The prognosis is favorable in simple catalepsy in proportion to the freedom of the intervals from affections of sensibility or motion. In pronounced hysteria and psychical affections the condition is often obstinate.

TREATMENT.—During the attack itself little can be done save an attempt, which may be repeated at intervals, to rouse consciousness by external stimulation. The ordinary applications, ammonia to the nostrils, cold douches, etc., often fail. A pinch of snuff will, however, often succeed. Another effectual stimulant is faradaism, which may be applied to a limb or to the cervical spine. The current should be gentle at first, and gradually increased. Emetics are also useful in cutting short an attack. Injections of tartar emetic into the veins have been used with success, but can hardly be recommended. The subcutaneous injection of apomorphia, $\frac{1}{20}$ to $\frac{1}{12}$ of a grain, is a safe and efficient remedy for such paroxysmal conditions (see p. 1329). In the intervals between the attacks the treatment is that of hysteria.

TRANCE; LETHARGY.

Trance or lethargy, as it occurs spontaneously, is a peculiar sleep-like state, from which the patient cannot be roused, or can be roused only imperfectly, and which is not due to organic disease of the brain or to any poison. The morbid state in which a patient has paroxysmal attacks of true sleep, from which he can be completely roused, should be distinguished from true trance.

Causes.—Trance, like catalepsy, occurs chiefly in association with hysteria, and has the same general causation. But the condition is rare. Briquet met with only three cases among the large number of cases of hysteria he had observed. Only four cases have come under my own notice. Most of the subjects are females between twelve and thirty; rarely it occurs in children or young men; still more rarely in adult men. Neuropathic heredity often exists; the mother of one patient, for instance, was insane. Among rare causes that have been described are excessive brain work, and exhausting diseases such as typhoid fever. Injury to the head has sometimes been followed by it, apparently rather through the emotional disturbance than through any physical lesion. Thus one patient, who had an attack after a blow on the head, had had similar attacks in connection with mental depression (Marduel). Emotional disturbance is often the immediate excitant, or an attack may follow an hysterical convulsion. Weir Mitchell has described a case in which conversation on any unpleasant subject would bring on an attack. In one celebrated case, that of Colonel Townsend, the state could be induced voluntarily, and in one attack so induced he died. Voluntary induction is not uncommon in the East. The production of the state in hysterical subjects has been already described.

Symptoms.—The onset is usually sudden. In one case that came under my notice, a girl went into a room by herself, and was found, shortly afterward, in a state of trance sleep that lasted forty-eight hours. Another case, recorded by Madden, came on under similar circumstances, and lasted for a fortnight. The onset has been occasionally attended by a sensation resembling the globus hystericus. In the cases after typhoid the delirium of the fever has passed into a condition of comatose sleep that lasted for several weeks; this sequel may be connected with the marked mental weakness or loss of speech that sometimes succeeds this disease.

During the trance sleep the face is usually pale. The limbs are relaxed, as a rule, throughout the attack, but sometimes there has been brief initial stiffness, or occasional transient cataleptic rigidity; tonic spasm, and distinct hysteriod convulsions have occurred from time to time in some cases. The eyelids are closed, and attempts to open them are resisted. The eyeballs are usually directed upward and to one side; the pupils are moderately dilated or contracted, and almost always act to light. Reflex action in the limbs has been lost in deep trance, increased in slight cases, and cutaneous stimulation sometimes induces muscular contracture. The reflex action from the conjunctiva and nose may even be lost, and pressure on the ovarian region, if this was tender, may have no effect. In profound trance the mental functions seem to be in complete abeyance, but in slighter degrees the patient may be aware of all that passes, although unable to make even the slightest manifestation of consciousness. The special senses may even be unnaturally acute. Occasionally there is evidence of spontaneous mental action, analogous to dreaming, manifested by exclamations, and even by movements; in rare cases, there is the "obedient automatism" so conspicuous in the induced trance, in which hallucinations can be induced and actions excited, by suggestions made to the patient.

The pulse is small and the sounds of the heart are weaker than normal, and have even been scarcely audible. Its frequency may be normal, increased, or lessened; in one case it fell to 40 per minute (Weir Mitchell). The respiration may present similar changes, and may become so gentle and deliberate as to be scarcely perceptible; it is said that even a mirror over the mouth may be undimmed. Rhythmical variations have been observed. The temperature is normal in the central parts, lowered in the periphery. Urine may be retained in the bladder or passed into the bed. Thus, the extreme degree of trance may involve such a depression of the vital functions that the patient may seem to be dead. This state has been called "death trance," and has furnished the theme for many a sensational story, but the most ghastly incidents of fiction have been paralleled by authenticated facts.

The duration of trance has been a few hours, a few days, or several weeks. Rarely it has been said to last for many months. In cases that last more than a few days, there are remissions in which the patient, half awake, will take food and then relapse into stupor. In cases of long duration the trance after a time gets less profound. The termination, if sudden, may be accompanied by vasomotor disturbance, and even by extravasations into the skin. In one case of death trance it is said that the preparations for fastening down the coffin lid caused a copious sweat to break out. After prolonged trance has passed away, profound nervous prostration remains for a time, sometimes with mental dullness. The condition may recur, even many times. A soldier slept for seventy or eighty hours on six occasions during the course of two years (Marduel). Most cases end favorably. The extreme depression of the vital functions seems to enable life to be maintained for a long time on a very small amount of nourishment. A few cases of death are on record; in one of these the trance succeeded typhoid fever.

PATHOLOGY.—No changes have been found after death to explain the nature of trance, which is as mysterious as is that of ordinary sleep. It cannot be explained by the assumption of mere cerebral anæmia, for this may be profound without trance, and the signs of vascular depression commonly succeed the onset. The phenomena, viewed in the light of the induced varieties, suggest rather a state of inhibition or arrest of action of the nerve cells subserving the higher psychical functions, and that the morbid state spreads to lower centres in varying degree.

DIAGNOSIS.—The diagnosis of trance rests on the impossibility of rousing the sleeper, combined with the absence of any evidence of a local cerebral lesion or a toxic cause. Other diagnostic symptoms are the pallor and vascular depression, the occurrence of convulsive phenomena of hysteroid type, and the previous occurrence of other manifestations of hysteria. These symptoms sufficiently distinguish trance sleep from apoplexy, for which, at the onset, it is sometimes mistaken. The distinction from catalepsy rests on the muscular relaxation.

In cases of "death trance," in which no sign of vitality can be recognized, the presence of life may be ascertained (1) by the absence of any sign of decomposition; (2) by the normal appearance of the fundus oculi as seen with the ophthalmoscope; (3) by the persistence of the excitability of the muscles to electricity. This excitability disappears in three hours after actual death. In a case observed by Rosenthal, thirty hours after supposed death, the muscles were still excitable, and in forty-four hours the patient awoke.

PROGNOSIS.—In most cases of lethargy the ultimate prognosis is good. The slighter the degree of the trance the shorter is likely to be its duration. The prognosis is grave only when the lethargy was preceded by a state of great physical depression, especially when the condition succeeded an acute disease.

TREATMENT.—The treatment has to be directed to two ends; the maintenance of life, and the arrest of the trance. Advantage must be taken of any intervals of semi-consciousness to give nourishment in a concentrated form. If swallowing is continuously impossible, food must be given by the nasal tube, or by enemata. Warmth should be applied to the extremities, and care taken to prevent bedsores. In severe cases, every attempt at arrest is often fruitless. In cases of moderate severity stimulation of the skin is effective in rousing the patient. The most powerful cutaneous excitant is strong faradaism. In one case, which had lasted for thirty-six hours, I found that the application of strong faradaism to the arm quickly cut short the trance. In another case, which lasted for several months, this treatment had for a long time no influence; afterward the patient could be partially roused for a short time by faradaism, and by repeating the application at the same hour every day, a tendency to periodical waking was established, the remissions became longer and more complete, and the attack was ultimately brought to an end. Nervine stimulants, such as ether and valerian, may be given by the bowel, or sulphuric ether may be injected subcutaneously. Alcohol must be given with caution and in small quantities; enemata of strong coffee are often more useful. It is probable that nitro-glycerine or the inhalations of nitrite of amyl would have considerable influence. Transfusion of blood has been proposed, and would be justified in cases following exhausting disease, when death is threatened. The recurrence of attacks must be prevented by the improvement of health, physical and moral.

NARCOLEPSY.—The term "narcolepsy" has been used in several senses, but is best applied to a condition, for which some name is needed, in which there is a tendency to fall into sound sleep for a short time, usually for a few minutes, rarely for an hour or more. The condition is distinguished from trance by the brevity of the attacks of sleep, by their strong tendency to recur, and by the fact that the patient can commonly be roused at any time from the condition. In some cases the malady has been apparently the result of some peculiarity of the nervous system. Thus, in one recorded case, a man had been liable, all through his life, to such attacks of sleep, which were induced by most varied influences. He had a nasal fistula, and whenever a probe was passed down this he fell asleep. In other cases the affection comes on in adult life, and passes away after lasting for a few months or years. Thus, in one girl the attacks commenced at sixteen, and continued till she came under treatment, at twenty-two. She would suddenly feel drowsy, her eyelids drooped, in a moment she was sound asleep, and after about five minutes woke up quite fresh. Intensely vivid dreams accompanied the sleep, but she never could recall their details; sometimes she would speak aloud in the dream. There was no change in the color of the face. After deficient sleep at night, the sleep attacks lasted longer, sometimes even half an hour. She could be readily awaked at any time, and by an effort could keep off the attack, but then could not help yawning continually, and felt uncomfortable. There was never headache. Such attacks occurred four or five times a day, when she was engaged in sedentary occupations or studies, but when she led an active life, as, for instance, on a holiday, they would be absent for two or three weeks.

The characters that separate this affection from ordinary trance have been already mentioned. It is most likely to be confounded with minor epilepsy, but from this it is sharply distinguished by the perfect resemblance of the attacks to ordinary sleep in their onset and character. When a feeling of somnolence is the warning of an attack of true *petit mal*, the unconsciousness lasts only a few moments.* An active life with change of scene is probably the most important element in treatment. In the case mentioned above, no drugs did so much good as a combination of caffein and nitroglycerine, which almost entirely arrested the attacks.

AFRICAN LETHARGY.—A curious malady, presenting similar somnolence but of very different nature, is the "sleeping sickness" of the West Coast of Africa, met with chiefly in the Congo and Sierra Leone regions. It affects exclusively negroes, and occurs in both sexes and at all ages, but is most frequent in males between twelve and twenty. Except that depressing emotions seem to predispose to it, the proximate causes are entirely unknown. Europeans living in the same localities are exempt. Swelling of the cervical glands sometimes occurs at the onset, and they are excised by the native doctors as a remedial measure; but the condition is not invariable, and its influence is doubtful. The general health at first is good. There is a gradually increasing tendency to somnolence, and the patient will fall asleep at his work or over his meals. At first he can be roused, and if treated by cutaneous stimulation and purging, the symptoms may be removed for a little time, but soon recur and increase in spite of treatment, until at last the patient is always asleep and refuses food. He gradually emaciates, and dies at the end of three or six months from the onset of the symptoms. Just before death the disposition to sleep often ceases. The disease is extremely fatal. Guèrin met with 148 cases, all of which died. The observations of Gore and others place the mortality somewhat lower—at about 80 per cent. Post-mortem examination has revealed only hyperæmia of the arachnoid, slight signs of chronic meningitis, but no considerable excess of fluid within the ventricles or outside the brain. The cerebral substance is usually pale. No treatment appears to influence the symptoms. Only one observer (McCarthy) has seen good from excision of the cervical glands. This mysterious affection clearly needs more systematic investigation than it has yet received.

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* The term "narcolepsy" has been applied also to such epileptic attacks; a needless and confusing use of the word.

HYPOCHONDRIASIS.

Hypochondriasis is a morbid state of the nervous system in which there is mental depression due to erroneous ideas of such bodily ailments as might conceivably be present. This limitation is necessary to distinguish the condition from those forms of actual insanity in which there is a delusion of the existence of some impossible ailment—impossible either in its nature or else by reason of its incompatibility with life. A patient, for instance, who thinks that his spinal cord has become detached from the brain, or who believes that his throat is hermetically closed, is insane, not hypochondriacal.* The term is, moreover, almost exclusively applied to the condition above defined when this is met with in men. A similar state in females is regarded as forming part of "hysteria." The name has come down to us from the times of Hippocrates and Galen, and, like "melancholy," had its origin in the idea that the conscious life was largely influenced by "vapors" produced in the abdominal organs. It was not until the early part of the present century that the disease was generally admitted to be one of the brain.†

Causes.—The affection consists in a particular disposition of the brain, which leads to habitual anxiety without adequate cause, and to the concentration of this anxiety on the individual's own health. This morbid tendency is often a matter of individual temperament, which may be the result of neurotic inheritance; the most severe forms occur in persons whose family presents a tendency to insanity. In other cases there is no morbid heredity, but the state is acquired through persistent ill health, which sets up the morbid mental habit of self-attention and concern. These two causal varieties may be termed the "acquired" and the "temperamental" forms. The age at which hypochondriasis comes on varies according to its causes. It is scarcely known in childhood, but when due to inherited temperament it often becomes established quite early in adult life, and sometimes is even distinct in youth. It may, however, commence at any period, and the acquired form often commences in or after middle age.

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* It is unfortunate that alienists sometimes apply the term to the cases of insanity with somatic delusions; this is contrary to its customary use, and productive of confusion.

† An interesting sketch of the history of hypochondriasis is given in Gull and Anstie's article on the disease, in Reynolds' "System of Medicine," vol. ii.

Hypochondriasis seldom develops without some exciting cause in the shape of a distinct bodily ailment, although this is often very trifling in degree. It suffices, however, to give rise to unpleasant sensations, which excite attention and give rise to concern. Dyspeptic troubles are the most common excitants; others are palpitation of the heart, and weakness of the nervous system; but there is hardly any part of the body in which functional disturbance may not be the starting point of the malady.

SYMPTOMS.—In hypochondriasis there are three chief elements; excessive anxiety regarding the personal health; undue attention to any indications of derangement, especially to the various sensations emanating from the organs of the body; and, lastly, as the consequence of these, there is some false idea that disease is present which does not exist. Occasionally, the last is absent; the patient may know and realize that he is free from any serious malady, but he is rendered miserable by the continuous discomfort he experiences.

This discomfort, as already stated, often has its origin in some actual disorder, but it is always vastly increased by the habitual attention paid to the sensory impressions proceeding from the organ deranged, or from other parts when there is no local disorder. The influence of attention has been already mentioned in the account of "cephalic sensations" (p. 1195) which often form part of the symptoms of hypochondriasis, and the statements there made are applicable to all the various sensory impressions from other organs that disturb the hypochondriac's life. It is this continuous attention that perpetuates the morbid state and renders its treatment so difficult. Not only are sensations attended to until consciousness is continually under their influence, but the patient is always searching for indications of the ill health he dreads. He scrutinizes his tongue before breakfast and his evacuations after breakfast, and notes during the day the influence of each meal on his abdominal feelings, of each exertion of his pulse, and of mental work upon his head. It is easy for the hypochondriac thus to collect each day a series of "symptoms" which he ponders over and endeavors to interpret. As an instance of this habit, I may quote, *verbatim*, the description given to me by one of these unfortunate sufferers, in whose physical condition no other flaw could be found than trifling occasional indigestion. "I do not breathe free; I do not breathe clear. After I did my work yesterday there was a pain in my temples and in the back of my head. There is a little pain in the heel when I press upon it. I have a sensation of tightness round the sides of the chest. I have also felt slight tightness about the knees. The appetite is not the thing at all. There is slight distention; I have not found it to-day, but I did yesterday. Last night I felt the food in my throat, and a noise in the chest such as you feel in the ear. My head is hot on the top now. Talking, even for a few minutes, seems to affect the eyes, and an uncomfortable feeling comes in them. This morning in the train, too, after it stopped, I seemed to feel for a moment as if I was going backward and forward. My forehead gets hot when I talk. Some days ago I had an uncomfortable feeling in the loins, and afterward in the bowels, and a week ago I had some pain in the armpit." This patient had no false idea of local illness; his symptoms were too vague and varying to permit him to entertain the idea, but it was impossible to convince him that there was not some grave general derangement of his health. In the subjects of the temperamental form, with neurotic heredity, there is often slight real derangement of the nervous system, headaches, neuralgic pain, vague giddiness, noises in the ears, sensations of weakness of the legs, etc., which keep up the mental state. Similar troubles occasionally characterize also the acquired form, especially in gouty subjects.

In many cases, moreover, slight derangement of the functions of some one organ constitutes an anchor for anxiety; attention is focused on the local sensations so that they attain an ever-growing intensity; and the conception of a definite ailment becomes fixed, sometimes ineradicably. In some instances the idea has its origin not so much in the sensations of the patient himself, as in the fact that circumstances have brought the disease prominently under his notice. This influence is, indeed, often operative when there is no persistent hypochondriasis. Most medical students imagine, at some period of their career, that they are the subjects of heart disease. But the study of medicine provides the antidote, as well as the bane, to all healthy minds. With mental instability, however, an idea often becomes firmly rooted. An elderly man firmly believed himself to be the subject of diabetes, from which a friend had died, and he persisted in his belief in spite of the assurance that there was no sugar in his urine.

The subjects of this disorder may have the appearance of perfect health, but those in whom the condition is of long duration are often careworn and depressed in aspect, and are preoccupied in manner. The sufferings are described in language that is obviously exaggerated. The amount of depression is often very great, but in cases of pure hypochondriasis there is scarcely ever the suicidal tendency common in melancholia. Many sufferers, indeed, are nervous, timid persons, who dread illness both for its own sake and as a possible path to the grave. Occasionally a temperamental hypochondriac may be met with who describes life as shorn of all pleasure by his sufferings, and asserts that he has suicidal impulses, but if the latter exist there is generally more than pure hypochondriasis.

To describe in further detail the symptoms of these cases, would be merely to multiply particular illustrations of the general facts above stated. There is hardly a malady that is not at times the subject of the morbid fancy. The general facts are true of all; it is only the applications that vary. One class of cases, however, commonly included in this category, deserves special mention, the cases of sexual hypochondriasis. Most of those who wrongly imagine that they have some ailment of the sexual organs do not really suffer from hypochondriasis. This erroneous belief is justified because it is founded on evidence which is adequate to cause the belief in those who have no means of estimating its value. The idea of spermatorrhœa sometimes does concentrate the attention on sensations from those parts, and may set up true hypochondriasis, but on the whole the sexual variety is rare except as part of pronounced insanity.

Diagnosis.—The diagnosis of hypochondriasis depends, in the first place, on the exclusion of the disease from which the patient imagines that he is suffering. This, of course, involves an accurate knowledge of the symptoms of each malady, and it is necessary to exclude real disease before the obvious mental state can be allowed to influence the diagnosis. The danger that a mistake of this character should be made depends on two circumstances. First, on neglect of the consideration just mentioned; the obvious temperament leads to a hasty diagnosis and deters from thorough examination. It is obvious that hypochondriasis does not exclude organic disease, and that hypochondriacs not only may, but at some time must, suffer from such affections. Secondly, error is sometimes due to the fact that the patient suffers from some uncommon disease, the symptoms of which are unfamiliar or unexpected. Thus, a man with very distinct symptoms of diphtheritic paralysis had been told that these were all due to nervousness and fancy, because the patient was a member of the medical profession, the diphtheria had been doubtful, and it had been acquired in an unusual way —by inoculation.

The other diagnostic question is the distinction of hypochondriasis from insanity. It is in the permanent hypochondriasis, with a family history of insanity, that this difficulty arises. The distinction depends chiefly on the reasonableness of the false ideas. A patient who has unpleasant sensations in the abdomen, and believes that he is suffering from cancer, in spite of the assurance that he is not, cannot on this account be regarded as insane. But a man who believes that his brain has been converted into sawdust is, by that belief, proved insane. Of course, pure hypochondriasis may coexist with other independent evidence of insanity, just as it may with any other malady. Many cases, however, are on the border line between soundness and unsoundness of mind—cases in which the patient believes that he is suffering from some disease, not in itself unreasonable, but still out of the question because the evidence of it would be tangible and does not exist. Such patients are often actually insane, although they cannot, and ought not to be, treated as such, unless the mind is unsound on other points.

Prognosis.—The distinction between the two forms of hypochondriasis, the acquired and the temperamental, is important in regard to prognosis. The subjects of the latter form seldom recover; if one false idea is removed, another takes its place. The acquired form, on the other hand, sometimes passes away altogether, and the prospect of recovery from it is good in proportion to the shortness of time it has existed, and to the degree of derangement of health in which it has arisen.

Treatment.—The details of the treatment of hypochondriasis have to be varied to suit the exigencies of each case; the principles are the same in all. The first thing is to correct whatever is actually wrong, both in the patient's general health and also in the organs to which his attention is directed. In acquired cases especially, there is often some weakness of the nervous system that may be lessened by nervine tonics and by an improvement in the patient's mode of life. The second element is the removal of the false ideas and the withdrawal of the attention of the patient from his physical condition. The statements made regarding the treatment of "cephalic sensations" (p. 1196) are applicable to all forms of hypochondriasis, and it is unnecessary to repeat them, or to apply them in detail to similar symptoms in other parts. It is essential to make the patient realize how misleading bodily sensations are regarding the actual condition of the parts from which the feelings seem to proceed, and how essential it is that the sensations should be disregarded. He should also be made to understand that his efforts to neglect them will not be at once successful, and that perseverance for a long time will be necessary. A grave responsibility rests upon physicians who are consulted by these patients. The mere consultation, even when the advice is wise, helps to perpetuate the morbid state, and when all that can be done to remove actual disorder has been accomplished, it is often right to refuse to be any longer a passive party to the perpetuation of the symptoms. Sometimes such a refusal, if the grounds for it are made clear, will do more real good to the sufferer than can be achieved by any other means.

NERVOUS WEAKNESS: NEURASTHENIA,

It has become fashionable to apply the term "neurasthenia" to conditions of weakness of the nervous system. The use of the word has brought with it a tendency to regard the condition thus denoted as a definite disease. Books have even been written about it, and it has been divided into various classes, according to the character of the symptoms that are present. It is convenient to be able to designate the condition by one word instead of two, but there is no more justification for regarding neurasthenia as a definite malady, to be distinguished from others and separately described, than there is for adopting a similar course with regard to "debility" among general diseases. "Neurasthenia" underlies and may cause a large proportion of the functional diseases of the nervous system, and when "neurasthenic" patients seek advice, there are generally symptoms of nerve disturbance sufficiently definite in character to bring the case into some special category.

In the description of these special diseases will be found suggestions as to the treatment most likely to remove the underlying weakness of the nervous system. To describe the symptoms and treatment of all that may be collected under the name, would be to repeat that which has been stated already in its proper place, especially in the sections on neuralgia, headache, cephalic sensations, hysteria and hypochondriasis. The chief elements in treatment are the removal of discoverable causes, the regulation of the mode of life, the administration of nervine tonics, and the improvement of the general nutrition, including that of the nervous system. For the last, the physical influences of rest and massage are often useful, on account of the complete repose given to the motor nervous system.

The use of the word "neurasthenia" is convenient chiefly when the patient suffers from so many slight functional disorders that it is difficult to select any one as sufficiently prominent to afford a designation. Even in these cases, much caution should be exercised in giving to the patient a name that may convey an erroneous impression, and may excite unnecessary alarm. It is often better not to gratify the craving for nomenclature that is manifested by many patients, but rather to explain to them that to give to their ailments a definite name would involve more error than truth.

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No. 1.

CATALOGUE

OF

MEDICAL, DENTAL,

Pharmaceutical & Scientific Publications,

WITH A CLASSIFIED INDEX,

PUBLISHED BY

P. BLAKISTON, SON & CO.,
(SUCCESSORS TO LINDSAY & BLAKISTON)
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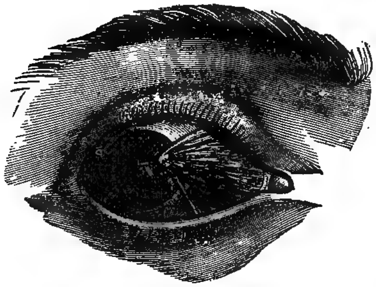

FIG. 27.—PTERYGIUM.

(Attention is called to the help in diagnosis of a cut of this character. It is followed by three engravings showing the operation for Pterygium.)

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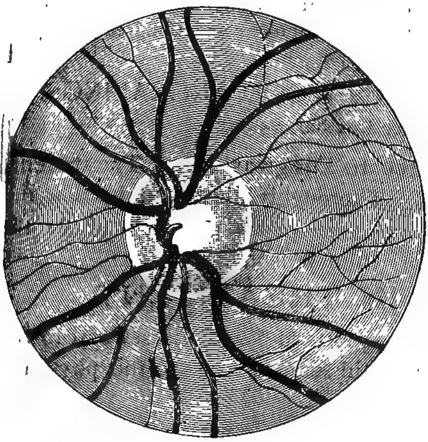

FIG. 83.—PHYSIOLOGICAL EXCAVATION.

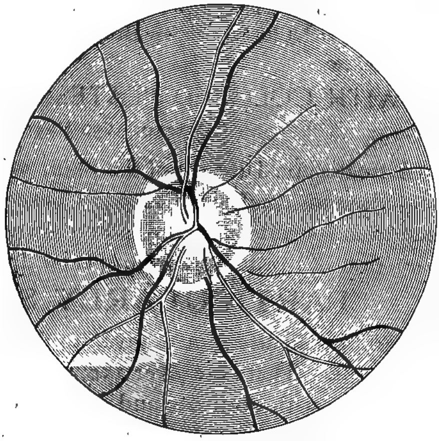

FIG. 84.—ATROPHIC EXCAVATION.

[EXPLANATION OF CUTS.—These illustrations show the difference between physiological and atrophic excavations; a similar engraving shows glaucomatous excavation. It is very important and difficult to distinguish these; the explanation in the text, however, which is very clear, seconded by the pictures, present the differences so plainly that they may always be recognized.]

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